PERFUSION INDEX DERIVED FROM PULSE OXIMETER AS A PREDICTOR OF HYPOTENSION FOLLOWING SPINAL ANAESTHESIA IN ELECTIVE CAESAREAN DELIVERY

DISSERTATION SUBMITTED TO THE TAMILNADU

DR.M.G.R. MEDICAL UNIVERSITY, CHENNAI

In partial fulfilment of the requirements for the degree of

M.D. BRANCH – X

(ANAESTHESIOLOGY)

Register No: 201720309

DEPARTMENT OF ANAESTHESIOLOGY

TIRUNELVELI MEDICAL COLLEGE HOSPITAL

TIRUNELVELI – 627011

MAY-2020

CERTIFICATE BY THE GUIDE

This is to certify that the dissertation entitled **"PERFUSION INDEX DERIVED FROM PULSE OXIMETER AS A PREDICTOR OF HYPOTENSION FOLLOWING SPINAL ANAESTHESIA IN ELECTIVE CAESAREAN DELIVERY"** submitted by **Dr.YATHISH.V**, to the Tamilnadu Dr. M.G.R Medical University, Chennai, in partial fulfillment of the requirement for the award of M.D. Degree Branch – X (ANAESTHESIOLOGY) is a bonafide research work carried out by her under my direct supervision & guidance.

Date:
Place: Tirunelveli

Dr.A. ABIRAMI, M.D.,
Assistant Professor,
Department of Anesthesiology
Tirunelveli Medical College,
Tirunelveli.

CERTIFICATE BY THE HEAD OF DEPARTMENT

This is to certify that the dissertation entitled **"PERFUSION INDEX DERIVED FROM PULSE OXIMETER AS A PREDICTOR OF HYPOTENSION FOLLOWING SPINAL ANAESTHESIA IN ELECTIVE CAESAREAN DELIVERY"** is a bonafide research work done by **Dr.YATHISH.V** under the guidance and supervision of **Dr.A.ABIRAMI, M.D.,** Assistant Professor, Department of Anaesthesiology, Tirunelveli Medical College, Tirunelveli, in the Department of Anaesthesiology, Tirunelveli Medical College, Tirunelveli,, in partial fulfilment of the requirements for the degree of M.D. in Anaesthesiology.

Dr.R.AMUTHA RANI M.D.,
Professor and HOD of Anesthesiology,
Department of Anesthesiology
Tirunelveli Medical College,
Tirunelveli.

CERTIFICATE BY THE DEAN

I hereby certify that this dissertation entitled **"PERFUSION INDEX DERIVED FROM PULSE OXIMETER AS A PREDICTOR OF HYPOTENSION FOLLOWING SPINAL ANAESTHESIA IN ELECTIVE CAESAREAN DELIVERY"** is a record of work done by **Dr.YATHISH.V** under the guidance and supervision of **Dr.A.ABIRAMI M.D.,** Assistant Professor, Department of Anaesthesiology, Tirunelveli Medical College, Tirunelveli, in the Department of Anaesthesiology, Tirunelveli Medical College, Tirunelveli, during his Postgraduate degree course period from 2017-2020. This work has not formed the basis for previous award of any degree.

Date :
Place : TIRUNELVELI

Prof.Dr. S. M.KANNAN,M.S., MCh.,(Uro)
The DEAN
Tirunelveli Medical College,
Tirunelveli - 627011.

COPYRIGHT

DECLARATION BY THE CANDIDATE

I solemnly declare that the dissertation titled **"PERFUSION INDEX DERIVED FROM PULSE OXIMETER AS A PREDICTOR OF HYPOTENSION FOLLOWING SPINAL ANAESTHESIA IN ELECTIVE CAESAREAN DELIVERY"** is a bonafide and genuine research done by me under the guidance and supervision of **Dr.A.ABIRAMI, M.D.,** Assistant Professor, Department of Anaesthesiology, Tirunelveli Medical College, Tirunelveli.

The Tamil Nadu Dr.M.G.R. Medical University, Chennai shall have the rights to preserve, use and disseminate this dissertation in print or electronic format for academic/research purpose.

Place: Tirunelveli
Date:

Dr.YATHISH.V MBBS.,
Postgraduate Student,
Register No.201720309
M.D Anaesthesiology,
Department of Anaesthesiology,
Tirunelveli Medical College,
Tirunelveli.

ACKNOWLEDGEMENT

I wish to express my heartfelt gratitude to our Dean, **Prof. Dr. S.M.Kannan.M.S., MCh.,** Tirunelveli Medical College for allowing me to do the study in this institution.

I would like to express my humble thanks to our professor & Head of the Department **Prof.Dr.R.AMUTHA RANI** M.D., Department of Anaesthesiology, Tirunelveli Medical College, Tirunelveli, whose valuable guidance and constant help have gone a long way in the preparation of this dissertation.

I express my sincere thanks to my professors **Dr Selvaraj, MD, Dr.V.Manorema, MD, Dr. G. Vijay Anand, M.D.** for their constant support, encouragement and suggestions which helped me greatly to expedite this dissertation .

I express my sincere thanks to my renowned teacher and my guide **Dr.A.ABIRAMI,M.D.,** Assistant Professor, Department of Anaesthesiology, Tirunelveli Medical College, Tirunelveli, for his guidance, valuable suggestions and constant encouragement throughout the study.

I express my thanks to all Assistant Professors, Staff members of the Department of Anaesthesiology and all my Postgraduates colleagues, C.R.R.I s and friends for their help during my study and preparation of this dissertation and also for their co-operation.

I wish to acknowledge my parents and family members for their everlasting blessings and encouragement.

I thank all my patients who participated in this study for their extreme patience and kind co-operation.

Above all I thank the Lord Almighty for his kindness and benevolence.

TIRUNELVELI MEDICAL COLLEGE

INSTITUTIONAL RESEARCH ETHICS COMMITTEE
TIRUNELVELI, STATE OF TAMILNADU, SOUTH INDIA PIN 627011
91-462-2572733-EXT; 91-462-2572944; 91-462-2579785; 91-462-2572611-16
online@tvmc.ac.in, tirec@tvmc.ac.in; www.tvmc.ac.in

CERTIFICATE OF REGISTRATION & APPROVAL OF THE TIREC REF NO:1141/ANES/2017

PROTOCOL TITLE: PERFUSION INDEX DERIVED FROM PULSE OXIMETER AS A PREDICTOR OF HYPOTENSION FOLLOWING SPINAL ANAESTHESIA IN ELECTIVE CAESAREAN DELIVERY
PRINCIPAL INVESTIGATOR: Dr.YATHISH.V, MBBS.,
DESIGNATION OF PRINCIPAL INVESTIGATOR: PG STUDENT
DEPARTMENT & INSTITUTION: ANESTHESIOLOGY-TIRUNELVELI MEDICAL COLLEGE, TIRUNELVELI

Dear Dr.YATHISH.V, MBBS, The Tirunelveli Medical College Institutional Ethics Committee (TIREC) reviewed and discussed your application during The IEC meeting held on 27.10.2017.

THE FOLLOWING DOCUMENTS WERE REVIEWED AND APPROVED

1.	TIREC Application Form	☐
2.	Study Protocol	☐
3.	Department Research Committee Approval	☐
4.	Patient Information Document and Consent Form in English and Vernacular Language	☐
5.	Investigator's Brochure	☐
6.	Proposed Methods for Patient Accrual Proposed	☐
7.	Curriculum Vitae of The Principal Investigator	☐
8.	Insurance /Compensation Policy	☐
9.	Investigator's Agreement with Sponsor	☐
10.	Investigator's Undertaking	☐
11.	DCGI/DGFT approval	☐
12.	Clinical Trial Agreement (CTA)	☐
13.	Memorandum of Understanding (MOU)/Material Transfer Agreement (MTA)	☐
14.	Clinical Trials Registry-India (CTRI) Registration	☐

THE PROTOCOL IS APPROVED IN ITS PRESENTED FORM ON THE FOLLOWING CONDITIONS

1. The approval is valid for a period of 2 year/s or duration of project whichever is later
2. The date of commencement of study should be informed
3. A written request should be submitted 3weeks before for renewal / extension of The validity
4. An annual status report should be submitted.
5. The TIREC will monitor The study
6. At The time of PI's retirement/leaving the institute, The study responsibility should be transferred to a person cleared by HOD
7. The PI should report to TIREC within 7 days of the occurrence of the SAE. If the SAE is Death, the Bioethics Cell should receive the SAE reporting form within 24 hours of the occurrence.
8. In the events of any protocol amendments, TIREC must be informed and the amendments should be highlighted in clear terms as follows:
   a. The exact alteration/amendment should be specified and indicated where the amendment occurred in The original project. (Page no. Clause no. etc.)
   b. The PI must comment how proposed amendment will affect the ongoing trial. Alteration in the budgetary status, staff requirement should be clearly indicated and The revised budget form should be submitted.
   c. If the amendments require a change in the consent form, the copy of revised Consent Form should be submitted to Ethics Committee for approval. If the amendment demands a re-look at the toxicity or side effects to patients, The same should be documented.
   d. If there are any amendments in The trial design, These must be incorporated in the protocol, and other study documents. These revised documents should be submitted for approval of The IEC, only then can they be implemented.
   e. Approval for amendment changes must be obtained prior to implementation of changes.
   f. The amendment is unlikely to be approved by the IEC unless all the above information is provided.
   g. Any deviation/violation/waiver in The protocol must be informed.

STANDS APPROVED UNDER SEAL

Dr.K.Shantaraman, MD
Registrar, TIREC
Tirunelveli Medical College, Tirunelveli – 627011
State of Tamilnadu, South India

Dr.J.SureshDurai, MD
Member Secretary, TIREC
Tirunelveli Medical College, Tirunelveli – 627011
State of Tamilnadu, South India

CERTIFICATE – II

This is to certify that this dissertation titled "**PERFUSION INDEX DERIVED FROM PULSE OXIMETER AS A PREDICTOR OF HYPOTENSION FOLLOWING SPINAL ANAESTHESIA IN ELECTIVE CAESAREAN DELIVERY"** of the candidate **Dr.YATHISH.V** with registration Number **201720309** for the award of **M.D.** Degree in the branch of **ANAESTHESIOLOGY (X).** I personally verified the urkund.com website for the purpose of plagiarism check. I found that the uploaded thesis file contains from introduction to conclusion page and result shows **17 percentage** of plagiarism in the dissertation.

Guide & Supervisor sign with Seal.

URKUND

Urkund Analysis Result

Analysed Document:	yathish thesis.docx (D57241372)
Submitted:	10/18/2019 2:19:00 PM
Submitted By:	yathishyathi7@gmail.com
Significance:	17 %

Sources included in the report:

https://www.ncbi.nlm.nih.gov/pubmed/23518802
https://www.researchgate.net/publication/301234580_Effect_of_granisetron_in_attenuation_of_hypotension_following_spinal_anaesthesia_in_parturients_undergoing_elective_caesarean_section_-_a_double_blind_randomized_controlled_trial
https://www.researchgate.net/publication/320430312_Pleth_variability_index_can_predict_spinal_anaesthesia-induced_hypotension_in_patients_undergoing_caesarean_delivery
https://www.researchgate.net/publication/321775950_Perfusion_index_as_a_predictor_of_hypotension_following_propofol_induction_-_A_prospective_observational_study
https://www.researchgate.net/publication/319072032_Perfusion_index_as_a_predictor_of_hypotension_following_spinal_anaesthesia_in_lower_segment_caesarean_section/fulltext/598e6338a6fdcc10d8ebbe20/319072032_Perfusion_index_as_a_predictor_of_hypotension_following_spinal_anaesthesia_in_lower_segment_caesarean_section.pdf

CONTENTS

INTRODUCTION

Spinal anaesthesia leads to hypotension during Caesarean delivery, which is due to the result of combination of decreased vascular resistance due to sympathetic blockade and decreased cardiac output due to blood pooling in blocked areas of the body.

Baseline volume status is already known to affect the degree of hypotension, but baseline peripheral vascular tone may also have significant influence. Peripheral vascular tone has been found to be decreased in parturients at term, especially in those who are multiparous. This decrease in peripheral vascular tone leads to trapping of blood volume in the extremities even before spinal anaesthesia, and the sympathetic blockade due to spinal anaesthesia will further increase the blood pooling. Therefore, parturients with low baseline vascular tone may be at an increased risk of developing hypotension after spinal anaesthesia.

Non‑invasive blood pressure(NIBP) measurement is used as standard method of monitoring intraoperative and post operative haemodynamics. But the limitation is that, beat to beat variation is not measured by this method.

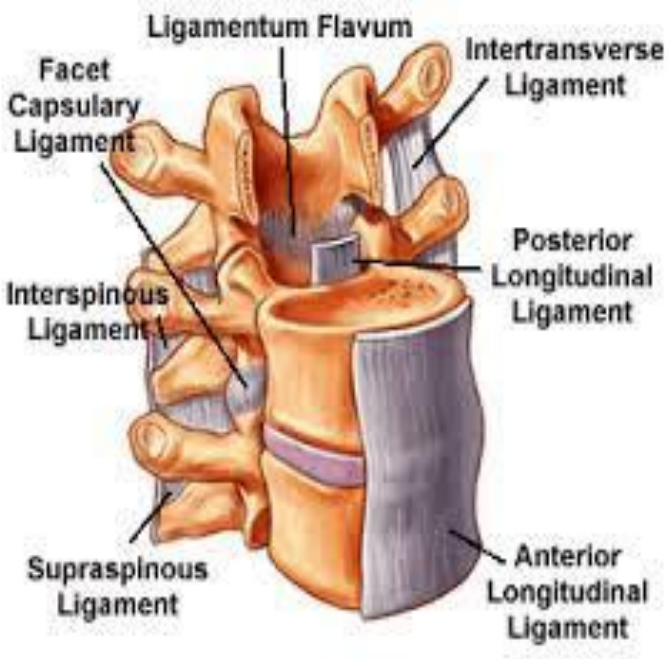
Perfusion index is nothing but the ratio of pulsatile blood flow and non-pulsatile component of blood in the peripheral tissue. This can be used to assess the peripheral perfusion dynamics that are caused due to changes in peripheral vascular tone.

AIMS AND OBJECTIVES:

1. To find out whether baseline Perfusion index can be used to predict hypotension following spinal anaesthesia for the elective lower segment caesarean delivery.
2. To find out the correlation between baseline perfusion index and incidence of hypotension following Spinal anaesthesia in parturients undergoing elective lower segment caesarean delivery.

ANATOMY OF SPINE

The skeletal framework of spine is formed by vertebral bones and fibrocartilaginous intervertebral disc with 7 cervical, 12 thoracic, 5 lumbar, 5fused sacral and 4 fused coccyx vertebra. Spinal cord is enclosed within the vertebral column and it is continuous cephalad with brainstem through foramen magnum and terminates upto the level of L1 in adults. The spinal cord tapers into conus medullaris from which filum terminale arises to attach to coccyx.



There are four synovial joints in each vertebra, each pair articulates with vertebra above and below. Anteriorly, the vertebral bodies are supported by anterior and posterior longitudinal ligaments. Posteriorly, the spinal cord is supported by ligamentum flavum, suprasinous ligament and

interspinous ligament, through which the spinal and epidural needle enters the interlaminar space and pierce the meningeal layer to reach the subarachnoid and epidural space respectively.

The lower spinal nerve roots traverse some distance and exit the intervertebral foramen, as the spinal cord ends at L1. It is called as cauda equina. From inner to outer, the spinal cord is surrounded by, piamater, arachnoid mater and duramater.

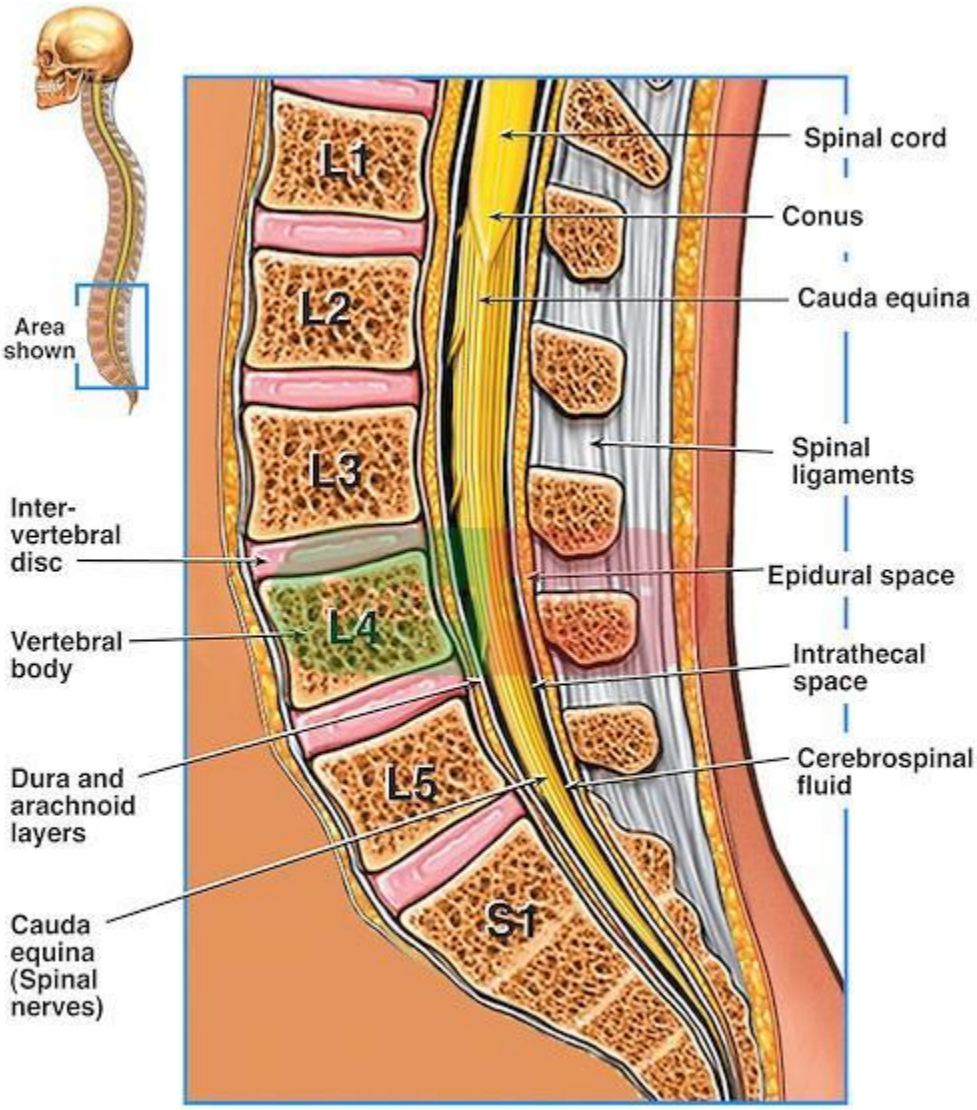
Duramater extends from foramen magnum to S2. Epidural space lies outside duramater, while subarachnoid space is between arachnoid and pia mater. Subdural space is present between dura and arachnoid mater.

Blood supply :

Blood supply to spinal cord is from a single anterior spinal artery and two posterior spinal arteries.

The anterior spinal artery arises from vertebral artery at base of the skull and course down along the anterior surface of the cord. It supplies the anterior two thirds of the cord, whereas posterior spinal arteries supply the posterior one-third.

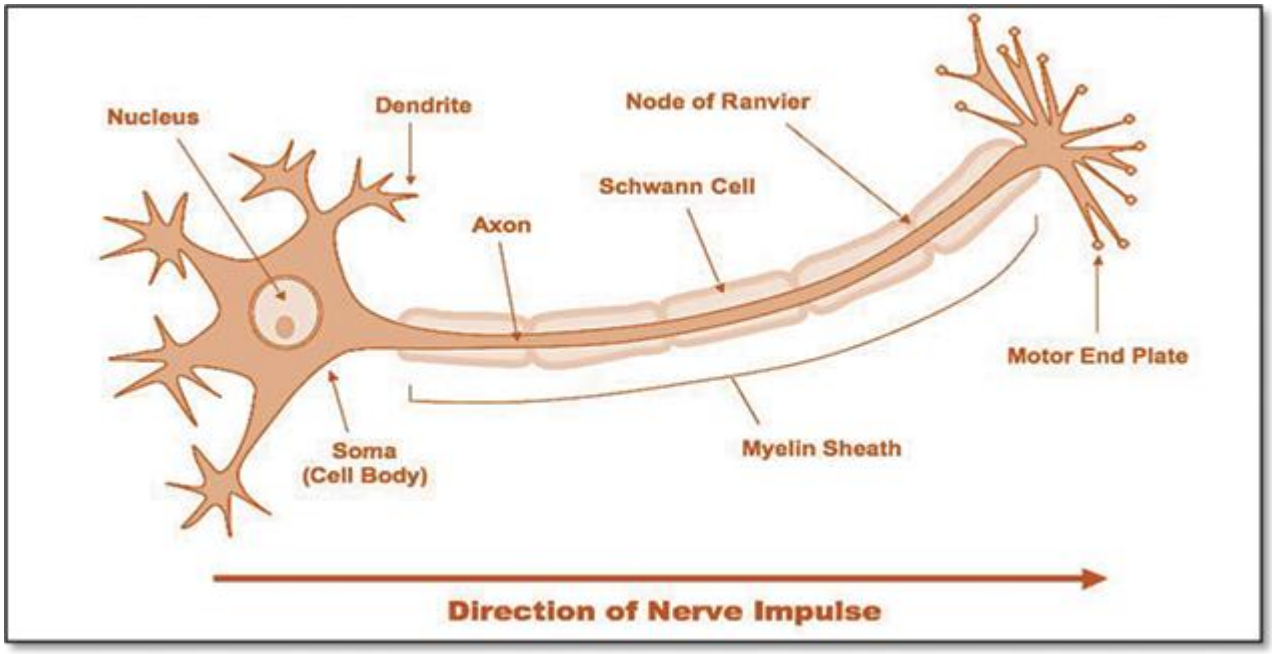
The posterior spinal arteries comes from the posterior inferior cerebellar arteries and course down along the dorsal surface of the cord medial to the dorsal nerve roots.



Cut-away view of spine

NERVE ANATOMY

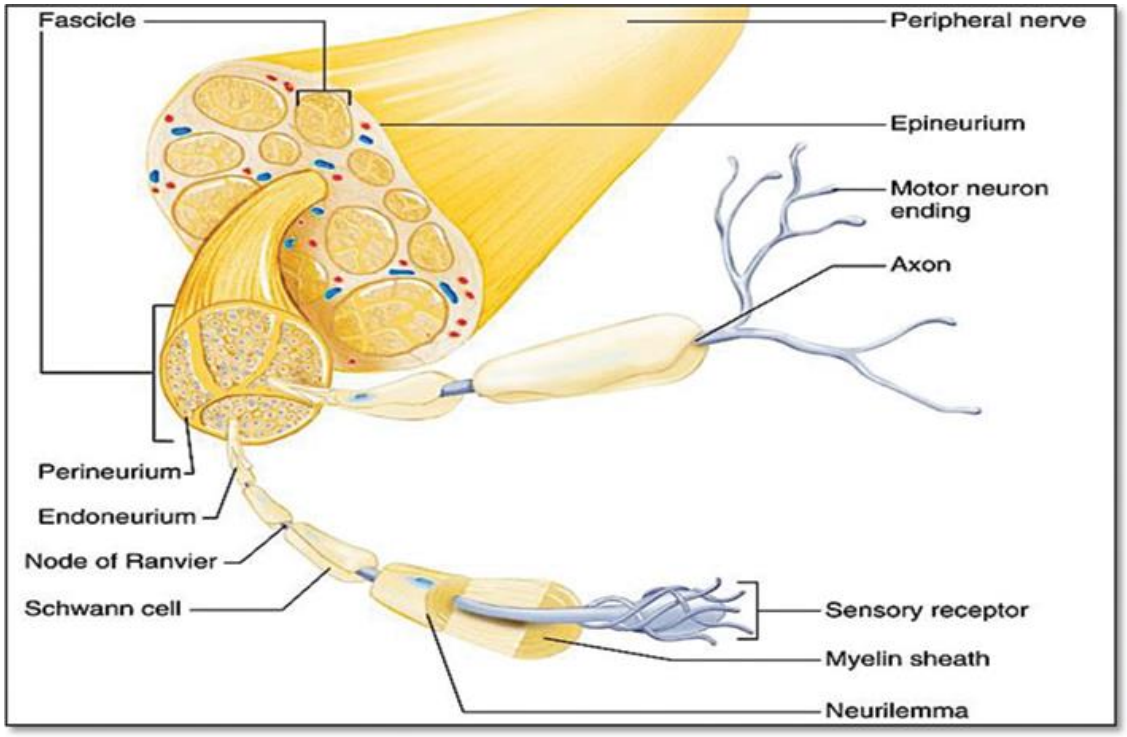
Neurons are the primary cells in the nervous system. The nervous system is made up of the central and peripheral nervous system. It can also be looked at in terms of parasympathetic and sympathetic nervous system. A group of neurons bundled together make up peripheral nerves.



Peripheral nerves contain both afferent and efferent fibres, which are bundled into one or more fascicles. Individual nerve fibres within the fascicle are surrounded by a layer of loose connective tissue called the endoneurium. The endoneurium houses the glial cells, fibroblasts and blood vessel capillaries, all of which are integral to the function of the nerve fibre. The fascicle is in turn surrounded by a dense layer of collagenous connective tissue called the perineurium.

A cylindrical sheath called the epineurium forms the outermost layer of a peripheral nerve. The main function of these layers is to protect

the nerve fibres and also act as barriers to agents acting on the nerves including local anaesthetics.



ELECTROPHYSIOLOGY OF NERVE CONDUCTION

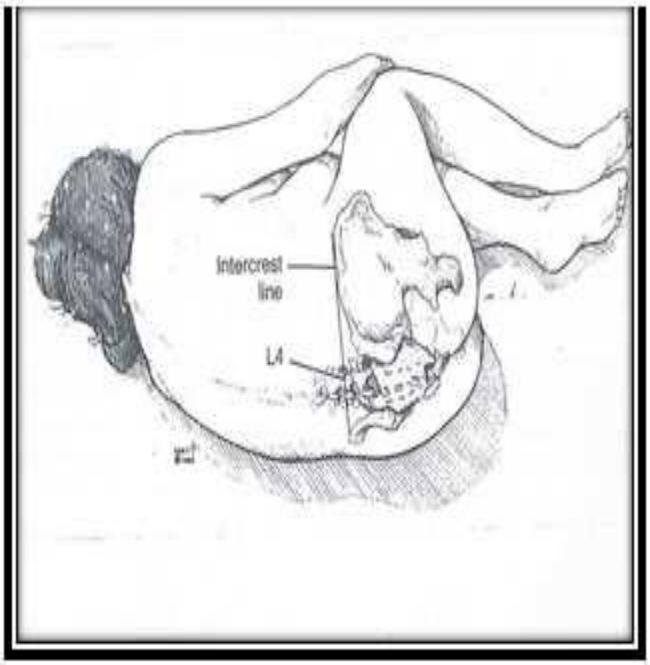
The resting membrane potential of a nerve cell is in the range of −60 to −70 mV. At rest, neurons are more permeable to potassium ions due to the presence of potassium leak channels. This explains why the resting neuronal membrane potential is closer to the equilibrium potential of potassium of −80 mV. The ionic disequilibria acts as the energy needed for propagation of action potentials on the cell surface The intracellular milieu of the nerve cell is negatively charged relative to the extracellular. Upon excitation of the nerve fibres, the electrical impulse propagates along the axon as a result of changes occurring in the adjacent membrane alternating from negative to positive values of about +50 mV due to rapid influx of sodium ions. At an electrical potential of +50 mV, there is rapid efflux of potassium ions in an attempt to maintain electrical neutrality of the cell. To restore the resting membrane potential, the sodium/potassium ATPase pumps sodium extracellularly, while the opposite happens to the potassium ions. The conduction of impulses along nerve fibres occurs as small brief, localised spikes of depolarisation on the surface of the cell membrane. Impulses travel in one direction as the axonal membrane that has just undergone depolarisation remains in the refractory state until the resting potential is restored by the Sodium/Potassium ATPass pumps.

ANATOMICAL CHANGES OF PREGNANCYAFFECTING REGIONAL ANAESTHESIA

Perivertebral ligaments including ligaventum flavum becomes soft and hypodense during pregnancy and will make the appreciation of passage of the epidural needle difficult. Due to exaggeration of physiological lumbar lordosis in pregnant women, it is difficult to achieve maximum lumbar flexion during positioning for regional blockade administration.

Changes noted in the vertebral column:

1) There is reduction of thoracic kyphosis with shifting caudal of apex of lumbar lordosis, making the spread of intrathecal local anaesthetic solutions unpredictable in supine posture.
2) Rotation of the pelvis on the long axis of the vertebral column shifting the imaginary line joining the iliac crest cephalad to the vertebral column.
3) Narrowing of the interspinous space in the lumbar region may make the administration of neuraxial technique difficult.



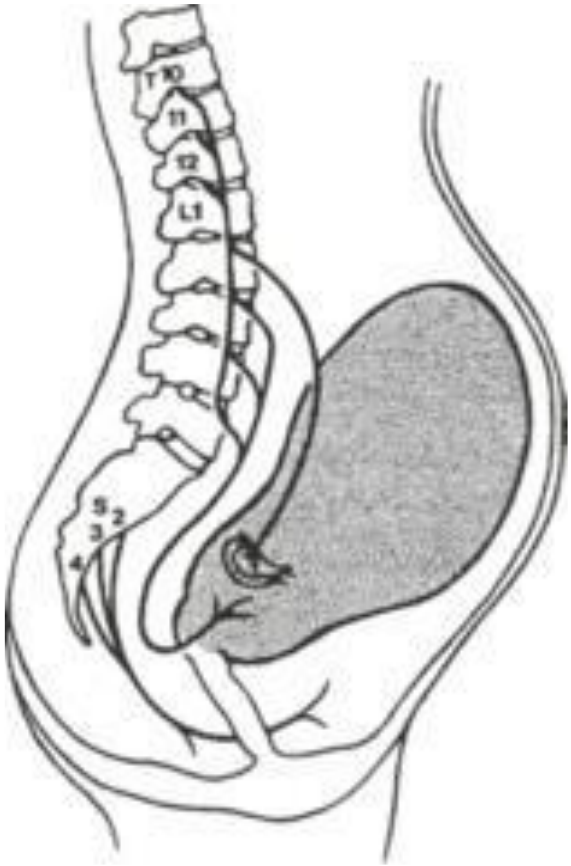
Following structural changes are noted in the spinal cord during pregnancy:

1. Epidural veins are engorged due to Inferior vena cava compression by the gravid uterus and may cause accidental intravascular injection of local anaesthetic during epidural administration.
2. Enlarged epidural space reduces the subarachnoid space volume, thus reducing the requirement of local anaesthetic during spinal anaesthesia.
3. Low specific gravity of Cerebrospinal fluid also alters the spinal local anaesthetic requirement.

PHYSIOLOGY

Uterine contractions during the first stage of labor resulting in myometrial ischemia releases histamine, bradykinin, serotonin.

Mechanoreceptors are stimulated by stretching and distension of lower uterine segment and cervix. These two noxious impulses are carried by sensory nerve fibers accompanying sympathetic nerve endings entering the spinal cord at T10 to L1 spinal segments. Stretching of the perineum at second stage of labor are carried by pudendal nerve to S2 to S4 spinal segments. Even though the incision in lower segment caesarean segment is below the umbilicus, sensory level of blockade of T4 to T5 is required for a painless caesarean delivery.



Physiology of neuraxial blockade:

Local anaesthetic agents block sodium channels along nerve membrane producing nerve blockade. Differential block is noted with spinal rather than with epidural block due to direction action on nerve fibres by local anaesthetic in spinal anaesthesia. Block regression is explained by uptake of local anaesthetic by blood vessels in subarachnoid space and spinal cord.

Systemic effects of regional anaesthesia:

1. RESPIRATORY FUNCTION:

In parturients, the functional residual capacity(FRC) is reduced due to cephalad movement of diaphragm by increased intra abdominal pressure and which pose a risk of hypoxemia. Supine posture after regional anaesthesia causes decrease in the FRC , increase in minute ventilation and oxygen consumption, hence they are easily prone for hypoxemia.

External intercostal muscle paralysis seen in high spinal does not affect respiration. Abdominal muscle paralysis during regional anaesthesia decreases peak expiratory flow rate and coughing ability.

2. CARDIOVASCULAR SYSTEM:

Supine hypotension syndrome in pregnancy is a major concern in regional anaesthesia, which becomes severe after regional anaesthesia. It can be prevented by adequate preloading, wedge placement underneath the right buttock region. Aortocaval compression leads to shunting of blood

through intraosseous vertebral veins, paravertebral and epidural venous plexus, which reduce the subarachnoid space volume secondary to increased epidural pressure. This compression is present as early as 13 to 16 weeks of gestation and reaches maximum by term.

In early trimester, the systolic blood pressure falls due to aortic dilation and diastolic fall is due to reduced vascular resistance. But the blood pressure is maintained by the increased sympathetic drive which is cut off by regional anaesthesia, which leads to exaggerated fall of blood pressure than non-parturients undergoing regional anaesthesia.

3. OTHER SYSTEMS:

In the GIT, the gravid uterus shifts the stomach cephalad altering the gastro esophageal junction and the circulating progesterone reduces the lower esophageal sphincter tone., placing the parturient at high risk of aspiration of gastric contents.

4. UTERINE BLOOD FLOW AND REGIONAL ANAESTHESIA:

Pain, stress and hyperventilation decreases uterine blood flow by sympathetically mediated release of norepinephrine and epinephrine. This leads to abnormal fetal heart rate patterns. Pain relief by regional anaesthesia decreases these catecholamines and thereby increases uterine blood flow.

5. OXYGEN CONSUMPTION:

30% to 40% increase in oxygen consumption during pregnancy which is accompanied by parallel increase in carbondioxide production. This is due to increased metabolic requirement by gravid uterus, fetus, placenta and increased cardiac output. Hence oxygen supplementation is a must during regional anaesthesia.

SPINAL ANAESTHESIA

Definition:

Spinal anaesthesia is a form of regional anaesthesia obtained by blocking the spinal nerves in the sub arachnoid space by injecting local anaesthetic solution in to CSF, which mainly act on the spinal nerve roots.

HISTORY:

- 1885 - J.C corning administered cocaine intrathecally for pain
- 1891 - Heinrich Irenaeus Quincke demonstrated technique of lumbar puncture for diagnostic purpose.
- 1898 – August Bier of Germany produced true spinal anaesthesia in man
- 1900 – Rudalph matas pioneer in spinal opioids
- 1905 – Pitkin popularized the method of introducing agents intrathecally.
- 1908 – Baker described the use of dextrose to increase, alcohol to decrease the density of local anaesthetic solution

Sites of action in order of importance are

1. Primarily on the spinal cord nerve roots.

2. Secondarily act on dorsal root ganglia and postero-anterior horn synapse.

Order of nerve block

1. Autonomic preganglionic b fibres

2. Temperature – cold then warmth is lost

3. Temperature discrimination is lost

4. Slow pain followed by fast pain

5. Tactile sense

6. Motor blockade – extensors then flexors

7. Pressure sense lost

8.Proprioception lost

Anaesthetic dose requirements

Pregnant women exhibit a more rapid onset and a longer duration of spinal anaesthesia than non-pregnant due to enhanced neural sensitivity to local anaesthetics.

Dose of hyperbaric local anaesthetic requirement in term pregnant is 25% lower than in non-pregnant is due to

i. reduction of the spinal CSF volume, which accompanies the distention of vertebral venous plexus;
ii. enhancement of neural susceptibility to the local anaesthetics;
iii. increase in rostral spread, caused by the widening of the pelvis;
iv. inward displacement of intervertebral foraminal tissue, due to increased abdominal pressure;
v. a higher level of the apex of the thoracic kyphosis during late pregnancy

PULSE OXIMETER

Pulse oximetry is a simple, non-invasive, reliable, reasonably accurate, cheap, continuous and risk free method of measuring arterial oxygen saturation in all patient age groups.

Spectrophotometry It is based on Beer-Lambert's law, a combination of two laws describing absorption of monochromatic light by a transparent substance through which it passes.

*Beer's Law :*The intensity of transmitted light decreases exponentially as the concentration of the substance increases. [August Beer, German Physicist (1825–1863)]

Lambert's Law: The intensity of transmitted light decreases exponentially as the distance travelled through the substance increases. [Johann Lambert, German Physicist (1728–1777)]

The Beer-Lambert's law is expressed as the following equation:

$Ie = Io \times e^{-DCa}$, Where,

Ie - intensity of transmitted light,

Io - intensity of the incident light

D - distance that the light is transmitted through the medium,

C - concentration of the solute (hemoglobin),

a - extinction coefficient, it is a constant for a given solute at a specified wavelength

e - base of natural logarithms (approximately 2.7182818285)

Oxyhaemoglobin absorbs more infrared light (wavelength of 940 nm) than red light (wavelength of 660 nm) and deoxyhaemoglobin absorbs more red light than infrared light. Isosbestic point is the point at which two substances absorb a certain wavelength of light to the same extent. This point may be used as reference points where light absorption is independent of the degree of saturation.

Pulse oximeters use a type of light source called “light emitting diodes (LEDs)” as they are cheap, very compact, emit light in accurate wavelengths, do not heat up much during use and hence less likely to cause burns.

Conventional pulse oximeters have two LEDs that emit light in the red light (660 nm) and infrared light (940 nm) wavelengths. Emission of these two wavelengths alternates at frequencies of 0.6–1.0 kHz, and the non-absorbed energy is detected by a photodetector. Depending on the amounts of oxyhaemoglobin and deoxyhaemoglobin present, the ratio of the amount of red light absorbed compared to the amount of infrared light absorbed changes.

Using this ratio, the pulse oximeter can then work out the oxygen saturation (SpO2). The pulse oximeter needs to analyze only arterial blood, ignoring the other tissues around the blood. Principle of plethysmography is used for this

Plethysmography : During each cardiac cycle, light absorption by tissue beds varies cyclically. During “diastole”, absorption is caused by non-pulsatile arterial blood, venous blood, tissue, bone, and pigments [direct current (DC) component].

During “systole”, the pulsatile expansion of the arteriolar bed produces an increase in path length thereby increasing the absorbance [alternating current (AC) component]. The microprocessor first determines the AC component of absorbance at each wavelength and divides this by the corresponding DC component. SpO2 determines the red:infrared absorption ratio.

Thus, the red:infrared ratio of these pulsatile differences can be used to compute the pulse oximeter reading (SpO2), which is an estimate of arterial oxygen saturation (SaO2).

The measured ratio is compared with stored ones in the microprocessor of the device and corresponding SpO2 is displayed. These algorithms are derived through SaO2 measurements in healthy volunteers breathing mixtures of decreasing oxygen concentrations and are usually unique for each manufacturer. The displayed SpO2 represents the mean of the measurements obtained during the previous 3–6 seconds, whereas the data are updated every 0.5–1.0 second.

The performance of each device is based on the reliability and complexity of the algorithms used in signal processing and to the speed and quality of the microprocessor.

HISTORY AND EVOLUTION OF PERFUSION INDEX(PI)

In 1935, Karl Matthes (German physician 1905–1962) developed the first 2-wavelength ear O2 saturation meter with red and green filters (later switched to red and infrared filters). His meter was the first device to measure O2 saturation.

The original oximeter was made by Glenn Allan Millikan in the 1940s.In 1949 Wood added a pressure capsule to squeeze blood out of ear to obtain zero setting in an effort to obtain absolute O2 saturation value when blood was re-admitted. The concept was similar to today's conventional pulse oximetry but was difficult to implement because of unstable photocells and light sources. This method is not used clinically. In 1964 Shaw assembled the first absolute reading ear oximeter by using eight wavelengths of light.

Pulse oximetry was developed in 1972, by Takuo Aoyagi and Michio Kishi, bioengineers, at Nihon Kohden using the ratio of red to infrared light absorption of pulsating components at the measuring site. Susumu Nakajima, a surgeon, and his associates first tested the device in patients, reporting it in 1975. It was commercialized by Biox in 1981 and Nellcor in 1983.

Biox was founded in 1979, and introduced the first pulse oximeter to commercial distribution in 1981. Prior to the introduction of pulse oximetry, a patient's oxygenation could only be determined by arterial blood gas, a single-point measurement that takes several minutes for sample collection and processing by a laboratory. In the absence of oxygenation, damage to the brain starts within 5 minutes with brain death ensuing within another 10–15 minutes.

With the introduction of pulse oximetry, a non-invasive, continuous measure of patient's oxygenation was possible, revolutionizing the practice of anesthesia and greatly improving patient safety.

In 1995 Masimo introduced perfusion index, quantifying the amplitude of the peripheral plethysmograph waveform.

Perfusion index has been shown to help clinicians:

1) To predict illness severity and early adverse respiratory outcomes in neonates
2) To provide an early indicator of sympathectomy after epidural anaesthesia, and improve detection of critical congenital heart disease in newborns.

In 2007, Masimo introduced the first measurement of the pleth variability index (PVI), which multiple clinical studies have shown provides a new method for automatic, non-invasive assessment of a patient's ability to respond to fluid administration. Appropriate fluid levels are vital to reducing postoperative risks and improving patient outcomes.

PERFUSION INDEX

Perfusion Index or P.I. is the ratio of the pulsatile component of the blood flow to the non-pulsatile static component of the blood flow in a patient's peripheral tissue, such as finger tip, ear lobe, or toe. Perfusion index indicates the pulse strength at the sensor site.

Range - 0.02% (extremely weak pulse) to 20% (very strong pulse)

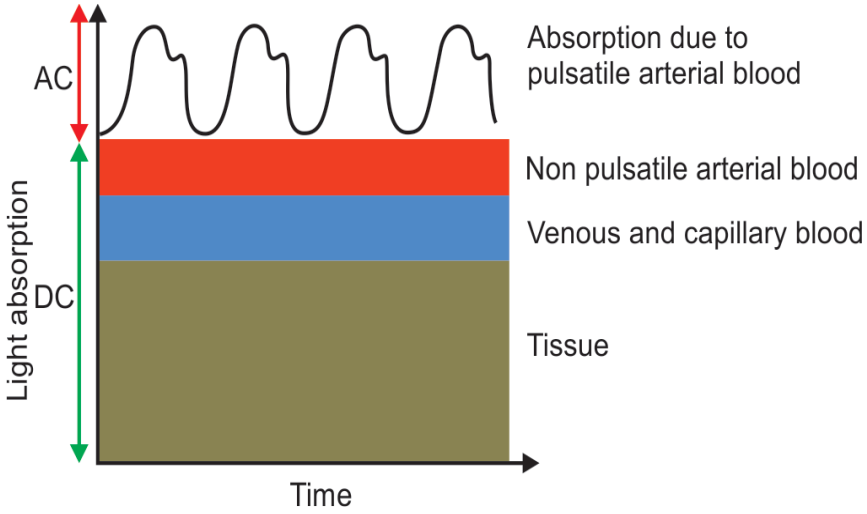
The ratio of AC (pulsatile) component to DC (non-pulsatile) components of the infrared signal correspond to the pulsatile and the non-pulsatile amounts of blood in the sensor type. The relationship of the pulsatile to the non-pulsatile amounts of blood at any particular site corresponds to PI at that particular site.

The perfusion index varies depending on patients, physiological conditions, and the site of monitoring. Because of this variability, each patient should establish his own "normal" perfusion index for a given location and use this for monitoring purpose.

Perfusion index is normally monitored with pulse oximeters. It is also a good indicator of the reliability of the pulse oximeter reading.

For most pulse oximeters for general use, the reading is unreliable or unavailable if PI is at or below 0.4%. There are oximeters, such as those from Masimo, which are designed for extreme low PI.

$$\text{Perfusion index} = \frac{\text{Pulsatile signals of infrared}}{\text{Non pulsatile signals of infrared}} \times 100$$



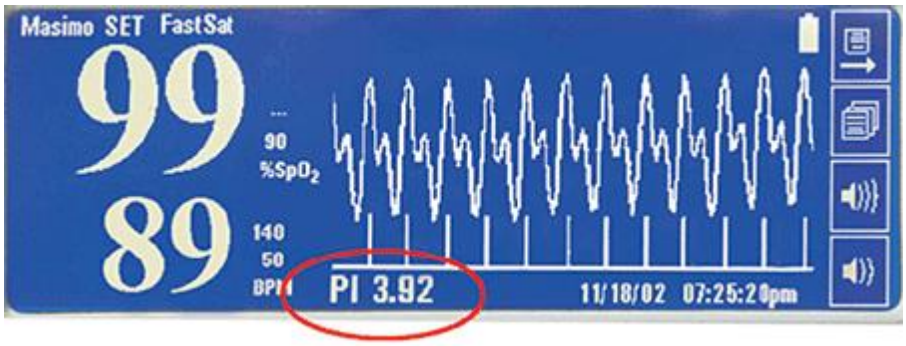
Perfusion index is expressed in percentage. Most people that use an oximeter at home would not need a perfusion index indicator because they are considered to be in general good health. A perfusion index adds a lot of sensitivity to the oximeter sensors thus adding to the cost of the oximeter.

The pleth (Plethysmograph), available in many pulse oximeters, is a graphical representation of the perfusion index. In a hospital, perfusion index, along with many other parameters, is used to monitor critically ill patients. Studies have shown that PI has a high correlation with capillary refill time and central-to-toe temperature difference. In neonatal acute care, a low P.I. is an objective and accurate measure of acute illness. It is superior to qualitative approach such as foot warmth.

Perfusion index is also used as an early warning of anaesthetic failure. Studies have shown that an increase in PI is an early indicator that epidural or general anaesthesia has initiated peripheral blood vessel dilation, which typically occurs before the onset of anaesthesia. Lacking in the spike would also help to identify the lack of anaesthetic effect.

As we learn more about PI, more clinical applications are being discovered. To make informed patient management decision, physicians often need to be aware of changes in perfusion index, peripheral perfusion and circulatory status. This is especially true in patients who are in critical conditions or who are anaesthetized, undergoing surgery or in labour.

Perfusion index also represents a non-invasive measure of peripheral perfusion that is obtained continuously from a pulse oximeter



Clinical Interpretation of the Perfusion Index

The PI value is relative to a particular monitoring site, (eg. the fingertip or toe), of each patient as physiological conditions vary between monitoring sites and individual patients at the time of monitoring.

Due to local vasoconstriction or vasodilatation in the skin at the monitoring site, there can be changes in PI. It is decreased in vasoconstriction and increased in vasodilatation. This is due to changes in the volume of oxygenated blood flow in the microcapillaries of the skin. The measurement of P.I. is independent of other physiological variables such as, temperature, oxygen consumption, heart rate variability or arterial oxygen saturation.

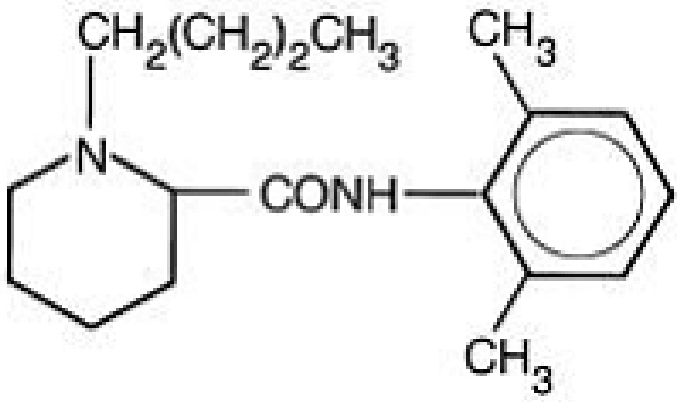
The PI generally changes in proportion to peripheral perfusion. In certain instances, however, such as in a patient attached to a heart-lung machine, perfusion can be good but the pulsatile part of the signal is nearly zero because of the absence of a pulse. Even in such an instance, the monitoring of P.I. in conjunction with examining the photoplethysmogram (pleth) waveform, can give the clinician an indication of the accuracy of the saturation readings.

Choosing a Monitoring Site in Adults

The fingertip is the standard monitoring site for pulse oximetry. The hand or foot (sometimes toe) is often used in neonatal patients. Surgical patients, however, are subject to unpredictable changes in peripheral perfusion, particularly with a large degree of variability in body temperature. These changes in peripheral perfusion may have variable effects at different sensor locations. Thus an alternative to the standard fingertip sensor site will be useful.

PHARMACOLOGY OF BUPIVACAINE

Bupivacaine (MARCAINE, SENSORCAINE), is a widely used amide local anaesthetic; its structure is similar to that of lidocaine except that the amine-containing group is a butyl piperidine Bupivacaine is an amino amide local anaesthetic with a slow onset. It is long acting and suitable for procedures lasting 2 – 2.5 hrs. It was first synthesized by Ekenstam in 1957 and was used clinically in 1963.



It is available as hyperbaric solution in concentrations of 0.5% and 0.75% with dextrose 8.25%. Available Isobaric solutions are in concentrations of 0.5% and 0.75%. Maximum dose is 2mg/kg body weight.

PHYSICO-CHEMICAL PROFILE.

Molecular weight(base)	288
pKa	8.16
Solubility in Alcohol	1 in 8
Solubility in Water	1 in 25
Octonol/water partition coefficient	High
Lipid solubility	28
Plasma protein binding	95%
Anaesthetic index	3.0-4.0

PHARMACODYNAMICS

Bupivacaine by virtue of its pharmacological effects, has a stabilizing action on all excitable membranes. In the central nervous system, stimulation can occur producing restlessness, tremors and convulsions in over dosage. Bupivacaine can also causes a reduction of automaticity in the heart.

PHARMACOKINETICS ABSORPTION

The absorption depends on:

- Site of injection(intercostals>caudal>epidural>brachial plexus > subcutaneous)
- Dose- the peak blood concentration increases with increase in dose.
- Presence of vasoconstrictors-delays absorption.

DISTRIBUTION

Bupivacaine is 95% protein bound to albumin and alpha-1 acid glycoprotein.

METABOLISM

Occurs in liver by N-dealkylation, primarily to pipecolyxylidine. N-desbutyl bupivacaine and 4-hydroxy bipivacaine are the other metabolites produced.

EXCRETION

Excretion is through urine(5% as pipecolyxylidine and 16% as unchanged form).The clearance is 0.47 l/min and elimination half life is 62 mins.

EFFECTS IN CVS

It has marked cardiotoxic properties. It can bind to myocardial proteins and thus decreases the rate of increase of phase 0 during the cardiac action potential. In higher concentration, the peripheral vascular resistance and myocardial contractility are reduced and this can lead to cardiovascular collapse.

EFFECTS IN CNS

In CNS it causes reversible neural blockade. It has characteristic biphasic effect in CNS. Initial excitation is caused by inhibition of inhibitory

interneuron pathways in cortex. In higher doses both facilitatory and inhibitory pathways are depressed.

ADVERSE REACTIONS

CNS

Excitation characterized by restlessness, anxiety, dizziness, tinnitus, blurred vision or tremors proceeding to convulsions, followed by rowsiness, unconsciousness and cardiac arrest.

CVS

Cardiotoxicity effects are due to high lipid solubility and high protein binding properties of the drug. Accidental intravenous injection causes dysrhythmias, atrioventricular block, ventricular tachycardia and ventricular fibrillation.

ALLERGY

Allergy is extremely rare. It manifests as urticaria, pruritis, angioneuroticedema etc.

ANESTHESIA FOR CESAREAN DELIVERY

Preanesthetic Evaluation:

1) Presence of any co-existing diseases, previous history of any surgery, obstetrics history, any drug allergies, and measurement of baseline heart rate and blood pressure.
2) Airway evaluation, examination of spine, cardiovascular and respiratory system examination.

Informed and written Consent

Monitoring:

• Pulse oximetry

•Electrocardiogram(ECG)

•Non-invasive blood pressure(NIBP)

• Oxygen sensors and volatile agent analyzer

SELECTION OF ANESTHETIC TECHNIQUE FOR CESAREAN DELIVERY

Neuraxial Anesthesia

• Mother's desire to witness child birth

• High risk with difficult airway

• Presence of comorbid conditions which could be exaggerated by other techniques

• General anaesthesia intolerance or failure

• Other benefits :

Post operative analgesia after surgery

Lower fetal drug exposure

Less blood loss

Allows presence of husband or support person .

General Anesthesia

• Patient refusal

• Contraindications for regional techniques

• Insufficient time to induce neuraxial anesthesia for emergency delivery

• Failure of regional techniques

• Fetal problems

NeuraxialAnesthetic Techniques for Cesarean Delivery

SINGLE-SHOT SPINAL

Advantages :

• Simple technique

• Lower doses of the local anesthetic and opioid required

• Rapid onset of action

Disadvantages :

• Duration of anaesthesia is limited

• Limited ability to titrate extent of sensory blockade

EPIDURAL ANAESTHESIA

Advantages :

• No dural puncture required, so chances of PDPH is limited

• In situ catheter placed can be used for earlier administration of labor analgesia

• Extent of sensory blockade can be titrated

• Continuous intraoperative anaesthesia and postoperative analgesia

Disadvantages :

• Technically Difficult

• Slower onset of anaesthesia

• Larger volume and dosage of drugs required

• Higher risks for maternal systemic toxicity

COMBINED SPINAL-EPIDURAL ANAESTHESIA

Advantages :

• May be technically easier than spinal anaesthesia in obese patients

• Low doses of local anaesthetic and opioid required

• Rapid onset of action

• Ability to titrate extent of sensory blockade

• Continuous intraoperative anaesthesia and postoperative analgesia

Disadvantages :

• Delayed verification of functioning epidural catheter

CONTINUOUS SPINAL ANAESTHESIA

Advantages :

• Rapid onset of dense anaesthesia

• Low doses of local anaesthetic and opioid required

• Ability to titrate extent of sensory blockade

• Continuous intraoperative anaesthesia

Disadvantages :

• Greater risk for post–dural puncture headache

•Chances of overdose and total spinal anaesthesia if the spinal catheter is mistaken for an epidural catheter

SPINAL ANAESTHESIA

Spinal anaesthesia is now the most commonly used anaesthetic technique for caesarean delivery. Spinal anaesthesia is a simple and reliable technique, technically easier to perform and provides rapid onset of dense neural blockade that is typically more profound, resulting in a reduced need for supplemental intravenous analgesics or conversion to general anaesthesia.

Only a small dosage of local anaesthetic is required to establish a functional spinal blockade; thus, spinal anaesthesia is associated with trivial maternal risk for systemic local anaesthetic toxicity and with minimal drug transfer to the fetus. Spinal anaesthesia is associated with

predictable and relatively prompt recovery that enables patients to quickly transition through the post anaesthesia care unit.

Contraindications :

•Patient refusal

•Elevated Intracranial pressure

•Skin or soft tissue infection at the site of needle entry

•Coagulopathy

•Maternal hypovolemia

COMPLICATIONS OF SPINAL ANAESTHESIA

•Hypotension

• Bradycardia

•Dyspnea

•Failure of Blockade

•High Blockade

•Nausea and Vomiting

•Hypothermia and Shivering

• Local infection

PATHOPHYSIOLOGY OF HYPOTENSION FOLLOWING SPINALANESTHESIA

Hypotension following spinal anaesthesia is mainly occurs due to :

- Marked decrease in systemic vascular resistance,
- The rate and extent of the sympathetic involvement— leading to peripheral vasodilatation and venous pooling of blood, and
- The onset and spread of the neuraxial blockade — determines the severity of hypotension.

Hypotension is a common sequel of neuraxial anaesthesia and, if severe and sustained, may lead to impairment of uteroplacental perfusion and result in fetal hypoxia, acidosis, and neonatal depression or injury.

Severe maternal hypotension can also have adverse maternal outcomes, including altered consciousness, pulmonary aspiration, apnoea, and cardiac arrest.

Definitions for maternal hypotension:

1) a decrease in systolic blood pressure of more than 20% to 30% from baseline measurements or

2) a systolic blood pressure lower than 90 mm Hg or

3) a mean arterial pressure lower than 65 mm Hg

Prevention of Hypotension

Strategies that are used to prevent hypotension after spinal anaesthesia for caesarean delivery are

1) Fluid administration(preloading)

2) Vasopressor administration,

3) Leg elevation

4) Left uterine displacement

SPINAL ANAESTHESIA AND AUTONOMIC NERVOUS SYSTEM

Sympathetic Denervation occurs during spinal anaesthesia. In spinal anaesthesia the severity of hypotension depends on the degree of sympathetic blockade. The level of sympathetic denervation determines the magnitude of cardiovascular responses in spinal anaesthesia. As the level of blockade increases, more number of sympathetic fibres are blocked and greater the change in cardio-circulatory parameters are anticipated. But we often find a low level of blockade producing severe hypotension as the sympathetic blockade is highly variable. This variability is due to in the arborisation of autonomic fibres.

In partial sympathetic blockade, a reflex increase in sympathetic activity occurs in sympathetically intact areas leading to vasoconstriction which compensates for the peripheral vasodilation taking place in the sympathetically denervated areas.

Anaesthesia at or above T5 increases the risk of hypotension and bradycardia. In spinal anaesthesia, hypotension is defined as a systolic blood pressure<90 mm Hg or a reduction in mean arterial blood pressure<65mm of Hg. In addition, in pregnancy, the gravid uterus compresses on the aorta and inferior vena cava further augmenting the symptoms in supine position.

Prevention of hypotension caused by vasodilatation can be done by a prophylactic preloading infusion of colloid or crystalloid or by during the performance of the neuraxial block as co-loading, and with use of vasopressors

Bradycardia

Bradycardia occurs due to the blockade of the thoracic sympathetic fibers - preganglionic cardiac accelerator fibers originating at T1-T4, and also from reflex slowing of the heart rate induced by vasodilation related reduction in venous return to the right atrium. The stretch receptors respond by a compensatory slowing of the heart rate.

Vasopressor agents :

Ephedrine and Phenylephrine, in titrated doses are used to maintain maternal blood pressure. Greater doses of ephedrine provided more effective prophylaxis, but, hypotension was still observed and reactive hypertension and umbilical artery metabolic acidosis were found to be very common.

Phenylephrine crosses the placenta at a lower rate than compared to ephedrine. It also undergoes faster metabolism in fetus.

The combination of both intra venous fluid therapy and vasopressor administration might be the most effective regimen to prevent hypotension.

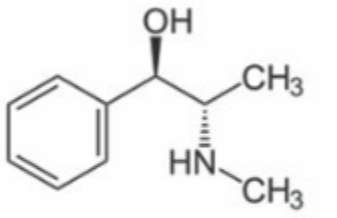
Physical methods to prevent hypotension includes :

*Use of lower limb compression bandages or pneumatic compression devices.

*Left uterine displacement.

EPHEDRINE

Ephedrine is an adrenergic amine present in many kinds of pharmaceutical preparations, obtained by synthesis or from natural sources Ephedrine is a sympathomimetic non-catecholamine drug with indirect action , stimulating the release of endogenous norepinephrine & direct action which stimulates both alpha and beta adrenergic receptors actions.



Ephedrine

Clinical Uses

a. Ephedrine — 5 to10mg IV in adults — increases systemic blood pressure in the presence of sympathetic nervous system blockade produced by regional anaesthesia or hypotension due to inhaled or injected anaesthetics.

b. In parturients with decreased systemic blood pressure due to spinal or epidural anaesthesia for treatment of maternal hypotension.

Cardiovascular effects

a. IV ephedrine leads to in increases in systolic and diastolic blood pressure, heart rate as well as cardiac output.

b. It increases myocardial contractility due to activation of beta1 receptors.

c. Tachyphylaxis

OXYTOCIN

Its a pituitary hormone secreted by the posterior pituitary gland.

Sources :

- Corpus luteum
- Placenta
- Synthetic pitocin

Dosage :

10 units by intramuscular route or 20-40 mUnit/min by Intravenous route are injected for post partum haemorrhage. 0.5-1 mUnit/min by intravenous route for the induction of labour.10-20 mUnit/min is administered along with other drugs for termination of pregnancy

Pharmacodynamics :

Uterine contractions are seen after 3-5 minutes and approximately 1 minute of administration through intramuscular and intravenous routes respectively. A steady state of the drug is reached after 40 minutes of parenteral route of administration. It is distributed throughout extracellular

fluid compartment of the mother; small amounts may cross the placental barrier and reach foetus.

Metabolism:

Rapidly via the liver and plasma by the enzyme oxytocinase a few steps of metabolism also takes place via mammary gland. It has a half-life of 1-5 minute. Kidney and liver help in the elimination of Oxytocin drugs unchanged form of this drug is rarely excreted in urine. Overdose can cause titanic uterine contractions, impaired blood flow to the uterus, uterine ruptures, seizures and amniotic fluid embolism contractions, impaired blood flow to the uterus, uterine ruptures, seizures and amniotic fluid embolism.

Contraindications :

Significant cephalopelvic disproportion Unfavourable foetal positions Obstetric emergencies which favours surgery Hyperactive or hypertonic uterus When vaginal delivery is contraindicated, Anaphylactic patients

Foetal distress, Polyhydramnios, Partial placenta previa, Elective labour induction

Side effects :

- Nausea or vomiting
- Memory problems or confusion
- Runny nose, sore throat, or coughing
- severe headaches
- hallucinations
- vomiting
- confusion
- Seizures and severe hypertension
- irregular heartbeats

Storage: The optimum temperature for storage of Oxytocin drugs is at 20-25 degree Celsius

REVIEW OF LITERATURE

Toyama et al conducted a study 2013 to examine whether baseline PI can be used predict the incidence of spinal anaesthesia-induced hypotension during Caesarean section.

The correlation between baseline PI and the degree of hypotension after giving spinal anaesthesia and also the predictive ability of spinal anaesthesia-induced hypotension during Caesarean delivery by Perfusion index were investigated. Its a prospective study in which parturients received 10mg of injection bupivacaine(hyperbaric) and 20µg of fentanyl.

Parturients undergoing Caesarean delivery January 2010 and March2011 were included in this study. 83 parturients underwent Caesarean delivery during this period; of which 39 parturients were enrolled in the study.

Baseline PI correlation was done with the degree of decreases in systolic and mean arterial pressure (r¼0.664, P,0.0001 and r¼0.491, P¼0.0029, respectively). The cut-off PI valueof 3.5 identified that parturients are at higher risk for spinal anaesthesia-induced hypotension with asensitivity of 81% and specificity of 86% (P,0.001).

The change of PI in parturients with baseline PI≤3.5 was not significant during the observational period, but PI in parturients with baseline PI.3.5 demonstrated marked decreases after SAB.

They demonstrated that high baseline PI will be associated with profound hypotension and that baseline PI could predict the incidence of spinal anaesthesia induced hypotension during Caesarean delivery.

Duggappa DR, et al conducted a study in 2017, to find out the correlation between baseline perfusion index and incidence of hypotension following Sub arachnoid block in lower segment caesarean section.

This was a prospective observational study in which 126 parturients were divided into two groups on the basis of their baseline PI. Group I included parturients with PI values ≤3.5 and Group II, parturients with PI >3.5. Spinal anaesthesia was performed with 10mg of 0.5% hyperbaric bupivacaine injection at L3–L4 or L2–L3 interspace. Mean arterial pressure <65 mmHg was considered as hypotension.

Incidence of hypotension in Group I was 10.5% and that of Group II was 71.42% ($P < 0.001$). It was found out that there is significant correlation between baseline PI >3.5 and number ofepisodes of hypotension and total dose of ephedrine required.

The sensitivity and specificity of baseline PI of 3.5 to predict hypotension was 69.84% and 89.29%, respectively. The area under the ROC curve for PI to predict hypotension was 0.848.

It was concluded that the Baseline perfusion index of >3.5 is associated with a higher incidence of hypotension following spinal anesthesia in elective LSCS.

M. Yokose et al conducted a study in 2015 to determine whether hypotension could be predicted by parameters derived from pulse oximeter, like perfusion index , heart rate, pleth variability index, ratio of low-frequency to high-frequency components of heart rate variability, and entropy of heart rate variability,which was measured before the induction of anaesthesia.

The predictive value of these parameters for detecting hypotension were assessed by using logistic regression and the grey zone approach in 81 parturients. Logistic regression revealed heart rate as the only independent predictor (OR 1.06; 95% CI 1.01–1.13; p = 0.032).The grey zone for heart rate was in the range of 71–89 bpm, and 60.5% of the parturients were found to be in the grey zone. They concluded that only pre-operative heart rate derived from pulse oximeter before giving anaesthesia may be used for predicting hypotension.

Sripada G Mehandale et al conducted a study in 2017, To find out whether Perfusion index can be used to predict hypotension following propofol induction and to determine the cut‑off value beyond which hypotension was more common. 50 adult patients belonging to ASA physical status I/II who were undergoing elective surgery under general anaesthesia were enrolled in this study. This was a prospective, observational study. Perfusion index, heart rate, blood pressure and Spo2 were recorded every minute from the baseline to 10 min after induction of

anaesthesia with a titrated dose of propofol, and also after endotracheal intubation. In this study, hypotension was defined as fall in systolic blood pressure by >30% of baseline or mean arterial pressure (MAP) to <60 mm Hg. Severe hypotension was treated (MAP of <55 mm Hg).

Baseline PI <1.05 predicted incidence of hypotension at 5 min with sensitivity of 93% and specificity of 71%. The positive predictive value (PPV) and negative predictive value(NPV) was 68% and 98% respectively. The area under the ROC curve (AUC) was 0.816 at 95% confidence interval (0.699–0.933), *P*<0.001**.**

They concluded that Perfusion index could be used for prediction of hypotension following induction of general anaesthesia using propofol, particularly before endotracheal intubation.

Mowafi et al conducted a study in 2009 to evaluate the efficacy of Perfusion index for detection of intravascular injection of a simulated epidural test dose containing 15 µg of epinephrine in adults during propofol-based anesthesia and comparing its reliability with the conventional heart rate (positive if >or=10 bpm) and systolic blood pressure (SBP) (positive if >or=15 mm Hg) criteria.

40 patients planned for elective general surgery under total IV anesthesia were randomized to receive either 3 mL of lidocaine 15 mg/mL with epinephrine 5 µg/mL or 3 mL of saline IV (n = 20 each). HR, SBP, and PI were monitored for 5 min after injection. Injecting the test dose

resulted in an average maximum PI decrease by 65% +/- 13% at 39 +/- 15 s. Moreover, maximal increases in HR and SBP were 19 +/- 8 bpm at 49 +/- 25 s and 17 +/- 7 mm Hg at 102 +/- 34 s after test dose injections, respectively. Using the PI criterion for intravascular injection (positive if PI decreases >or=10% from the preinjection value) the sensitivity, specificity, positive predictive, and negative predictive values were 100% (95% confidence interval [CI]; CI = 83%-100%). On the contrary, sensitivities of 95% (CI = 76%-99%) and 90% (CI = 70%-97%) were obtained based on HR and SBP criteria, respectively.

PI is a reliable alternative to conventional hemodynamic criteria for detection of an intravascular injection of epidural test dose in propofol-anesthetized adult patients.

Dr Joseph George et al conducted a study in 2019 To find out the correlation between baseline perfusion index and incidence of hypotension following sub arachnoid block in Lower segment caesarean section.

It was a prospective observational study, 30 parturients belonging to American society of Anesthesiologists physical status 2 pregnancies, that are not associated with any other co-morbidities were scheduled for elective lower segment caesarean section under spinal anaesthesia . Spinal anaesthesia was performed with 10mg of 0.5% hyperbaric bupivacaine (heavy) at L3-L4 or L2-L3 interspinous space using a 25G Quincke needle.

They defined hypotension as a 25% decrease in systolic blood pressure from baseline value.

The incidence of hypotension was found out to be 66.7%. They found a significant correlation between baseline PI and fall in the SAP from baseline ($r= 0.368$, $P < 0.05$). The optimal cutoff point across a range of cutoff points for PI was found to be 3.6 with a sensitivity of 80% and specificity of 60%,

It was concluded that Baseline perfusion index >3.6 is associated with higher incidence of hypotension following SAB in elective caesarean delivery.

Ginosar Y et al conducted a study in 2009 that perfusion index(PI) derived from pulse oximeter provides an earlier and clearer indication of sympathectomy following epidural anesthesia than arterial pressure and skin temperature.

40 patients received lumbar epidural catheters. They were randomized to receive 10 ml 0.5% bupivacaine or 10 ml 0.25% bupivacaine. PI in the toe, mean arterial pressure (MAP) and toe temperature, all these parameters were assessed at baseline and at 5, 10 and 20 min following epidural anesthesia. The effect of epidural anesthesia over time was assessed by repeated measures analysis of variance. Clinically evident sympathectomy criteria was defined as a 100% increase

in the PI, a 15% decrease in the MAP and a 1 degrees Celcius increase in toe temperature.

29 subjects had photoplethysmography signals that met a prior signal quality criteria for analysis. By 20 min, PI was found to be increased by 326%, compared to 10% decrease and a 3% increase in the MAP and toe temperature, respectively.

It was concluded that Perfusion index was an earlier and more sensitive indicator of development of epidural-induced sympathectomy than MAP or skin temperature.

MATERIAL AMD METHODS

The study was conducted at obstetrics and gynaecology operation theatre, department of anaesthesiology, Tirunelveli medical college and hospital between January 2018 - June 2018.

This study was done in 120 patients who underwent elective lower segment caesarean section. Ethical committee approval and informed written consent from patients involved in this study are obtained before starting this study.

Study design

Prospective, double-blinded, observational study.

Inclusion Criteria

Parturients between 20yrs and 35yrs of age posted for elective caesarean section

Exclusion criteria

Parturients with

a. Placenta praevia

b Cardiovascular or cerebrovascular disease

c.Preeclampsia

d. Body mass index $\geq$40

e. Gestational diabetes mellitus

f. Gestational age <36 or >41weeks

g. Contraindications to regional anaesthesia

h. Those requiring emergency LSCS

STUDY DESIGN

Based on the baseline perfusion index, parturients are divided as follows;

* Parturients with PI of ≤3.5 come under Group A

* Parturients with PI >3.5 come under Group B

STUDY MANOEUVRE:

- Preoperative assessment will be done
- Anaesthetic machine is checked before starting the procedure
- Ensure the availability of working laryngoscope, oral airway, laryngeal mask airway and endotracheal tube of various sizes
- Make sure that the essential emergency drugs are available
- Ensuring the operating table tilts are corrected
- Standard monitoring as per ASA guidelines was performed for baseline values and intraoperative monitoring.
- Perfusion index was measured in supine position using a specific pulse oximeter probe (Masimo Radical 7®; Masimo Corp., Irvine, CA, USA). To ensure uniformity in all the parturients , PI was measured in left index finger.
- All the baseline values including PI was recorded in supine position by the anaesthesiologist who was not involved in further intraoperative monitoring of the patient.

- Parturients with baseline PI of ≤3.5 are categorised as Group A and those with a PI of >3.5 as Group B.
- Intravenous (IV) access was established in the left upper limb. Every parturient was prehydrated with 500 ml of Ringer lactate over 20 min. After prehydration was over, the baseline values were recorded.
- During administration of neuraxial blockade, the Masimo® pulse oximeter was disconnected from the patient to prevent observer bias and oxygen saturation was recorded using a different pulse oximeter which did not showed PI.
- The anaesthesiologist who was blinded to the baseline PI values performed spinal anaesthesia, using 25‑gauge Quincke spinal needle in left lateral decubitus position with injection 0.5% hyperbaric(heavy) bupivacaine ,10 mg at the L2–L3 or L3–L4 interspace. The parturient was returned to the supine position with a left lateral tilt of 15° to facilitate left uterine displacement. Oxygen was given at a rate of 4L/min via face mask.
- IV fluids were given at a rate of 100ml/min. The level of sensory block was checked at 5 min after the spinal injection with a cold swab. If aT6 sensory block level was not achieved, then these parturients were excluded from the study.

- After 20mins, maximum cephalic spread was checked, heart rate, respiratory rate and SpO2 were recorded at 2 min intervals after the SAB up to 20 min and then at 5 min intervals by the same anaesthesiologist who administered Subarachnoid block till the end of surgery.
- If MAP was <65 mm of Hg, it was defined as hypotension and was treated with 6 mg injection ephedrine IV bolus and 100 ml of Ringer lactate(RL). The first 60 min following SAB was considered for spinal anaesthesia‑induced hypotension. If Heart rate was <55 beats/min, was treated with injection atropine 0.6 mg IV bolus.
- After extraction of the baby, Apgar score was recorded at 1st and 5th min. Injection oxytocin 10 units was given as uterotonic following baby extraction at a rate of 200 mU/min as a separate infusion.
- Parturients requiring extra oxytocics or any additional surgical interventions were excluded from the study. Other side effects like nausea, vomiting were also recorded.

METHODOLOGY

STATISTICAL TESTS USED:

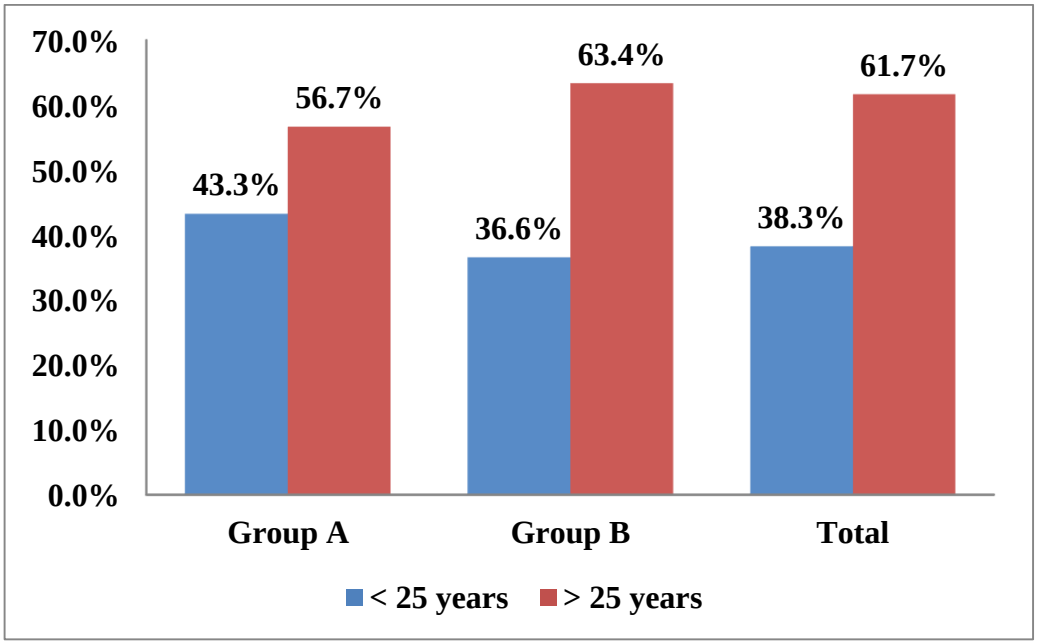
Data was entered into Microsoft Excel (Windows 7; Version 2007) and analyses were done using the Statistical Package for Social Sciences (SPSS) for Windows software (trial version 22.0; SPSS Inc, Chicago). Descriptive statistics such as mean and standard deviation (SD) for continuous variables, frequencies and percentages were calculated for categorical variables. Bar and Pie charts were used for visual representation of data. To check for correlation between perfusion index and hypotension episode and ephedrine dose pearson correlation was used. To find the diagnostic accuracy of perfusion index and sensitivity, specificity of the same ROC curve was constructed using SPSS. To check for comparison of Pulse rate and mean arterial pressures between both groups during course of anaesthesia students t test was used. Level of significance was set at 0.05.

RESULTS

Table 1: Age group distribution in both groups:

Age	Group A N = 60	Group B N = 60	Total N = 120	P value
< 25 years	26 (43.3%)	22 (36.6%)	46 (38.3%)	**0.061**
> 25 years	34 (56.7%)	38 (63.4%)	72 (61.7%)	
Total	60 (100%)	60 (100%)	120 (100%)	
Mean	24.6	25.8	24.8	
Standard deviation	2.3	2.2	2.4	
Range	20 - 28	23 - 29	20 – 29	

Chart 1: Age group distribution in both groups:



Majority of the study participants were distributed more in >25 years of age in both age groups and in total. The age did not differ significantly in both groups hence both the groups are comparable in terms of age

Table 2: Height and weight distribution profile in both groups:

Sex	Group A N = 60	Group B N = 60	Total N = 120	P value
Height **Mean±SD** **Range**	155.2±1.6	156±1.5	155.9±1.7	0.064
Weight **Mean±SD** **Range**	66.1±2.2	66.8±2.3	66.4±2.2	0.067

The mean height and weight in group A and B were 155cms and 66kg, 156cms and 66.8kg respectively. There was no significant difference in height and weight distribution of both groups with P value 0.067.

Table 3: ASA distribution in both groups:

ASA	Group A N = 60	Group B N = 60	Total N = 120	P value
I	0 (0.0%)	0 (0.0%)	0 (0.0%)	
II	60 (100%)	60 (100%)	60 (100%)	1.000

Both groups had equal distribution of ASA category. There were 60 ASA grade II in both the groups.

Table 4: Duration of surgery profile in both groups:

Duration of surgery	Group A N = 60	Group B N = 60	Total N = 120	P value
Duration of surgery Mean±SD Range	49.2±4.4 40 - 55	49.6±3.0 46 – 57	49.4±3.8 40 - 57	0.508

There was no significant difference in the duration of surgery among both the groups. The mean duration of surgery in group A is 49.2 mins while in group B was 49.6 mins.

Table 5: Fluid requirement in both groups:

Fluid requirement	Group A N = 60	Group B N = 60	Total N = 120	P value
Mean±SD	1000.8±44.6	1150±37	1010±42	**0.012**
Range	900 - 1050	1000 - 1150	900 - 1150	

There was a significant difference in the fluid requirement of both the groups. The fluid required in group A is 1000ml while the fluid required in group B is 1150 ml this difference was statistically significant with P value 0.012.

Table 6: Perfusion index in both groups:

Perfusion index	Group A N = 60	Group B N = 60	Total N = 120	P value
Mean±SD	2±0.3	5.2±0.9	3.6±1.7	**0.001**
Range	2 – 3	4 - 7	2 - 7	

.

The mean perfusion index in group A is 2 and mean perfusion index in group B is 5.2. There was a significant difference in mean perfusion index of both the groups with P value 0001.

Table 7: Maximum cephalic spread in both groups:

Maximum cephalic spread	Group A N = 60	Group B N = 60	Total N = 120	P value
T2	13(21.7%)	13(21.7%)	26 (21.7%)	0.588
T4	47 (78.3%)	47 (78.3%)	94 (78.3%)	
Total	60 (100%)	60 (100%)	120 (100%)	

The proportion of maximum cephalic spread till T2 in group A is 21.7% and group B is 21.7%. The proportion of maximum cephalic spread till T3 in group A is 78.3% and group B is 78.3%. There was no significant difference in both the groups with P value 0.588.

Chart 3: Maximum cephalic spread in both groups:

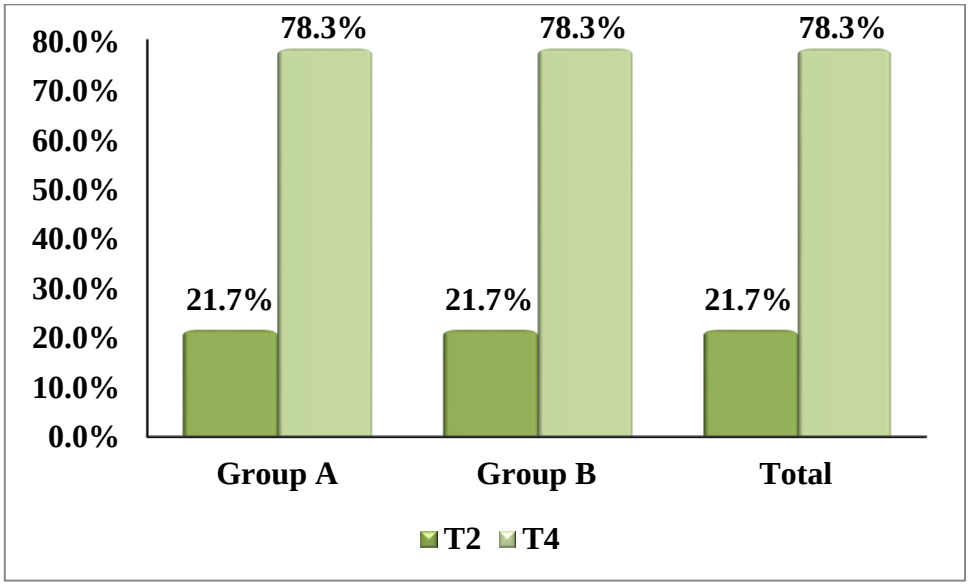


Table 8: Nausea in both groups:

Nausea	Group A N = 60	Group B N = 60	Total N = 120	P value
Present	2(3.3%)	5(8.3%)	7 (5.8%)	**0.243**
Absent	58 (96.7%)	55(91.7%)	113 (94.2%)	
Total	60 (100%)	60 (100%)	120 (100%)	

The proportion of nausea in group A is 3.3% and group B is 8.3%. The proportion of no nausea in group A is 96.7% and group B is 91.7%. Though the percentage of nausea is more in group B there was no significant in both the groups with P value 0.243.

Chart 4: Nausea in both groups:

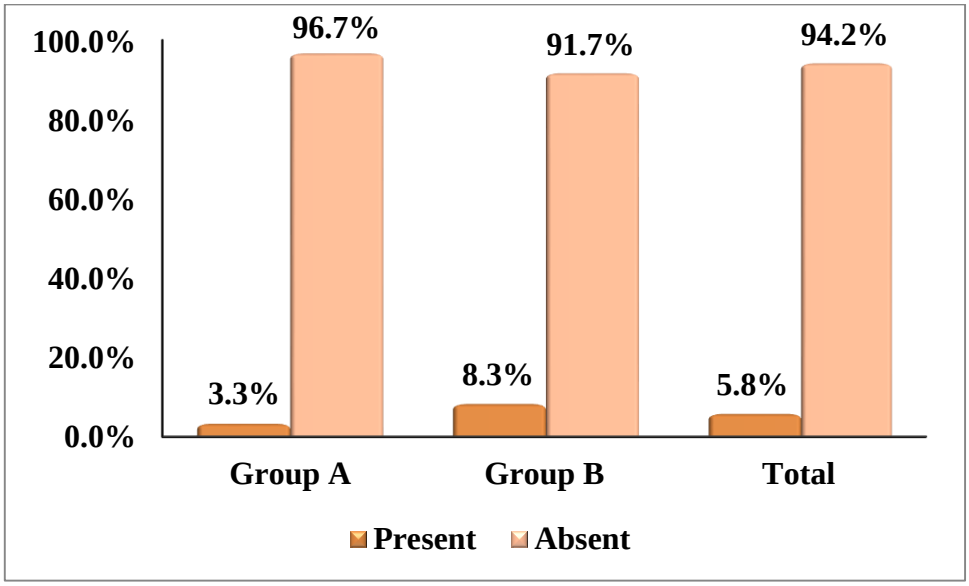


Table 9: Ephedrine dose required in both the groups:

Ephedrine dose	Group A N = 60	Group B N = 60	Total N = 120	P value
None	51(85%)	17(28.3%)	68 (56.7%)	**<0.001**
1 -2 dose	8 (13.3%)	37 (61.7%)	45 (37.3%)	
3 – 4 dose	1 (1.7%)	8 (10%)	9 (6%)	
Total	60 (100%)	60 (100%)	120 (100%)	

In group A the percentage of not requiring ephedrine dose is 85% and group B is 28.3%. The proportion requiring 1-2 doses in group A is 13.3% and group B is 61.7%. The percentage of ephedrine dose required in group B was more compared to group Aand this was statistically significant with P value <0.001.

Chart 5: Ephedrine dose required in both the groups:

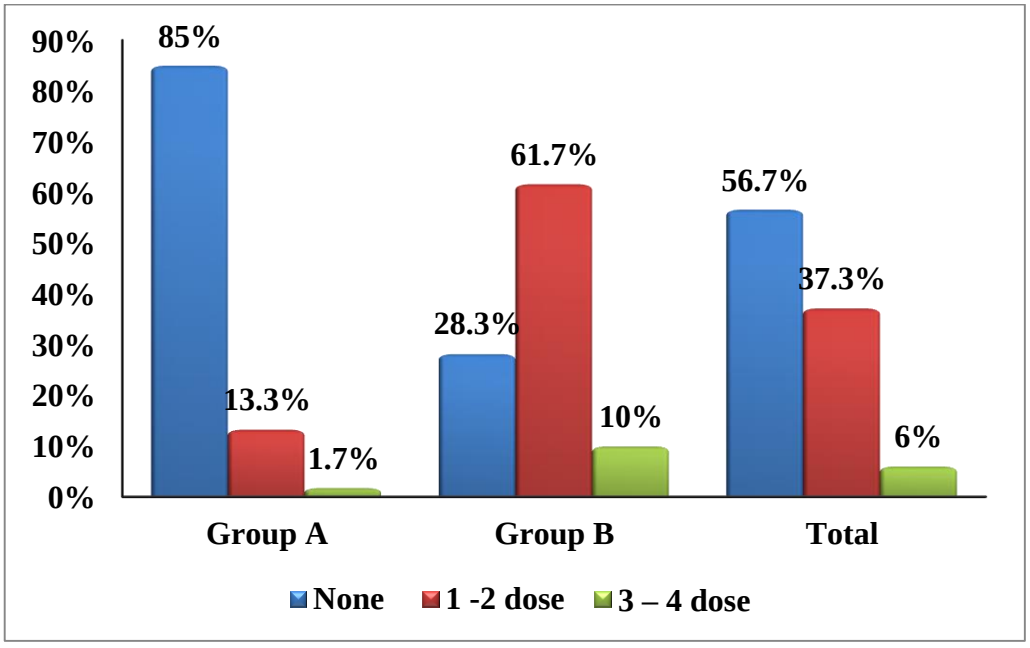


Table 10: Hypotension in each group:

Hypotension	Group A N = 60	Group B N = 60	Total	P value
Present	11 (18.4%)	39 (65%)	50 (41.7%)	**<0.001**
Absent	49 (81.6%)	21 (35%)	70 (58.3%)	

The incidence of hypotension in group A is 18.4% and group B is 65% this difference was statistically significant with P value< 0.001

Chart 6: Hypotension in each group:

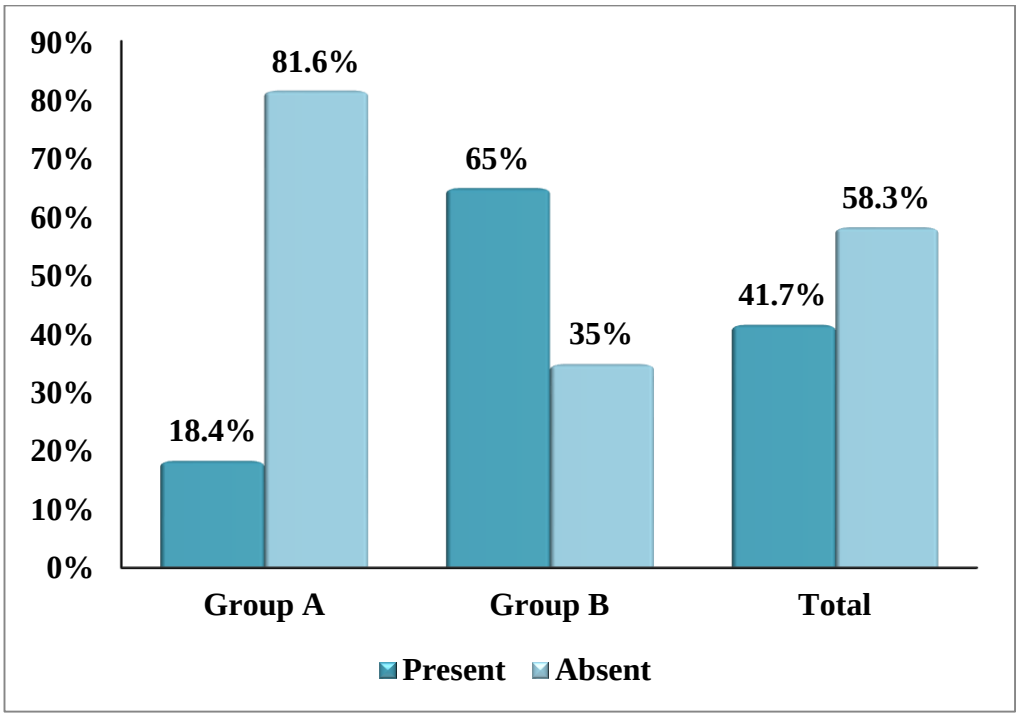


Table 11: Hypotension episode in each group:

Hypotension	Group A N = 60	Group B N = 60	P value
0	49 (81.6%)	21 (35%)	**<0.001**
1	8 (13.3%)	20 (33.3%)	
2	2 (3.4%)	15 (25.1%)	
3	1 (1.7%)	2 (3.3%)	
4	0	2 (3.3%)	

In group A, the percentage of hypotension episode is 81.6% and group B is 35%. The proportion having 1 episode in group A is 13.3% and group B is 33.3%. The proportion having 2 episodes in group A is 3.4% and group B is 25.1%.The percentage of having 3 episodes in group A is 1.7% and group B is 3.3%. There was no one in group A with 4 episodes of hypotension. But in group B there were 2 people with 4 episodes of hypotension. The hypotension episodes were more in group B compared to group A and this was statistically significant with P value <0.001.

Table 12: Comparison of Pulse rate during the course of anaesthesia in both groups:

Heart rate(min)	Group A		Group B		P value
	Mean	SD	Mean	SD	
1	75.5	7.1	76.6	6.9	0.380
2	78.6	6.5	80.1	7.1	0.237
4	81	6	82	6.7	0.410
6	82.8	5.6	84.5	6.4	0.127
8	83.5	4.1	84.7	5.0	0.159
10	82.3	6.2	83.5	5.6	0.248
12	78.4	6.2	80.3	7.3	0.126
14	77.7	6.1	78.7	5.8	0.397
16	76.7	6	77.6	6.6	0.455
18	75.9	6.2	76	5.6	0.902
20	74.9	5.9	74.9	6.0	1.000
25	73.7	6.1	74.6	5.8	0.421
30	73	6.3	73.9	6.3	0.482
35	72.8	5.3	73.5	5.3	0.517
40	72.4	4.7	72.6	4.6	0.726
45	71	4.1	71	3.9	0.946
50	71.1	3.8	72.4	4.1	0.071
55	71.3	3.4	72.3	3.8	0.319
60	72	2.8	72.2	2.9	0.321

The mean pulse rate in both groups did not differ significantly from each other during the course of the anaesthesia. All the P value were more than 0.05

Chart7: Comparison of Pulse rate during the course of anaesthesia in both groups:

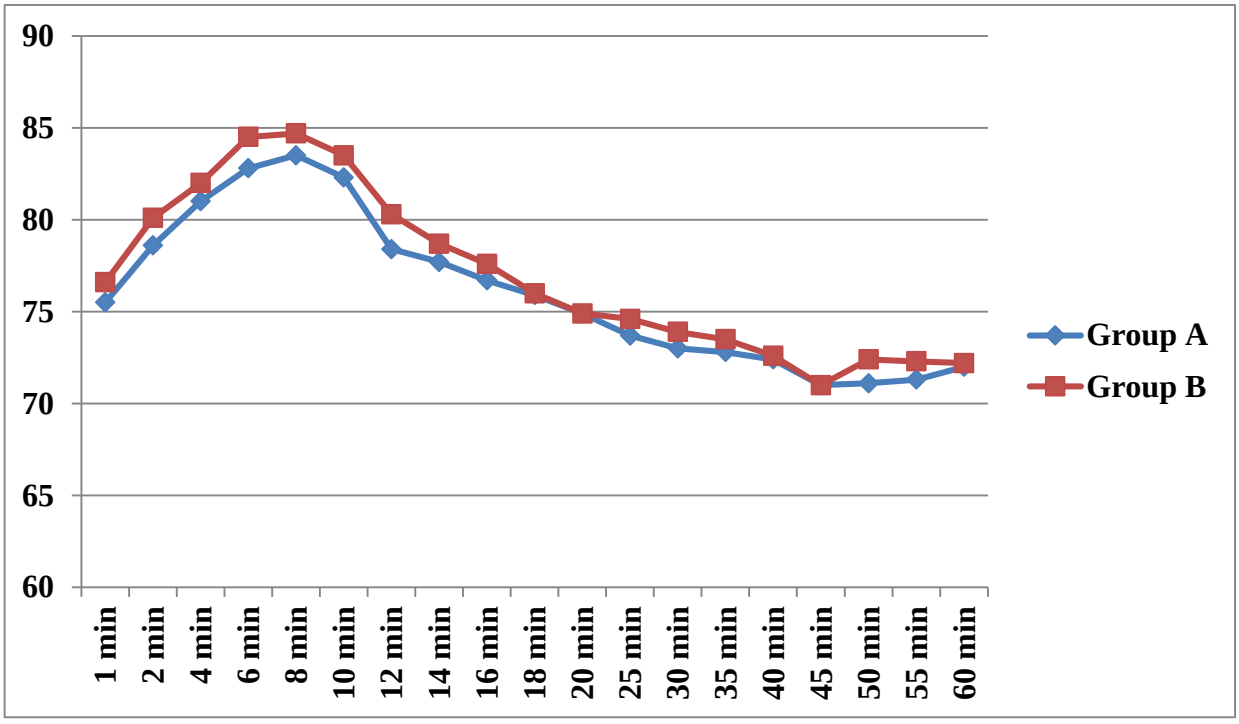


Table 13: Comparison of Mean arterial pressure during the course of anaesthesia in both groups:

MAP (min)	Group A		Group B		P value
	Mean	SD	Mean	SD	
1	86.4	7.6	85.5	7.5	0.001
2	79.9	6.6	82	6.5	0.021
4	75.9	6.6	78.8	6.3	0.817
6	72.6	5.5	72.4	6.9	0.001
8	72.3	4.5	69.1	5.5	0.001
10	73	4.1	70.2	4.7	0.045
12	73.1	4.1	70.2	4.7	0.329
14	73.8	4.4	71.2	5.2	0.252
16	73.9	4.3	73	4.7	0.360
18	74.2	3.5	73.6	4.0	0.251
20	74.8	3.1	74.4	3.4	0.490
25	74	3.2	75	3.2	0.033
30	73.9	3.6	74.7	3.6	0.204
35	73.7	3.2	75.2	4.3	0.027
40	74	3.4	74.3	3.2	0.597
45	74.2	3.0	74.7	3.3	0.349
50	73.7	2.6	74.8	3.0	0.042
55	74.3	3.1	75.4	2.9	0.132
60	73	2.1	74.5	2.2	0.015

The mean arterial pressure in both groups did not differ significantly from each other during the course of the anaesthesia on most times. In initial minutes of 1, 2, 6 and 8 mean arterial pressure of group B was higher and this difference was statistically significant with P value <0.05. All the other minutes the MAP was similar in both groups with P value were more than 0.05.

Chart 8: Comparison of Mean arterial pressure during the course of anaesthesia in both groups:

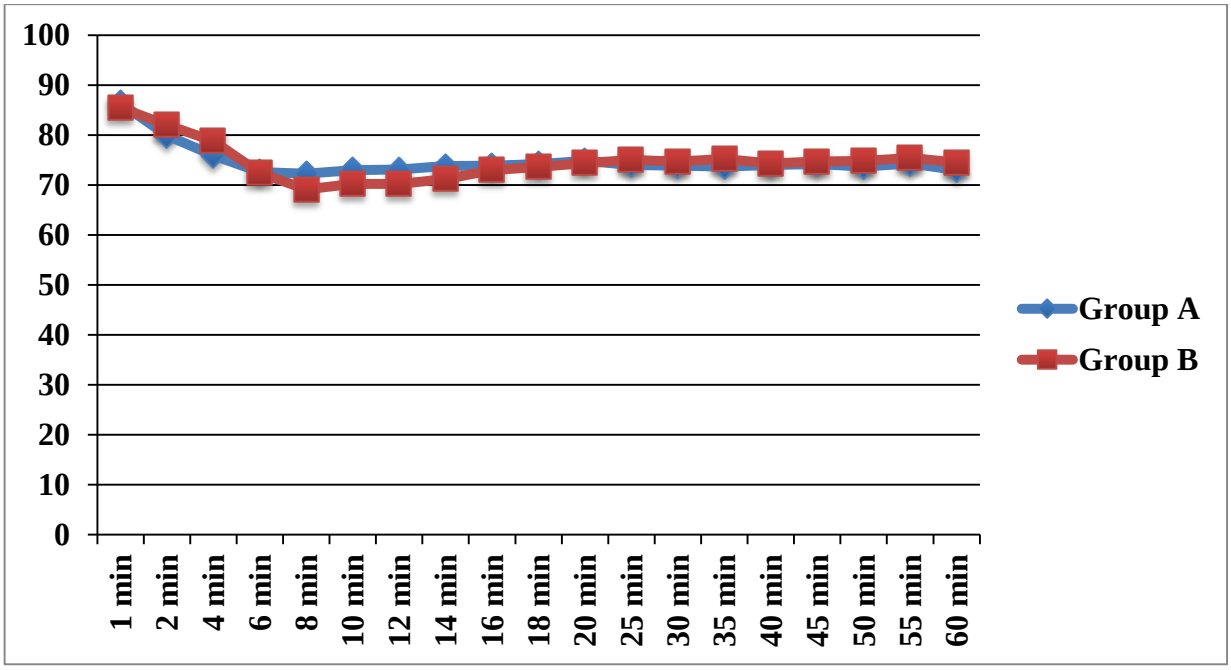


Table 14: Correlation between perfusion index and hypotension episodes and ephedrine dose

	Hypotension episode		Ephedrine dose	
	Correlation coefficient	**Significance**	**Correlation coefficient**	**Significance**
Perfusion Index	**0.570**	**<0.001**	**0.732**	**<0.001**

There was significant correlation of perfusion index with hypotension episodes and ephedrine dose. They both are positively correlated. That is as perfusion index increases the number of hypotension episodes and ephedrine dose increases. There was 57% correlation between perfusion index and number of hypotension episodes which was significant with p value < 0.001. There was 73.2% correlation between perfusion index and ephedrine dose which was significant with p value < 0.001.

Chart 9: Correlation between perfusion index and ephedrine dose

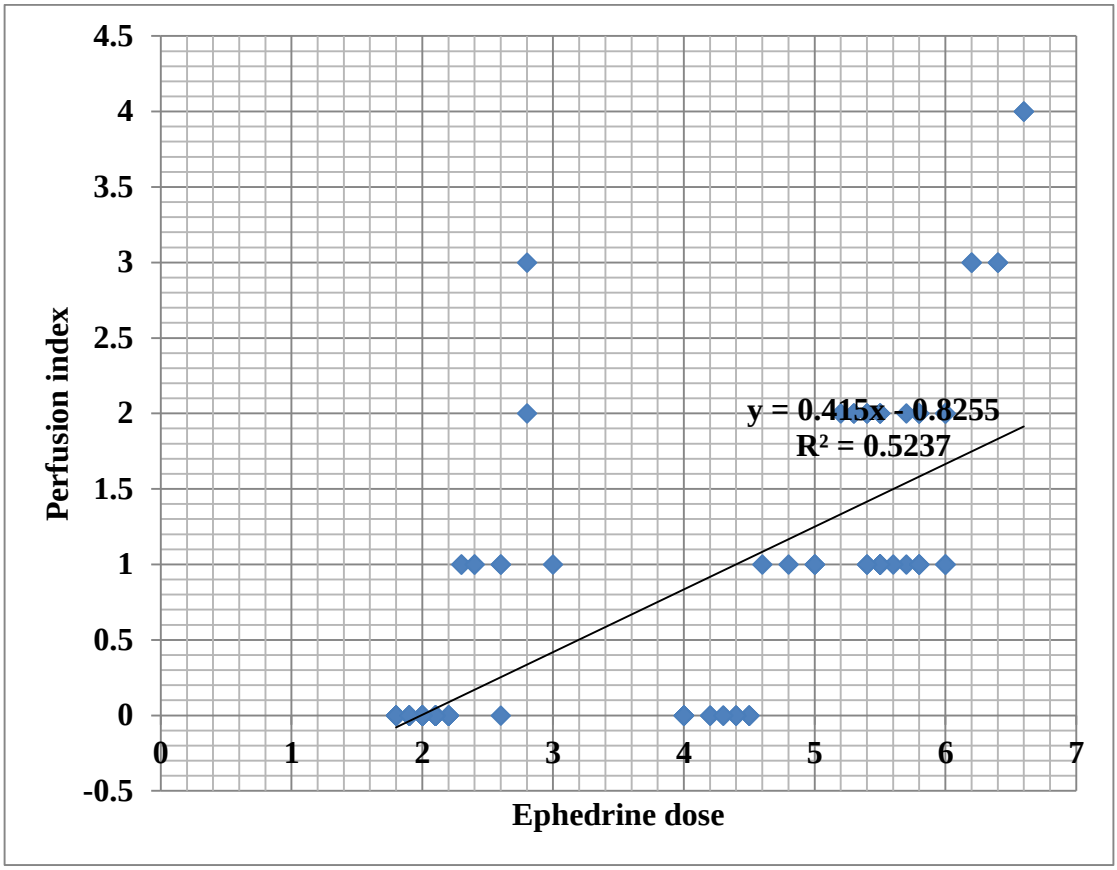


Chart 10: Correlation between perfusion index and hypotension episodes

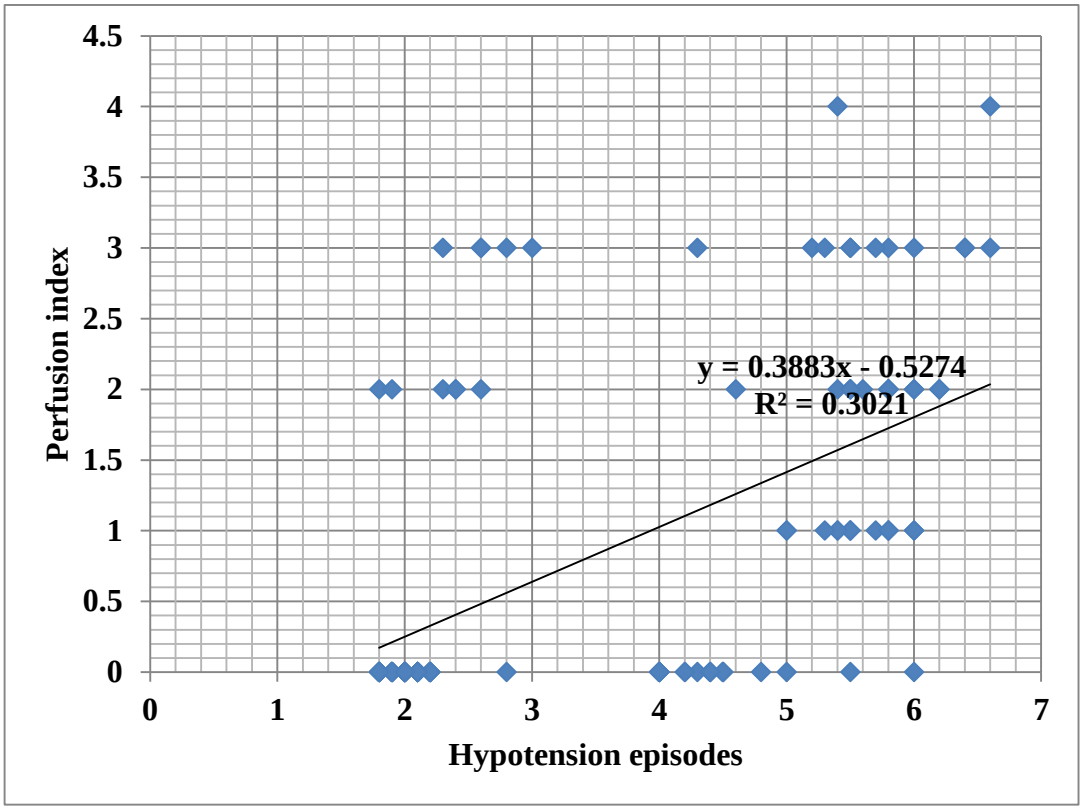


Chart 8: ROC curve depicting baseline PI against incidence of hypotension:

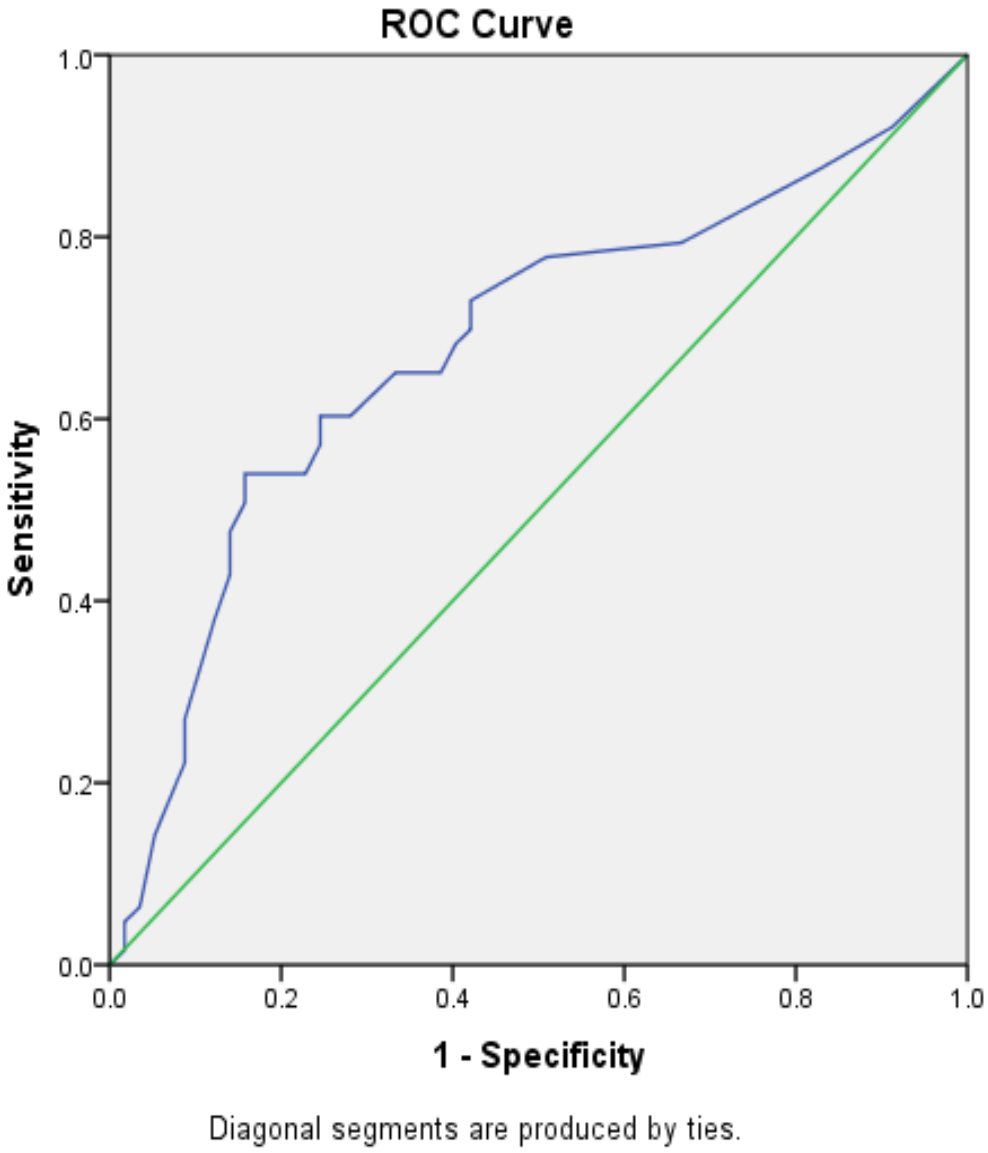


Table 15: Area under the curve for ROC:

Area under the curve	Standard error	Significance	95% confidence interval	
			Lower bound	Upper bound
0.681	0.050	0.001	0.584	0.778

The ROC curve for perfusion index with cut off 3.5 has given area under the curve value 68% which implies 68% diagnostic accuracy in predicting hypotension with 3.5 cutoff.

Table 16: Sensitivity and Specificity of Perfusion index in predicting hypotension:

Perfusion index	Sensitivity	Specificity
Cut off 3.5	65%	67%

The cut off 3.5 of perfusion index 3.5 had the best sensitivity which was 65% and specificity 67%. Hence we can conclude that using 3.5 as cut off for perfusion index is both sensitive and specific in predicting hypotension.

DISCUSSION

Hyotension after administration of spinal anaesthesia for lower segment caesarean section is very common. There is no definitive monitoring system which may help to predict development of hypotension following SAB, so that additional precautions have to be taken.

In our study, the incidence and severity of hypotension, vasopressor requirement were found to be higher in parturients with baseline Perfusion index values were > 3.5.

Normal pregnancy is characterised by decrease in systemic vascular resistance, increase in cardiac output and total blood volume. This reduction of the systemic vascular resistance may vary with each parturient depending on many factors. The decrease in the vascular tone will correspond to higher perfusion index values as there is increase in pulsatile component due to vasodilatation. Sympathectomy due to spinal anaesthesia(SA) will cause peripheral vascular tone to further decrease and increase blood pooling and hypotension.

Parturients with high baseline perfusion index will be expected to have a lower peripheral vascular tone and thus they are at higher risk for developing hypotension following SA.

The cut‑off value of baseline perfusion index for predicting spinal anaesthesia induced hypotension was chosen as 3.5 based on a study conducted by Toyama et al, they did regression analysis and ROC curve

analysis and concluded that a baseline perfusion index cut‑off point of 3.5 could be used to identify parturients who are at risk for developing hypotension following SA.

In our study, the baseline PI >3.5 and probability of hypotension were significantly correlating, which were similar to the study conducted by Toyama et al.

Toyama et al. found a sensitivity and specificity of 81%and 86%, respectively, for baseline PI with a cut‑off of 3.5 to predict hypotension, whereas in this study, the specificity was 65% and sensitivity was 67%.

In our study, consumption of IV fluid was significantly higher than that in the study by Toyama et al. This is because we used injection ephedrine and fluid bolus for treating hypotension while they used injection phenylephrine only to treat hypotension.

Prostaglandins, methylergometrine are very powerful vasoconstrictors and hence the patients receiving these drugs were excluded from the analysis as they can influence the observations.

Duggappa DR, et al conducted the study to explore the predictive ability of Perfusion index following SAB in elective lower segment caesarean section. On Spearman rank correlation, they found out a highly significant correlation between baseline PI >3.5, number of hypotensive episodes, the total dose of ephedrine required and total IV fluids given.

A higher requirement of vasopressor was seen in parturients with baseline PI >3.5. Sensitivity was 89.29% and specificity was 69.84%, whereas in our study, the specificity was 65% and sensitivity was comparable, 67%. In our study, the consumption of IV fluid was similar to the study by Duggappa DR, et al.

Mowafi et al. used PI to detect intravascular injection of the epinephrine‑containing epidural test dose, so its reliability to detect vasoconstriction has been previously demonstrated successfully.

Ginosar et al. demonstrated that increase in PI following epidural anaesthesia was a clear and reliable indicator of sympathectomy.

A study performed by Yokose et al demonstrated that PI had no predictive value for hypotension in parturients undergoing LSCS following SAB as in this study they used colloids for co-loading and the definition of hypotension was different when compared to our study.

CONCLUSION

We conclude from this study that Perfusion Index can be used for predicting hypotension in parturients undergoing elective lower segment caesarean delivery under spinal anaesthesia. Our results found out that parturients with baseline Perfusion index >3.5 are at a greater risk of developing hypotension following Spinal anaesthesia than compared to those with the baseline PI ≤3.5.

BIBLIOGRAPHY

1. Toyama s, kakumoto m, morioka m, matsuoka k, omatsu h,Tagaito y, *et al.* Perfusion index derived from a pulse Oximeter can predict the incidence of hypotension during Spinal anaesthesia for caesarean delivery. Br j anaesth 2013;111:235‐41.
2. Duggappa DR, Lokesh M, Dixit A, Paul R, Raghavendra Rao RS, Prabha P. Perfusion index as a predictor of hypotension following spinal anaesthesia in lower segment caesarean section. Indian J Anaesth 2017;61:649-54.
3. Yokose m, mihara t, sugawara y, goto t. The predictive ability Of non‐invasive haemodynamic parameters for hypotension During caesarean section: a prospective observational study.Anaesthesia
4. Mowafi ha, ismail sa, shafi ma, al‐ghamdi aa. The Efficacy of perfusion index as an indicator for intravascular Injection of epinephrine‐containing epidural test dose in Propofol‐anesthetized adults. Anesth analg 2009;108:549‐53.
5. Hanss r, bein b, ledowski t, lehmkuhl m, ohnesorge h,Scherkl w, *et al.* Heart rate variability predicts severe Hypotension after spinal anesthesia for elective caesarean Delivery. Anesthesiology 2005;102:1086‐93.

6. Gaiser r. Physiological changes of pregnancy. In: chestnut dh,Editor. Chestnut's obstetric anaesthesia: principles and Practice. 5th ed. Philadelphia: mosby elsevier publishing;2014. P. 15‐38.
7. Hales jr, stephens fr, fawcett aa, daniel k, sheahan j,Westerman ra, *et al.* Observations on a new non‐invasive Monitor of skin blood flow. Clin exp pharmacol physiol 1989;16:403‐15.
8. Ginosar y, weiniger cf, meroz y, kurz v, bdolah‐abram t,Babchenko a, *et al.* Pulse oximeter perfusion index as an early Indicator of sympathectomy after epidural anesthesia. Acta Anaesthesiol scand 2009;53:1018‐26.
9. Lima ap, beelen p, bakker j. Use of 6. Lima ap, beelen p, bakker j. Use of a peripheral perfusion Index derived from the pulse oximetry signal as a noninvasive Indicator of perfusion. Crit care med 2002;30:1210‐3.
10. Park ge, hauch ma, curlin f, datta s, bader am. The effects of Varying volumes of crystalloid administration before caesarean Delivery on maternal hemodynamics and colloid osmotic Pressure. Anesth analg 1996;83:299‐303.
11. Ajne g, ahlborg g, wolff k, nisell h. Contribution of Endogenous endothelin ‐1 to basal vascular tone during Normal pregnancy and preeclampsia. Am j obstet gynecol 2005;193:234‐40.

12. Barwin bn, roddie ic. Venous distensibility during pregnancy Determined by graded venous congestion. Am j obstet gynecol 1976;125:921‐3.
13. Sakai k, imaizumi t, maeda h, nagata h, tsukimori k, Takeshita a, *et al.* Venous distensibility during pregnancy.Comparisons between normal pregnancy and preeclampsia.Hypertension 1994;24:461‐6.
14. Bowyer l, brown ma, jones m. Forearm blood flow in Pre‐eclampsia. Bjog 2003;110:383‐91.
15. Clapp jf 3rd, capeless e. Cardiovascular function before, During, and after the first and subsequent pregnancies. Am j Cardiol 1997;80:1469‐73.

PROFORMA

NAME: AGE: SEX: IP NO:

ASA: HEIGHT: WEIGHT:

DIAGNOSIS:

SURGICAL PROCEDURE:

BASELINE PERFUSION INDEX:

PREOPERATIVE : Mean arterial pressure

Heart rate

AFTER SUBARCHNOID BLOCK:

Level of sensory blockade (after 5mins):

Maximum cephalic spread (after 20mins):

INTRAOPERATIVE VITALS:

Time in mins	Heart rate	MAP	Spo2
1			
2			
4			
6			
8			
10			
12			
14			
16			
18			
20			
25			
30			
35			
40			
45			
50			
55			
60			

TOTAL DOSE OF EPHEDRINE:

FLUID REQUIREMENT:

ADVERSE EFFECTS: Nausea/ Vomiting/ Respiratory depression

ANY ADDITIONAL DRUGS :

DURATION OF SURGERY:

நோயாளிகளுக்கு அறிவிப்பு மற்றும் ஒப்புதல் படிவம்
(மருத்துவ ஆய்வில் பங்கேற்பதற்கு)

ஆய்வு செய்யப்படும் தலைப்பு:
பங்கு பெறுவரின் பெயர்:
பங்கு பெறுவரின் வயது:

		பங்கு பெறுவர் இதனை ✓ குறிக்கவும்
1.	நான் மேலே குறிப்பிட்டுள்ள மருத்துவ ஆய்வின் விவரங்களை படித்து புரிந்து கொண்டேன். என்னுடைய சந்தேகங்களை கேட்கவும், அதற்கான தகுந்த விளக்கங்களை பெறவும் வாய்ப்பளிக்கப்பட்டுள்ளது என அறிந்து கொண்டேன்.	☐
2.	நான் இவ்வாய்வில் தன்னிச்சையாக தான் பங்கேற்கிறேன். எந்த காரணத்தினாலோ எந்த கட்டத்திலும், எந்த சட்ட சிக்கலுக்கும் உட்படாமல் நான் இவ்வாய்வில் இருந்து விலகி கொள்ளலாம் என்றும் அறிந்து கொண்டேன்.	☐
3.	இந்த ஆய்வு சம்பந்தமாகவோ, இதை சார்ந்து மேலும் ஆய்வு மேற்காள்ளும் போதும் இந்த ஆய்வில் பங்குபெறும் மருத்துவர் என்னுடைய மருத்துவ அறிக்கைகளை பார்ப்பதற்கு என் அனுமதி தேவையில்லை என அறிந்து கொள்கிறேன். நான் ஆய்வில் இருந்து விலகிக் கொண்டாலும் இது பொருந்தும் என அறிகிறேன்.	☐
4.	இந்த ஆய்வின் மூலம் கிடைக்கும் தகவலையோ, முடிவையோ பயன்படுத்திக் கொள்ள மறுக்க மாட்டேன்.	☐
5.	இந்த ஆய்வில் பங்கு கொள்ள ஒப்புக் கொள்கிறேன் எனக்கு கொடுக்கப்பட்டஅறிவுரைகளின் படி நடந்து கொள்வதுடன், ஆய்வை மேற்கொள்ளும் மருத்துவ அணிக்கு உண்மையுடன் இருப்பேன் என்று உறுதியளிக்கிறேன். என் உடல் நலம் பாதிக்கப்பட்டாலோ, அல்லது எதிர்பாராத, வழக்கத்திற்கு மாறான நோய்குறி தென்பட்டாலோ உடனே இதை மருத்துவ அணியிடம் தெரிவிப்பேன் என உறுதி அளிக்றேன்.	☐

பங்கேற்பவரின் கையொப்பம் / ..... இடம் .....
கட்டைவிரல் ரேகை
பங்கேற்பவரின் பெயர் மற்றும் விலாசம் .....
ஆய்வாளரின் கையொப்பம் /..... இடம் .....
ஆய்வாளரின் பெயர் .....
மையம் .....
கல்வியறிவு இல்லாதவற்கு (கைரேகை வைத்தவர்களுக்கு) இது அவசியம் தேவை
சாட்சியின் கையொப்பம் /..... இடம் .....
பெயர் மற்றும் விலாசம் ....

SL NO	AGE	HEIGHT (cms)	WEIGHT (kg)	ASA	GROUP A (PERFUSION INDEX)	LEVEL OF SENSORY BLOCK (AFTER 5MINS)	MAXIMUM CEPHALIC SPREAD(AFTER 20MINS)	ADVERSE EFFECTS	DURATION OF SURG (mins)
1	22	153	65	2	2.2	T6	T4		45
2	22	154	66	2	1.8	T6	T4		50
3	21	157	65	2	1.9	T6	T4		55
4	20	159	63	2	2	T6	T4		55
5	27	157	67	2	1.8	T6	T4		44
6	26	153	70	2	1.9	T6	T4		55
7	24	155	69	2	2.2	T6	T2		45
8	25	154	69	2	2.1	T6	T4		40
9	28	154	66	2	2.2	T6	T4		46
10	27	156	68	2	2.1	T6	T4		40
11	20	154	67	2	1.9	T6	T4		50
12	21	155	63	2	1.8	T6	T4		50
13	23	157	66	2	1.9	T6	T4		50
14	22	153	67	2	2	T6	T4		45
15	22	155	66	2	2	T6	T4		50
16	25	154	64	2	2	T6	T4		47
17	26	156	70	2	2.1	T6	T4		48
18	27	157	70	2	2	T6	T2		49
19	20	158	66	2	2	T6	T2		45
20	28	155	64	2	2.1	T6	T4		54
21	27	155	65	2	2	T6	T2		55
22	23	154	65	2	1.8	T6	T4		53
23	24	153	64	2	2.2	T6	T4		55
24	25	154	63	2	2.2	T6	T4		53
25	25	156	63	2	2.2	T6	T4		52
26	27	157	63	2	2.1	T6	T4		51
27	22	157	67	2	2.1	T6	T4		46
28	23	155	63	2	1.9	T6	T2		48
29	21	153	63	2	2	T6	T4		44
30	22	154	66	2	2	T6	T2		50

31	26	154	68	2	1.9	T6	T2		55
32	25	155	66	2	1.8	T6	T4		54
33	24	154	67	2	1.8	T6	T4		53
34	28	153	66	2	1.8	T6	T4		50
35	26	156	69	2	2	T6	T4		44
36	22	156	70	2	2	T6	T4		40
37	20	157	63	2	2	T6	T4		45
38	21	156	66	2	2.1	T6	T4		46
39	21	158	65	2	2.1	T6	T4		45
40	22	155	64	2	2.2	T6	T4		45
41	21	158	67	2	2.2	T6	T2		50
42	21	158	68	2	2.1	T6	T2		55
43	25	154	63	2	2.1	T6	T4		44
44	27	154	66	2	2	T6	T4		40
45	25	156	69	2	1.8	T6	T4		44
46	24	153	66	2	1.8	T6	T4		46
47	25	153	65	2	1.9	T6	T2		49
48	24	155	63	2	1.8	T6	T4		55
49	24	158	66	2	2	T6	T4		54
50	22	156	67	2	1.9	T6	T4		53
51	23	154	69	2	2.6	T6	T4		50
52	24	155	70	2	2.3	T6	T2		55
53	24	155	70	2	2.6	T6	T4		52
54	25	154	66	2	2.4	T6	T4		55
55	22	155	64	2	2.3	T6	T4		50
56	22	157	66	2	2.6	T6	T4		50
57	21	158	63	2	2.4	T6	T4		50
58	20	154	67	2	3	T6	T4	nausea	54
59	22	154	66	2	2.8	T6	T2		50
60	26	155	68	2	2.8	T6	T2	nausea	50

SL NO	group A	HEART RATE																			MEAN ARTERIAL PRESSURE																			TOTAL EPHEDRINE DOSE(6mg = 1)	FLUID REQUIREMENT (ml)	
		1	2	4	6	8	10	12	14	16	18	20	25	30	35	40	45	50	55	60	1	2	4	6	8	10	12	14	16	18	20	25	30	35	40	45	50	55	60			
1		80	88	92	88	86	84	80	84	90	94	96	96	92	90	84	74	76			90	86	80	76	70	74	70	72	68	70	74	70	74	74	72	76	75				0	950	
2		70	76	78	76	80	84	84	88	86	80	78	74	72	74	76	78	74			100	90	76	70	72	74	78	74	70	72	72	70	66	68	68	70	72				0	1000	
3		84	88	86	90	92	94	88	84	86	88	82	80	74	70	72	68	66	66		83	80	77	71	73	77	73	71	74	71	73	75	72	77	78	77	74	73			0	1050	
4		68	70	76	80	86	88	76	80	74	72	70	72	66	70	68	64	70	74		73	70	70	69	69	68	70	75	75	77	73	73	74	70	70	71	73	71			0	1050	
5		84	88	86	90	82	84	84	82	86	86	84	84	80	80	78	80	76			90	80	86	83	81	88	90	87	83	83	81	79	77	77	74	77	75				0	950	
6		76	80	84	90	80	80	76	74	76	76	78	74	74	72	74	74	76	76	72	93	88	84	77	73	70	70	73	77	77	74	75	71	78	73	73	74	74	73	0	1050	
7		74	80	78	78	88	84	72	72	76	76	74	70	74	78	76	74	72			103	90	90	80	83	81	80	84	85	82	81	81	82	79	80	79	79				0	950	
8		78	70	76	80	86	88	76	80	74	72	70	72	66	70	68	70	68			80	80	76	73	71	77	79	77	73	73	76	76	72	71	75	75	74				0	900	
9		68	76	74	72	80	86	90	88	76	76	76	74	72	70	70	72	74			84	78	76	72	71	71	69	73	73	71	76	75	78	74	74	71	71				0	950	
10		64	66	68	64	74	78	80	86	78	76	74	70	70	70	72	68	68			80	80	79	74	74	75	77	72	72	74	77	71	71	70	72	73	73				0	900	
11		68	70	70	80	84	86	86	80	72	76	76	76	78	74	74	72	74			78	76	75	75	74	72	68	67	66	71	72	71	73	75	71	73	73				0	1000	
12		72	76	76	78	84	88	88	86	88	88	78	70	76	76	74	74	76			77	77	71	68	70	71	71	70	69	69	70	71	73	73	71	70	70				0	1000	
13		74	76	78	76	80	84	84	88	86	80	78	74	72	74	76	78	74			81	70	70	69	69	68	70	75	75	77	73	73	74	70	70	71	73				0	1000	
14		78	80	78	78	88	84	72	72	76	76	74	70	74	78	76	74	72	76		79	71	69	66	69	70	71	71	70	73	71	69	70	70	69	71	71	73			0	950	
15		88	84	88	90	86	78	74	76	76	74	72	76	76	76	74	78	74			79	77	72	71	70	70	71	75	75	76	77	77	74	77	74	72	72				0	1000	
16		80	78	84	88	88	84	80	80	78	78	78	74	78	72	70	70	70			77	70	70	69	69	68	70	75	75	77	73	73	74	70	70	71	73				0	1000	
17		86	80	80	84	80	78	78	76	72	78	76	76	76	74	74	74	74			79	75	75	71	70	71	74	76	75	75	73	74	76	76	74	76	74				0	1000	
18		84	80	88	86	88	86	80	80	80	76	76	76	74	78	76	74	74			80	80	79	74	74	75	77	72	72	74	77	71	71	70	72	73	73				0	1000	
19		80	84	84	88	86	88	86	80	78	78	76	74	78	78	78	72	76			90	90	90	80	83	81	80	84	85	82	81	81	82	79	80	79	79				0	950	
20		80	70	76	80	86	88	76	80	74	72	70	72	66	70	68	64	70	74		95	86	80	76	70	74	70	72	68	70	74	70	74	74	72	76	75	74			0	1050	
21		86	88	88	90	84	86	78	78	78	76	76	74	78	74	72	72	70	74		99	90	90	84	80	77	72	73	75	71	73	71	77	75	71	77	74	72			0	1050	
22		70	66	80	80	82	68	66	66	66	68	68	66	64	64	66	64	64	70		98	90	87	80	78	72	77	73	77	77	75	72	78	77	72	74	76	72			0	1050	
23		64	70	70	78	78	80	74	70	70	70	68	66	66	70	70	70	74	70		79	77	74	74	72	72	71	70	73	74	76	78	78	74	77	75	75	72			0	1050	
24		68	66	70	78	78	80	80	76	70	70	66	66	66	62	62	66	66	68		81	70	68	70	70	71	71	70	72	73	70	69	70	71	71	74	71	75			0	1050	
25		64	70	78	78	80	70	70	66	66	68	68	66	64	70	72	68	68	70		88	80	77	72	71	71	75	75	74	78	73	75	75	77	72	72	74	75			0	1050	
26		78	80	88	88	86	86	88	80	76	76	74	74	70	70	68	68	68	66		89	80	80	74	74	75	75	77	78	80	78	74	77	72	77	77	72	74			0	1050	
27		72	80	80	86	86	88	80	78	74	74	72	66	66	64	62	66	66			79	77	71	68	70	71	71	70	69	69	70	71	73	73	71	70	70				0	1000	
28		74	80	88	88	86	88	70	70	76	74	76	74	76	70	70	68	68			98	90	90	80	83	81	80	84	85	82	81	81	82	79	80	79	79				0	1000	
29		76	76	78	88	88	86	86	86	80	80	80	78	74	74	72	70	74			95	80	76	73	71	77	79	77	73	73	76	76	72	71	75	75	74				0	950	
30		70	80	88	88	86	88	78	70	70	68	66	66	64	66	66	64	64			91	78	76	72	71	71	69	73	73	71	76	75	78	74	74	71	71				0	1000	

31		84	80	88	86	88	86	80	80	80	76	76	76	74	78	76	74	74				90	90	76	70	72	74	78	74	70	72	72	70	66	68	68	70	72			0	1050	
32		88	78	84	88	88	84	80	80	78	78	78	74	78	72	70	70	70	70			80	80	77	71	73	77	73	71	74	71	73	75	72	77	78	77	74	73		0	1050	
33		80	70	70	80	84	86	86	80	72	76	76	76	78	74	74	72	74	76			89	70	70	69	69	68	70	75	75	77	73	73	74	70	70	71	73	71		0	1050	
34		86	88	88	90	84	86	78	78	78	76	76	74	78	74	72	72	70	72			79	74	69	70	71	74	72	70	74	73	76	73	72	71	74	78	79	80		0	1000	
35		76	80	88	86	88	86	80	80	80	76	76	76	74	78	76	74	68				75	70	66	**62**	66	68	68	70	72	75	73	71	69	71	70	70	71			0	950	
36		78	76	78	76	80	84	84	88	86	80	78	74	72	74	76	68	68				83	80	73	72	70	71	74	72	77	72	78	74	72	74	78	76	74			0	900	
37		72	70	78	78	80	70	70	66	66	68	68	66	64	70	72	68	68				83	80	73	72	70	71	74	72	77	72	78	74	72	74	78	76	74			0	950	
38		80	88	88	86	80	76	76	74	74	70	70	70	68	64	64	64	64				85	80	78	72	74	70	70	71	69	71	74	72	70	73	76	77	76			0	1000	
39		86	84	84	86	80	80	78	76	76	78	70	72	72	72	74	74	70				80	80	76	73	71	77	79	77	73	73	76	76	72	71	75	75	74			0	950	
40		76	86	86	88	90	78	70	70	68	66	68	66	66	68	64	66	70				89	80	78	74	74	75	78	77	72	77	73	73	75	70	72	74	74			0	950	
41		66	78	78	80	80	80	78	76	74	70	70	70	72	76	76	76	74				91	88	80	76	77	76	74	75	78	75	73	72	72	75	76	73	74			0	1000	
42		88	88	78	80	80	80	86	76	76	74	76	76	74	70	70	72	72	72			94	90	88	84	80	78	76	77	74	76	78	80	80	81	82	80	80	82		0	1050	
43		70	78	78	80	80	84	70	70	76	76	76	78	72	72	72	72	70				93	90	84	80	76	76	77	79	79	77	79	80	81	82	83	80	81			0	950	
44		72	78	80	84	86	86	80	76	76	74	74	76	76	72	72	70	74				99	90	88	86	80	76	80	82	78	77	74	77	75	74	76	77	74			0	950	
45		78	88	88	90	84	86	78	78	78	76	76	74	78	74	72	70	72				103	90	90	88	80	76	77	80	78	79	80	76	77	79	75	77	75			0	1000	
46		72	80	88	86	88	86	80	80	80	76	76	76	74	78	76	74	74				100	88	80	76	77	80	81	83	81	79	80	80	78	76	77	79	72			0	1000	
47		70	88	92	88	86	84	80	84	90	94	96	96	92	90	84	74	76				86	80	73	72	70	71	74	72	77	72	78	74	72	74	78	76	74			0	1050	
48		76	76	78	76	80	84	84	88	86	80	78	74	72	74	76	78	74	76			88	80	78	72	74	70	70	71	69	71	74	72	70	73	76	77	76	79		0	1050	
49		80	88	86	90	92	94	88	84	86	88	82	80	74	70	72	68	66	66			80	74	69	70	71	74	72	70	74	73	76	73	72	71	74	78	79	80		0	1050	
50		76	70	78	78	80	70	70	66	66	68	68	66	64	70	72	68	68	70			83	80	76	72	**65**	74	72	74	77	74	78	74	75	77	75	74	72	75		1	1000	
51		66	78	84	88	88	84	80	80	78	78	78	74	78	72	70	70	70				79	70	66	**63**	68	68	70	70	72	75	73	71	69	71	70	70	71			1	1050	
52		68	70	70	80	84	86	86	80	72	76	76	76	78	74	74	72	74	70			77	70	**64**	68	70	71	70	69	70	73	73	71	70	71	72	73	71	72		1	1050	
53		80	88	88	90	84	86	78	78	78	76	76	74	78	74	72	72	70	74			89	80	74	72	70	66	**62**	70	74	76	76	77	75	73	75	71	72	71		1	1050	
54		64	76	78	76	78	72	70	70	76	70	70	68	66	66	70	70	70	72			84	80	73	**64**	70	71	73	73	72	70	69	73	77	74	72	70	70	72		1	1000	
55		66	76	76	78	78	70	70	72	74	68	68	70	70	72	70	68	68				83	77	70	66	**63**	69	71	70	72	70	69	70	71	70	74	76	74			1	1000	
56		76	80	84	84	86	80	80	82	80	80	82	82	84	80	80	78	80				82	72	**64**	69	70	71	73	73	73	77	76	74	77	75	75	72	71			1	1000	
57		86	88	80	80	78	76	70	70	68	66	66	68	64	64	66	64	66				88	76	70	68	**62**	70	70	69	70	71	72	71	70	73	74	72	71			1	1050	
58		80	76	76	76	80	68	66	68	68	66	68	64	64	66	64	66	64	66			80	70	**63**	68	69	70	**64**	69	**65**	71	73	70	70	71	70	69	73	72		3	1000	
59		64	80	80	78	76	70	70	76	76	78	74	74	72	76	78	70	70				93	83	73	**64**	68	70	**65**	69	71	72	71	70	74	74	75	72	74			2	1000	
60		68	80	84	84	86	80	80	82	80	80	82	82	84	80	80	78	80				91	80	**62**	68	70	68	**62**	68	71	70	77	75	77	74	75	72	72			2	1050	