PREDICTION OF HYPOTENSION USING INFERIOR VENA CAVA COLLAPSIBILITY INDEX TO GUIDE FLUID MANAGEMENT AFTER SPINAL ANAESTHESIA.

BY:

DR. NAGASOBBANAA MANUKARAN

DISSERTATION SUBMITTED TO SRI DEVARAJ URS ACADEMY OF HIGHER EDUCATION AND RESEARCH, TAMAKA, KOLAR, KARNATAKA

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M.D. (ANAESTHESIOLOGY)

GUIDE:

DR. RAVI M

M.B.B.S, DA, DNB, MNAMS

PROFESSOR

DEPARTMENT OF ANAESTHESIOLOGY

SDUMC, KOLAR

DEPARTMENT OF ANAESTHESIOLOGY
SRI DEVARAJ URS MEDICAL COLLEGE, TAMAKA,
KOLAR-563101

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DR .RAVI MADHUSUDHANA
PROFESSOR,
DEPARTMENT OF ANAESTHESIOLOGY
SDUMC, KOLAR

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PROFESSOR & HEAD OF DEPARTMENT
DEPARTMENT OF ANAESTHESIOLOGY
SDUMC, KOLAR

DATE:

PLACE:

DR. PRABHAKAR
MBBS, MD, MNANS, FICP, AFISC, FIAMS
PRINCIPAL AND PROFESSOR
DEPARTMENT OF MEDICINE
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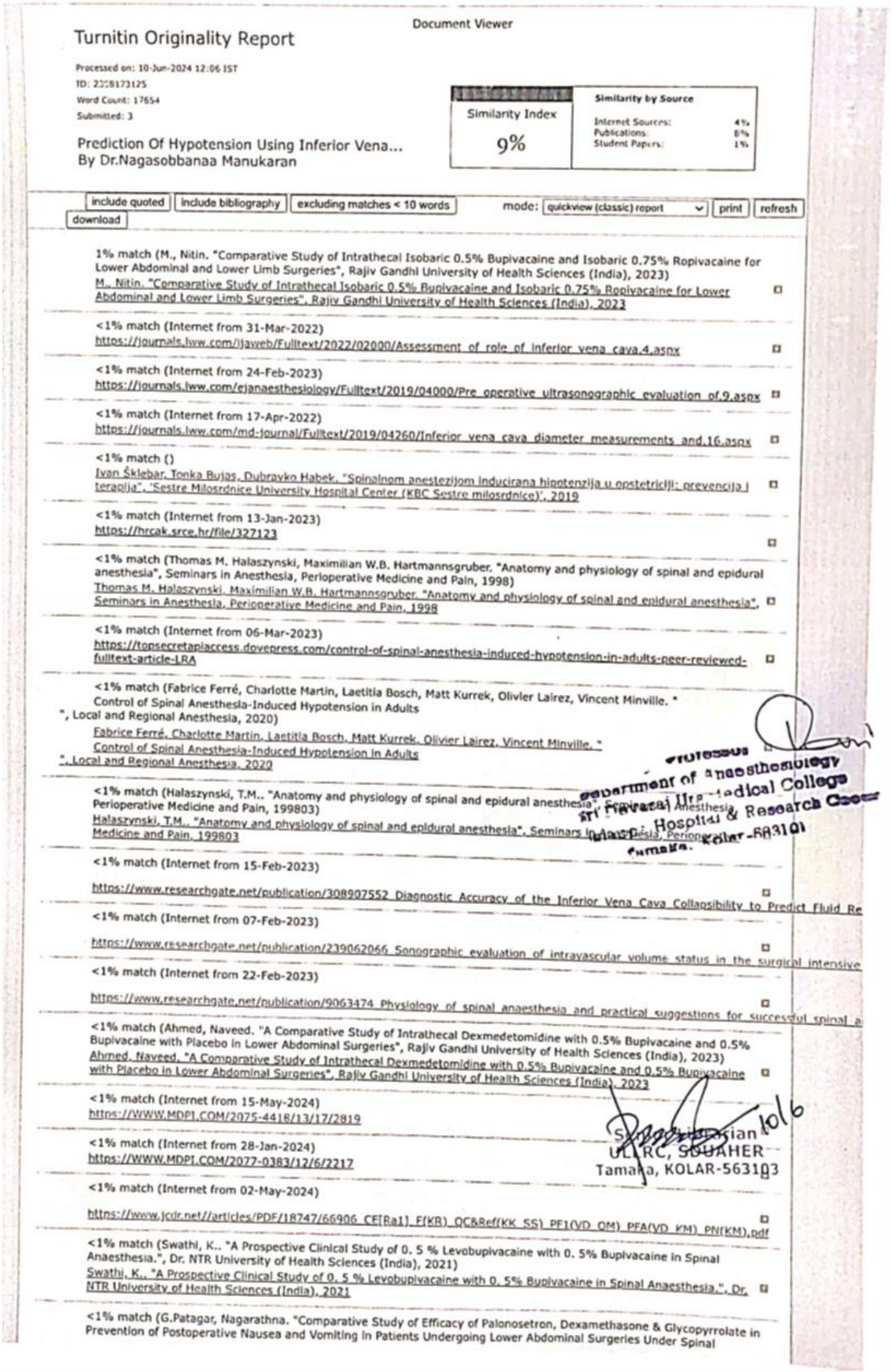

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Sri Devaraj Urs Hospital & Research Centre
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Prediction Of Hypotension Using Inferior Vena Cava Collapsibility Index To Guide Fluid Management After Spinal Anaesthesia

Abstract

Background:

Objective:

Material and method:

Results:

Conclusion:

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Department of Anaesthesiology
Sri Devaraj Urs Medical College
Tamaka, Kolar-563101

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Place: Kolar **DR. NAGASOBBANAA MANUKARAN**

ABSTRACT

BACKGROUND: Assessing the inferior vena cava collapsibility index (IVCCI) through ultrasonography can provide important information on intravascular volume status and the effectiveness of volume resuscitation. The IVCCI's calculation, sensitivity, and specificity make it a reliable indicator of volume responsiveness, offering potential for predicting perioperative hypotensive events and the need for perioperative fluid.

OBJECTIVE: To assess the degree of relationship between preoperative IVCCI and incidence of hypotension post SA.

MATERIAL AND METHOD: The observational single group prospective clinical study conducted at R. L. Jalappa Hospital in Kolar from September 2022 to December 2023 focused on ASA 1&2 non-obstetric patients undergoing spinal anesthesia for elective surgeries. Informed consent was obtained. IVC measurements performed in the supine position right before shifting into operation theatres using an ultrasonography machine. The maximum and minimum anteroposterior diameters of the IVC at the end of expiration and inspiration noted during the same respiratory cycle. The IVCCI calculated. Changes in vital parameters were observed during and after SA. A decrease of more than 30% in blood pressure or a recorded MAP <60 mmHg after SA was considered as hypotension. A P value of 0.05 was considered statistically significant.

RESULTS: Study participants were with an average age of 44 years, balanced distribution between males and females, an average BMI of 25 kg/m2, and falling into ASA grades 1 and 2 in similar numbers. Their vital signs were normal and comparable. Overall hypotension following spinal anesthesia was found in 49% of cases which was

directly correlating with IVCCI values. Hypotension observed in 46% and 9% of cases based on SBP and MAP drop respectively. The research showed that IVCCI is a highly effective test with a sensitivity of 98% and a specificity of 77% to 79% ($P < 0.05$).

CONCLUSION: The study suggests that pre-spinal anesthesia IVCCI measurements are highly predictive of post-spinal hypotension. Increased IVCCI values are strongly associated with hypotension, making IVCCI a valuable tool for identifying high-risk patients for post-spinal hypotension. These findings are consistent with previous research showing that IVCCI can anticipate hemodynamic fluctuations and indicate intravascular volume status.

LIST OF ABBREVIATIONS USED

5-HT3	5-hydroxytryptamine 3 receptor antagonist
ACEI	Angiotensin-converting-enzyme inhibitor
ASA	*American Society of Anesthesiologists*
BJR	Bezold-Jarisch reflex
BMI	Body Mass Index
BP	Blood Pressure
BSA	Body Surface Area
Cm	centimetre
CNS	Central Nervous System
CO	Cardiac Output
COPD	Chronic Obstructive Pulmonary Disease
CSF	Cerebrospinal fluid
CVP	Central Venous Pressure
g	gram
HES	*Hydroxyethyl starch*
HTN	Hypertension
IAP	Intra-atrial pressure
ITP	Intra-thoracic pressure
IVC	Inferior Vena Cava
IVCCI	Inferior Vena Cava Collapsibility Index
LA	Left atrium
LVEDV	left Ventricular End Diastolic Volume
MAP	Mean Atrial Pressure
ml	millilitre
mm	millimetre
PE	Pulmonary embolism
RA	Right atrium
RAP	Right atrial pressure
SA	Spinal anaesthesia
SAIH	Spinal anaesthesia induced hypotension
SBP	Systolic blood pressure
SV	Stroke volume
SVR	Systemic vascular resistance
TMP	Transmembrane pressure
USG	Ultrasonogram

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INTRODUCTION

INTRODUCTION

Spinal anesthesia is an effective and safe procedure that is commonly used in a variety of surgical procedures. Spinal anaesthesia offers benefits like quick action, affordability, technically easy, and minimal side effects. The primary adverse reactions associated with it are hypotension and bradycardia. [1, 2]This could result in consequences such as coronary ischemia and delirium. [3, 4.]

Hypotension is a consequence of vasodilatation caused by the blockade of preganglionic sympathetic fibres, leading to peripheral vasodilatation. Upon reaching the T4–T6 level, spinal anaesthesia results in a reduction in SVR by 23–26%, LVEDV by 20%, and CVP by 2–3 mmHg.[5]

Intraoperative hypotension is commonly observed following spinal anaesthesia, occurring in 15.3% to 33% of cases. The severity of hypotension is influenced by the patient's preoperative fluid status, which in turn is influenced by factors such as ASA physical status, preexisting medical conditions, medications history, and fasting status. Existing prediction models typically focus on non-modifiable risk factors like age over 40, emergency procedures, hypertension history, and baseline systolic blood pressure below 120 mmHg. [1, 6,7]

Neither crystalloid nor colloid (preloading or co-loading) has been proven to be more effective in preventing hypotension.[1]Studies have described that vasopressors such as mephentermine, phenylephrine, or ephedrine can be used to prevent or manage hypotension. However, there is currently no precise predictive tool available to accurately assess the likelihood of hypotension occurring after spinal anaesthesia in high-risk

patients (such as those with old age, cardiac diseases, or autonomic neuropathy) in order to avoid volume overload.[8]

Recently, there has been significant interest in using ultrasound to forecast the occurrence of spinal-induced hypotension[9,10]. This imaging technique also provides real time data about hemodynamic changes. This enables clinicians and anaesthetist to anticipate and prepare for perioperative hypotensive episodes in advance. [11.]Ultrasonography (USG)-guided assessment of IVC measurements and IVCCI provide reliable intravascular volume status and the effectiveness of volume resuscitation. Thus, the IVCCI quantifies how much IVC collapses during inspiration and how much it expands during expiration.

IVC collapsibility index (IVCCI) is calculated as: (max IVC during expiration – Min IVC during inspiration / (max IVC during expiration) [12.] With a sensitivity of 71% and specificity of 81%, IVCCI can rather accurately predict volume responsiveness in spontaneously breathing individuals. [12.] Newer researches have shown that this method can reliably predict the probability of spinal-induced hypotension. [13,14,15]. Nonetheless, there has been a notable variation in the IVC collapsibility index's predictive value across various research investigations.[13,14,15,16]

Though ultrasound evaluation of the collapsibility index (IVCCI) of the inferior vena cava is a simple method for determining fluid responsiveness in critically ill individuals, the current evidence regarding the reliability of ultrasound-guided IVC examination in predicting fluid responsiveness is inconclusive. [18,19]. In view of this, this study was undertaken.

AIMS & OBJECTIVES

OBJECTIVES

OBJECTIVES OF THE STUDY

- To assess the degree of relationship between preoperative IVCCI and incidence of hypotension.
- To determine the ideal volume of crystalloid to be administered intraoperatively for optimum fluid correction based on preoperative IVCCI to prevent post- spinal hypotension.

REVIEW OF LITERATURE

REVIEW OF LITERATURE

Historical Review of Spinal Anaesthesia[20,21]

Cerebrospinal fluid was first identified by Domenico Cotugno in 1764, and its circulation was detailed by F. Magendie in 1825, who also gave it its name. Alexander Wood later introduced the hollow needle and glass syringe in 1853. Around the same time, in 1860 Cocaine was extracted from Erythroxylon coca by Neimann and Lossen while its pain-relieving properties were documented by Schroff in 1862. As for its introduction into medicine, Carl Koller introduced it as a local anesthetic for ophthalmology with the support of Sigmund Freudin 1884.

In 1885, J. Leonard Corning, a Neurologist from New York, conducted the first spinal anaesthesia procedure. He experimented on a dog whereby he unintentionally punctured the dura and injected cocaine into the subarachnoid space. Subsequently, he intentionally repeated the intradural injection using 3% cocaine for duration of 60 minutes, proposing its application in surgical procedures. Corning expressed his belief in documenting this observation, regardless of its ultimate significance, stating, Be the destiny of this observation, what it may, it had seemed to me, on the whole worth recording.

The German Heinrich Iraneus Quinke standardised the lumbar puncture as a rather easy medical procedure in 1891. Coincidentally, in that very year, Essex Wynter detailed the lumbar puncture technique in England.

On August 16, 1898, in Keil, August Bier administered the first planned spinal anaesthesia in a human patient. He administered 3 ml of 0.5% cocaine into the subarachnoid space of a 34-year-old labourer for a lower limb surgery. Following its

successful application on six patients, both Bier and his assistant proceeded to inject cocaine into each other's theca.

In 1905, Heinrich Braun, a renowned German Surgeon, documented the usage of procaine for operative spinal anaesthesia. Additionally, mentioned that the application of intrathecal epinephrine extends the duration of spinal anaesthesia. However, due to concerns regarding potential neurological complications, this method was not widely accepted. It was not until 1945 that Prickett and his colleagues published their findings on the neurological safety of intrathecal epinephrine, thus paving the way for its use in prolonging spinal anaesthesia. Subsequently, in 1966, bupivacaine was introduced for intradural block.[20,21]

Anatomy of spinal cord[22-27]

As an anaesthesiologist, an in depth understanding of the structure of the spinal column, especially the lumbar vertebrae are crucial. The typical length of the spinal cord in males' measures 45 cm, while in females it is 42 cm. The average weight is around 30 g.

The spinal column is composed of 33 vertebrae. [28,29]

Cervical - 7

Thoracic - 12

Lumbar - 5

Sacrum - 5(fused)

Coccyx - 4(fused)

The typical adult spinal column consists of four curves:

1. Cervical curve with anterior convexity

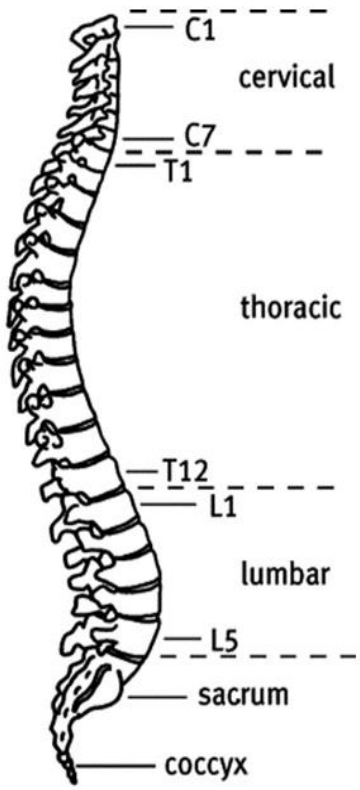
2. Thoracic curve with posterior convexity

3. Lumbar curve with anterior convexity

4. Sacrococcygeal curve with posterior convexity.

The alignment of the spine is particularly crucial when the patient is lying down. The highest point of the spinal curve occurs at the third lumbar vertebra (L3), while the lowest point is at the fifth thoracic vertebra (T5)

FIGURE.1: VERTEBRAL COLUMN



Normally, vertebra consists of several components. First, there is the body, located at the front, which carries and transfers the weight. Intervertebral discs divide it from neighboring vertebral bodies. Secondly, there is the vertebral arch, which is connected to the body. The arch is made up of two pedicles at the front and two laminae at the back, providing a protective enclosure for the spinal cord. At the junction of the pedicles and

laminae is the transverse process, while the meeting point of the laminae is the spinous process. These processes serve as attachment points for ligaments and muscles. Lastly, there are four articular processes, two superior and two inferior, which facilitate joint movement.

THE POSTERIOR INTERVERTEBRAL GAP:

The central portions of the lamina are quite superficial and create the interlaminar foramen. This is the region whereby spinal puncture is commonly done. Typically, in normal position or stretched position, the interlaminar foramen is small and triangular. However, during flexion, the lower articular processes move upwards and widen the foramen, transforming it into a diamond-shaped aperture.

THE LUMBAR VERTEBRAE [30]:

The lumbar vertebrae exhibit distinct characteristics that set them apart from other vertebrae.

The bodies of the lumbar vertebrae are notably large and kidney-shaped, while the vertebral foraminae are triangular in shape and intermediate in size between those found in the thoracic and cervical regions.

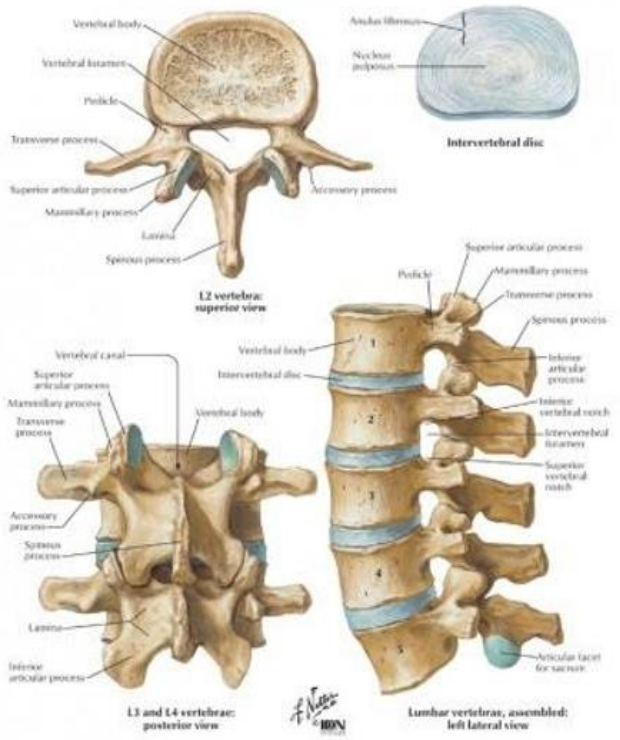
Furthermore, the pedicles exhibit a concise and robust nature, whereas the transverse processes undergo a progressive lengthening pattern from L1 to L3, followed by a subsequent reduction in size. In contrast, the laminae are relatively short. The lumbar spinous process, positioned almost horizontally, assumes a quadrangular shape. Notably, its posterior and inferior edges are thickened, preventing any overlapping due to its elongated, oblong structure.

An important point to note is that the Lumbosacral angle is shaped by the fifth vertebra, as well as its transverse processes. Despite their short and thick nature, these processes are sturdy and emerge from both the side and the arch of the vertebral body.

INTERVERTEBRAL DISCS:

Intervertebral discs form a significant portion, approximately 20%, of the vertebral column's length. These discs are structured with an outer fibrous covering called the annulus fibrosus, which encloses a soft, gel-like material known as the nucleus pulposus. Their key functions involve providing flexibility to the column and acting as shock absorbers. However, with disc degeneration and vertebrae osteoporosis, individuals may observe a decrease in height and potentially develop a kyphotic deformity as they age.

FIGURE 2: LUMBAR VERTEBRAE SUPERIOR, LATERAL AND POSTERIOR VIEW



VERTEBRAL LIGAMENTS:

A thorough comprehension of the ligaments present in the vertebral column, which the spinal needle traverses, is of utmost importance for an anesthesiologist while performing spinal anaesthesia. With practice, the operator can acquire the ability to identify the different sensations of resistance provided by these ligaments as the needle moves forward.

SUPRASPINOUS LIGAMENT:

The ligamentum nuchae is a resilient and compact fibrous band that spans from the tips of the spinous processes of the 7th cervical vertebra to the sacrum. With age, this fibrous band may calcify, making it difficult to successfully insert a spinal needle through it.

INTERSPINOUS LIGAMENT:

The interspinous ligament connects the spinous processes, joining with the Supraspinous ligaments in the posterior region and with the ligamentum flavum in the anterior region.

LIGAMENTUM FLAVUM:

The Ligamentum flavum extends between one lamina's inner surface and lower border and the upper border and outer surface of the lamina below. It is primarily made up of Yellow elastic fibres and makes up more than half of the posterior wall of the vertebral canal, with the bony laminae making up the rest.

The ligamentum flavum is thinnest in the cervical region and thickest in the lumbar region. These ligaments serve as muscle spares, aiding in the restoration of posture after bending and in the maintenance of an upright posture.

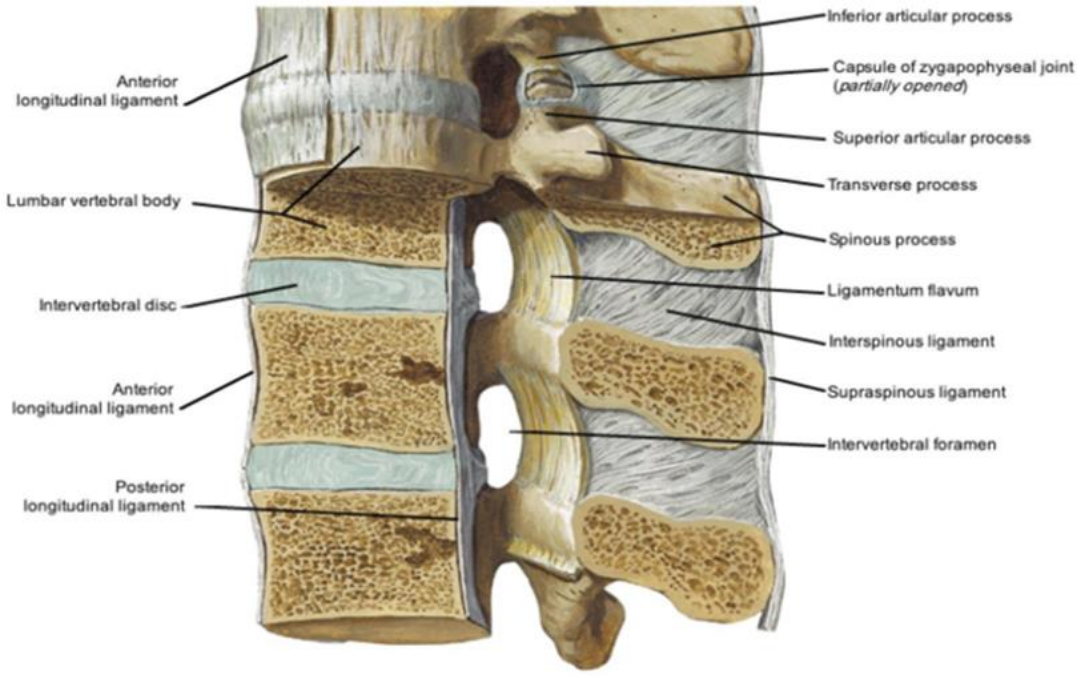
ANTERIOR LONGITUDINAL LIGAMENT

It extends along the front surface of the vertebral bodies from the second cervical vertebra to the sacrum.

POSTERIOR LONGITUDINAL LIGAMENT

It runs along the back surfaces of vertebral bodies, with the basi vertebral veins separating it from them.

FIGURE 3: VERTEBRAL LIGAMENTS



VERTEBRAL CANAL

It spans from the foramen magnum to the sacral tip, with the vertebral bodies and intervertebral discs forming its anterior boundary, and the laminae, ligamentum flavum, and vertebral arch comprising its posterior boundary.

THE VERTEBRAL CANAL CONSISTS OF:

- The meninges, along with the enclosed spinal cord and cerebrospinal fluid (CSF).
- Nerve roots
- The extradural space's vessels, fat, and areolar tissue.

SPINAL CORD [31,32,33]

The Spinal cord is a lengthy part of the CNS that occupies upper two-thirds of vertebral canal. It measures between 42 and 45 centimeters in length and weighs approximately 30 grams. The spinal cord extends from the upper border of the atlas vertebra to the lower border of the first lumbar vertebra or the upper border of the second lumbar vertebra. Above, it connects with the medulla oblongata, while below, it narrows into a conical conus medullaris. A fragile fibrous filament descends from the apex of the conus medullaris to the back of one segment of the coccyx, known as the filum terminale.

The spinal cord features two enlargements, namely the cervical and lumbar enlargements, which correspond to the nerve supply of the upper and lower limbs. The cervical enlargement spans from C3 to L2, while the lumbar enlargement ranges from T9 to T12. Initially, at birth, the spinal cords tip is positioned at the lower border of the L3 vertebra. However, in adults, the spinal cord terminates at the vertebral level of L1- L2.

THE MENINGES:

The spinal cord is surrounded by three layers of membranes, from the outermost to the innermost layer.

DURAMATER:

The spinal meninges consist of a circular sac or sleeve that envelops the spinal cord. It is made up of an Inner (meningeal) layer, which is a continuation of the cranial duramater, and an Outer (endosteal) layer, which is the periosteum lining the vertebral canal. These layers are separated by the extradural space. At the top, the spinal meninges are securely attached to the circumference of the foramen magnum.

Typically, it extends below to the lower border of the S2 vertebra, and then proceeds as the sheath of the filum terminale until it attaches to the periosteum on the back of the coccyx. The primary fibres of the dura mater run longitudinally; when performing a lumbar puncture, the needle should be inserted with its bevel separating these fibres instead of cutting through them.

ARACHNOID MATER:

It is a fragile avascular membrane that lies closely against the dura mater. It is situated above the subdural space and below the subarachnoid space. Superiorly, it extends to the cerebral arachnoid, while inferiorly it expands, covering the cauda equina, and terminates at the lower edge of the S2 vertebra.

PIA MATER:

The brain and spinal cord are closely enveloped by a vascular sheath known as the innermost membrane. Delicate septa extend from this sheath into the substance of the brain and spinal cord. Along the length of the anterior median fissure, the spinal pia thickens anteriorly and forms the linea splendens.

Additionally, it projects into the subarachnoid space and attaches to the dura through a series of pointed processes down to the first lumbar nerve, forming the ligamentum denticulatum.

SUBARACHNOID SPACE:

Situated between the arachnoid and pia mater, the subarachnoid space houses delicate trabeculae and the cranial and spinal nerves. These structures are enveloped by cerebrospinal fluid for support and nourishment. In the cranial and thoracic regions, this space takes on a ring-like appearance with a depth of approximately 3 mm.

However, below the first lumbar region, it becomes circular in nature. This space also connects with the tissue spaces surrounding the blood vessels in the pia mater, which accompany them as they enter the spinal cord.

The fine ramifications that encircle individual nerve cells (known as Virchow robin space) have been characterized as the fragmentation of these continuations. It has been postulated that this serves as a pathway for the diffusion of a spinal anesthetic solution into the spinal cord.

SPINAL SEGMENTS:

The spinal cord is divided into segments by the pairs of spinal nerves that stem from it. There are a total of 31 pairs, with 8 in the cervical region, 12 in the Thoracic region, 5 in the Lumbar region, 5 in the sacral region and 1 in the coccygeal region. The nerve roots within the dura do not have epineural sheaths, which makes them vulnerable to the effects of analgesic drugs when in direct contact.

SPINAL NERVES:

The confluence of the anterior root and posterior root results in the formation of spinal nerves. The anterior root is accountable for efferent and motor functions. Neurons situated in the intermediolateral horn of the spinal cord of T1 to L2 give rise to sympathetic preganglionic axons. Disrupting these fibers may impact the reaction of specific endocrine glands to surgical stress. Conversely, the posterior root is larger than the anterior root and conveys afferent impulses from the viscera and the entire body.

Sensations of pain, touch, temperature, and deep sensation from bone joints, muscles, and tendons are conveyed through each dorsal root containing a ganglion. It also transports efferent fibers from the viscera (along with sympathetic fibers) and vasodilator fibers. Pain and temperature fibers enter the posterior horn and terminate around the cell in gray matter. Subsequently, they cross to the opposite side within three segments and ascend in the lateral spinothalamic tract to reach the thalamus. Deep or muscle sensory impulses ascend in the posterior column and spinocerebellar tracts, while vibration impulses ascend in the posterior column.

SENSITIVITY OF DIFFERENT FIBRES:

Local anesthesia affects all nerve fibers, with smaller, slower conducting fibers being more prone to blockage compared to larger, faster conducting fibers within each fiber type. Myelinated preganglionic B fibers, which conduct faster, are about three times more sensitive to local anesthetics than the slower non-myelinated postganglionic C fibers.

Large A fibres are the most resilient LAs, with Aδ fibres, responsible for pain and temperature sensation, being more susceptible than C fibres, despite their faster conduction. Sensory Aα fibres seem to be more prone to blockade compared to motor Aα

fibres, even though they share the same conduction velocity. This discrepancy could be attributed to the higher frequency at which sensory fibres conduct. The sensitivity to blockade follows the order of preganglionic, temperature, pain, touch, proprioception, and motor fibres.

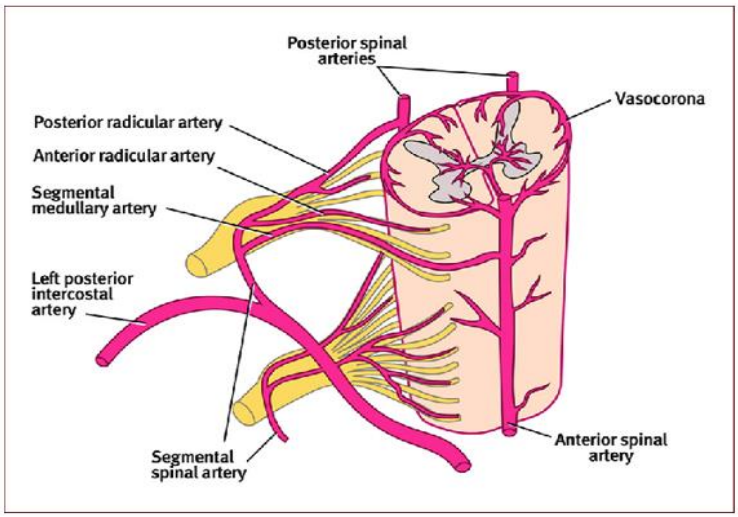
BLOOD SUPPLY OF THE SPINAL CORD[34]:

The spinal cord receives its main blood supply from an anterior artery and two posterior arteries that descend from the level of the foramen magnum.

Anterior spinal artery is a solitary vessel that originates at the foramen magnum through the fusion of a branch from each vertebral artery. It extends along the entire length of the spinal cord and receives contributions from lumbar and other minor arteries. Typically, there are 2-3 connections in the cervical and thoracic regions.

However, there is a singular artery known as the radicular magna (Artery of Adam Kiewicz) that unilaterally supplies the lumbar enlargement. This artery provides nourishment to approximately 3/4 of the cord's substance, including the lateral and anterior columns.

FIGURE 4: BLOOD SUPPLY OF SPINAL CORD



posterior inferior cerebellar arteries. Their main function is to provide blood supply to the posterior one-third of the spinal cord. Additionally, the blood flow is further enhanced by spinal branches of the vertebral, ascending cervical, posterior intercostal, lumbar, and lateral sacral arteries, which pass through the intervertebral foramina.

VENOUS DRAINAGE:

Spinal veins positioned at the front and back of the spine flow into segmental veins in the neck, azygous veins in the thorax, lumbar veins in the abdomen, and lateral sacral veins in the pelvis.

NERVE SUPPLY:

The dura and arachnoid mater's posterior aspect do not contain nerve fibres, leading to the lack of pain sensation during dural puncture. Conversely, the anterior aspect is innervated by the Sino vertebral nerves, with each nerve entering an intervertebral foramen and travelling up one segment and down two segments.

CEREBROSPINAL FLUID [34]:

The term CSF was first introduced in 1825 by French Physiologist F. Magandie. CSF typically refers to a clear and colorless fluid that occupies the spaces and cavities surrounding the CNS. It possesses an osmotic pressure similar to that of plasma, primarily produced by the choroid plexus of the lateral ventricle, and absorbed by the arachnoid villi and granulations.

The average adult produces cerebrospinal fluid at a rate of 25 ml per hour or 600 ml per day. Under normal physiological conditions, the entire volume of spinal fluid is replaced every 6 hours.

CSF features include a specific gravity of 1.006 at 37°C, a total volume ranging from 130-150 mL with the volume in the subarachnoid space of 25-35 mL, and a pressure of 70-180 mmH_2O

COMPOSITION OF CSF:

- Protein 15-45mg/dL
- Glucose 50-80mg/dL
- Non-protein nitrogen 20-30mg/dL
- Chloride 120-130mEq/L
- Sodium 140-150mEq/L
- Bicarbonate 25-30mg/mL
- pH 7.32 (7.27 – 7.37)
- pCO2 48 mmHg
- Cells < 5 cells / mm3

CIRCULATION:

The movement of cerebrospinal fluid (CSF) initiates in the lateral ventricles and traverses the foramina of Munro to access the third ventricle. It subsequently travels through the aqueduct of Sylvius to reach the fourth ventricle, and ultimately exits via the foramen of

Magendie into the cisterna magna. Following this, it passes through the two foramina of Luschka into the cisterna ponti.

Originating from the fourth ventricle, the cerebrospinal fluid flows into the central canal of the spinal cord. It then makes its way from the central subarachnoid space to the spinal subarachnoid space by passing through the foramen magnum. Finally, the fluid is absorbed into the cranial venous sinuses through the arachnoid villi.

FUNCTIONS OF CSF:

CSF, or cerebrospinal fluid, serves several important functions within the body. Firstly, it acts as a protective cushion, providing a barrier between the delicate brain tissue and the rigid cranium. This helps to prevent any potential damage or injury to the brain.

Additionally, CSF plays a role in the drainage of metabolites, ensuring that waste products are efficiently removed from the brain. Lastly, while not its primary function, CSF also contributes to the nutrition and oxygen supply of nerve cells to some extent.

Overall, these functions highlight the vital role that CSF plays in maintaining the health and functionality of the brain.

TECHNICAL ASPECT[34]:

When a needle is introduced into the subarachnoid space, it passes through the following structures:

- Epidermis
- Subcutaneous layer
- Supraspinous ligament

- Interspinous ligament
- Ligamentum flavum
- Areolar tissue or epidural space
- Spinal dura mater

The highest point of the iliac crests typically aligns with a line that intersects the spine of L4 (when standing) or the L4-L5 interspace (when lying on the side). This line is known as the Tuffier's topographic line.[24]

PHYSIOLOGY OF NEURONAL BLOCKADE[35]

EFFECT OF SPINAL ANAESTHESIA ON THE SYMPATHETIC NERVOUS SYSTEM

The impact of spinal anaesthesia on the sympathetic nervous system is crucial due to its ability to block preganglionic sympathetic fibres, resulting in significant hypotension. The level of sensory blockade directly correlates with the extent of sympathetic block and subsequent physiological alterations. Variations in the sensitivity of sympathetic fibres to LAs play a role in determining the degree of sympathetic blockade, as well as the speed of onset and duration. Early responses to spinal anaesthesia include sympathetic denervation leading to vasodilatation.

EFFECT OF SPINAL ANAESTHETIC ON SENSORY NERVES:

Sensory anaesthesia is a result of two pharmacological effects. It involves the blocking of dorsal root ganglia and the blocking of sensory afferent dorsal spinal nerve roots.

The sensitivity to the blockade follows a specific order, with preganglionic pain and temperature being the most sensitive, followed by touch, proprioception, and motor units (specifically B, C, delta, gamma, beta, and alpha type of fibres). Interestingly, the recovery from the block tends to happen in the opposite order.

EFFECT OF SPINAL ANAESTHESIA ON SOMATIC NERVES:

Somatic motor nerves, due to their larger size, exhibit greater resistance to the effects of local anaesthesia compared to other fibres in the subarachnoid space. This results in a differential blockade between motor and sensory fibres.

EFFECT OF SPINAL ANAESTHESIA ON CARDIOVASCULAR SYSTEM:

The cardiovascular changes resulting from subarachnoid administration of local anesthetics do not stem directly from their effects on the cardiovascular system. The response of the cardiovascular system varies greatly between individuals and depends on the extent of spread and subsequent preganglionic sympathetic denervation. The reduction in blood pressure that ensues is a key feature of Spinal Anesthesia.

The anesthesiologist recognizes that the initial sign of the LA being given in the subarachnoid space is the decrease in systolic blood pressure. Spinal anesthesia reduces after load by causing vasodilation in the arteries and arterioles, which also decreases preload through venous pooling. As a result, there is less strain on the ventricle walls during contraction, ultimately leading to enhanced blood flow to the affected tissues and organs.

The absence of vasopressors during the administration of subarachnoid anesthesia leads to a reduction of 10% to 30% in peripheral vascular resistance. However, if SA anesthesia

is administered at a lower level, compensatory vasoconstriction in areas unaffected by sympathetic nerves may prevent a decrease in blood pressure from being observed.

The vasodilation in regions where sympathetic nerves are blocked is not at its maximum potential and can be further enhanced by hypercarbia, acidosis, hypoxia, barbiturates, opioids, and other vasodilating medications. Subarachnoid anesthesia exerts a significant influence on the capillary bed, leading to an elevation in the blood volume within capillary tissues. As a consequence, a greater proportion of the total blood volume is allocated to these peripheral sites in comparison to regular conditions.

EFFECT OF SPINAL ANAESTHESIA ON HEART RATE

The administration of local anesthetic agents during Spinal Anaesthesia can lead to a decrease in heart rate. This decrease is often accompanied by a reduction in cardiac output and mean arterial blood pressure, especially when the spinal level is high. Nausea may precede bradycardia, which can be effectively managed by timely intravenous administration of atropine. The blockade of preganglionic cardiac accelerator fibers and increased venous capacitance, resulting in reduced heart filling, are the underlying causes of bradycardia. These accelerator fibers originate from the first four thoracic spinal segments, and a significant decrease in heart rate is typically observed when the sensory level reaches T4.

When utilizing the Trendelenburg position, the anesthesiologist must exercise caution to prevent the segmental level from rising above T4 if the local anesthetic has not yet taken effect. In instances of bradycardia and hypotension, it is advisable to choose a neutral, supine position instead. It is best to avoid the reversed Trendelenburg position, as it can lead to a further reduction in cerebral perfusion. Bradycardia and hypotension should be

managed with atropine, vasopressors, and intravenous hydration. It is important to note that baroreceptors in the great veins and right atrium take precedence over carotid sinus reflexes, which is why tachycardia does not occur with high SA.

EFFECT OF SPINAL ANAESTHESIA ON CARDIAC OUTPUT

The impact of spinal anaesthesia on cardiac output is noteworthy. Within the initial 15 minutes after subarachnoid blockade, there is a potential increase in cardiac output ranging from 5% to 15% due to the reduction in after load. However, the primary effect of spinal blockade is a decrease in cardiac output caused by a decline in venous return to the heart. The extent of this reduction is influenced by the level of sympathetic denervation. Notably, higher levels of subarachnoid anaesthesia and the reverse Trendelenburg position contribute to a more pronounced depression of cardiac output.35

A decrease of 36% and 21% in cardiac output has been reported when patients with a T6 to T3 sensory level of SA are positioned in a 30- to 49-degree head-up position as opposed to a horizontal supine position. This reduction in cardiac output is likely to impact all organs uniformly. In cases of clinical levels of SA, there is no notable alteration in the distribution of cardiac output or in the absolute levels of blood flow to different tissues and organs.21

HYPOTENSION

SA commonly leads to hypotension, which is the most prevalent adverse reaction. This is primarily attributed to the inhibition of preganglionic sympathetic fibers that transmit motor signals to the smooth muscle in the peripheral blood vessels. The degree of blood pressure decrease is directly proportional to the extent of sympathetic fiber blockage. A decline in peripheral vascular resistance occurs prior to a reduction in cardiac output.

The clinical importance of hypotension lies in its ability to mirror the impact of changes in peripheral vascular resistance and cardiac output on its occurrence. Small decreases in arterial blood pressure are mainly linked to alterations in peripheral vascular resistance, while a systolic blood pressure under 90 mmHg is primarily affected by a decrease in cardiac output. Once viewed as a complication, it is now recognized that hypotension can actually result in reduced bleeding and decreased blood loss during surgical procedures.

The duration of sympathetic denervation usually surpasses the length of the surgical procedure. The resolution of the sympathetic block is influenced by the type and lipid solubility of the local anesthetic used, along with the presence of epinephrine in the anesthetic solution. If the sensory blockade drops below L2, any hypotension detected is not due to a sympathetic blockade caused by local anesthetics.

PHYSIOLOGY OF HYPOTENSION:

Spinal anesthesia leads to hypotension primarily due to the paralysis of preganglionic sympathetic fibers responsible for transmitting motor impulses to the smooth muscles of the peripheral vasculature. The severity of hypotension is directly proportional to the degree of blockage in the sympathetic fibers.

The precise mechanism by which sympathetic blockade reduces blood pressure remained unclear. Two theories were proposed:

- One theory suggested that widespread arterial and arteriolar dilation led to a reduction in peripheral vascular resistance, which could explain most of the decrease in blood pressure.

- The other theory suggested that the decrease in blood pressure was due to a reduction in cardiac output caused by peripheral pooling and a decrease in venous return to the heart.

Although both theories are valid, neither one is sufficient on its own to fully explain all the changes in circulatory physiology that occur due to spinal anaesthesia. The effectiveness of the sympathectomy induced by spinal anaesthesia depends on the extent of the block. The exact level at which arterial blood pressure decreases after central neuraxial block is still unknown. If the block extends beyond the T5 level, it becomes increasingly challenging to compensate for the hemodynamic alteration, resulting in a significant reduction in blood pressure.

Spinal anaesthesia typically leads to hypotension within the initial 15-20 minutes, and if left untreated, the blood pressure reaches its lowest point within 20-25 minutes after the subarachnoid injection. Consequently, the first 30 minutes of spinal anaesthesia are regarded as a critical period due to the potential rapid decrease in blood pressure, which can be alarming in some individuals.

Once the blood pressure has dropped to its minimum, the systolic blood pressure typically rises by 5-10 mmHg within the following 10-15 minutes before stabilizing.

This stabilization continues until the anesthetic effect on nerve roots diminishes. The slight increase in blood pressure is a result of compensatory circulatory activity triggered by reflexes mediated by the sympathetic outflow that has been partially blocked, and possibly due to the return of smooth muscle tone in the denervated part of the peripheral vasculature.

RISK FACTORS FOR POST SPINAL ANESTHESIA HYPOTENSION

Anticipating patients who are prone to spinal hypotension enables improved risk assessment, prompt referral to the suitable healthcare facility, timely administration of vasopressors, and heightened awareness of the anaesthesiologist. Factors that have been identified to forecast the onset of hypotension consist of[37]:

Patient characteristics

Preoperative Anxiety:

The use of a verbal analog anxiety scoring system to evaluate perioperative anxiety has shown that it can predict the occurrence of spinal hypotension. This highlights the significance of effectively addressing patient anxiety before surgery.[38].

Age:

Maternal age is an unchangeable risk factor for the occurrence of hypotension, with the age bracket typically falling between 25 and 35 years.[39,40].

Maternal weight gain:

Inadequate maternal weight gain, defined as less than 11kg, has been linked to the occurrence of spinal hypotension. [41].

Body mass index (BMI):

Inconsistencies in findings are seen when BMI is used as a predictor of spinal hypotension. Nain et al found a correlation between higher BMI and elevated hypotension and vasopressor usage. Conversely, López Hernández et al and Ngaka et al did not find

evidence to support this relationship. The 2018 consensus advises against relying on high BMI as a predictor for spinal hypotension.[42-44].

Foetal birth weight:

A spinal hypotension is frequently observed in cases where the neonatal birth weight exceeds 3900g.[44].

Heart rate:

Preoperative heart rate has been identified as a potential predictor of spinal hypotension, with reported values ranging between 73 and 90 beats per minute. [40,46].

CONSEQUENCES OF HYPOTENSION DURING SPINAL ANAESTHESIA

Spinal anaesthesia induces hypotension through various pathophysiological mechanisms, with the most notable one being the swift initiation of sympatholysis caused by increased nerve fibre sensitivity to LAs during pregnancy.[47,2]. The extent of obstruction in the sympathetic chain correlates with the extent of cranial diffusion of the local anesthetic in the subarachnoid space, making it challenging to anticipate and typically extending beyond several dermatomes above the sensory block level.[48].

Increased sensitivity to LAs, along with aortocaval compression caused by the pregnant uterus, are the primary factors contributing to the higher occurrence and severity of hypotension in pregnant women when compared to non-obstetric patients.[47,2,48]. Pregnant patients also demonstrate heightened sympathetic activity in contrast to parasympathetic activity. [2,50]. Sympatholysis consequently results in an increased level of peripheral vasodilatation and a prevalence of parasympathetic activity. As a result, The decrease in

venous return and cardiac pre-load causes bradycardia, in addition to inducing nausea and vomiting.

The reduced preload consequently leads to a decrease in cardiac output (CO), resulting in systemic hypotension. This condition is further exacerbated by aortocaval compression. [49,50.] A higher sympathetic block leads to a proportional decrease in the activation of compensatory mechanisms through baroreceptors. This, in turn, raises the likelihood of cardioinhibitory reflexes like the Bezold-Jarisch reflex, ultimately resulting in cardiac arrest and mortality [49,51].

Spinal anaesthesia for caesarean section is associated with a higher incidence of nausea and vomiting compared to non-obstetric surgery. These symptoms are mainly attributed to hypotension, which leads to a decrease in cerebral blood flow, temporary brainstem ischemia, and stimulation of the vomiting centers.[52]. Studies conducted with near-infrared spectroscopy (NIRS) have demonstrated that this could also lead to temporary cerebral hypoxia, which is associated with a notable reduction in maternal cerebral blood volume, cerebral oxygen saturation, and oxygenation53. Additionally, this aligns with the finding that administering oxygen through inhalation could potentially avert cerebral hypoxia and lower the likelihood of experiencing nausea [54,55].

Severe and prolonged maternal hypotension can lead to vertigo and a reduced level of consciousness, a scenario that is less likely to happen if the decrease in BP is promptly addressed. SA decreases splanchnic blood flow by around 20%, an effect that is further exacerbated in the presence of systemic hypotension.[56]. Splanchnic hypoperfusion leads to the secretion of emetogenic substances, like serotonin, from the gastrointestinal system, contributing to the pathophysiology of nausea and vomiting. [52.]

Unilateral spinal anaesthesia

Despite the recent significant reduction in the dose in SA, it is still possible to administer unilateral spinal anaesthesia in orthopaedic surgery. This technique aims to create a predominantly one-sided sympathetic block, thereby minimizing any hemodynamic effects.[2,56,57,58] Unilateral spinal anaesthesia can be of significant use to patients who are at a more risk of Syndrome of inappropriate antidiuretic hormone secretion (SAIH), such as older individuals. This can be accomplished by administering the LA The patient should be positioned in the lateral decubitus position, while carefully inserting the needle through the opening towards the side where the operation will be performed.

When employing hyperbaric solutions, it is suggested to orientate the limb to be operated on towards the lower side. Conversely, for hypobaric solutions, the limb should be positioned towards the upper side. It is essential to maintain this posture for around 20 minutes until a predominantly unilateral block is attained.

Bupivacaine remains a popular choice for spinal anesthesia, with suggested doses typically between 3.5 mg and 8 mg.[59] However, a significant disadvantage of lesser- dose spinal anaesthesia is the potential for a block that is not adequately high or a block that wears off prematurely.

Titrating spinal anaesthesia

A potential method is using a spinal catheter to administer a LA, enabling the adjustment of dosage to regulate the degree and speed at which the associated sympathetic block takes effect.[60-63] Titrating spinal anaesthesia results in lower hypotension rates and decreases the requirement for vasopressor medications when compared to traditional spinal anaesthesia, even at low doses..[62,64] The usage of spinal catheters have been

detailed as being inserted using a small Tuohy needle through a paramedian approach, with the requirement that the catheter does not extend more than 2 centimeters into the intrathecal space.

The isobaric bupivacaine local anesthetic (2.5 mg) is administered initially, with the option for re-administration every 15 minutes to achieve the desired sensory level. This approach yields a hemodynamic profile that is notably more stable when contrasted with both traditional spinal and general anaesthesia methods. Notably, even in patients at higher risk, this technique has shown to reduce the occurrence of hypotensive episodes and myocardial ischemia when compared to alternative anesthetic approaches.

The post-dural-puncture headaches have been shown to be significantly influenced by the size of the needle used. Due to the use of a relatively large Tuohy needle in this technique, there is an higher probability of experiencing a post-dural-puncture headache compared to traditional spinal anaesthesia.

Nevertheless, given that this method is primarily employed in older individuals, who face a higher likelihood of experiencing subarachnoid hemorrhage, the higher rate of PDPH may not carry substantial clinical importance. Elderly patients may have a lower susceptibility to this complication, making the risk-to-benefit ratio potentially favorable for this specific patient group.[64,65]

THE EFFECT OF CIRCULATING BLOOD VOLUME AND BODY POSITION

During spinal anaesthesia, pre-existing hypovolemia can result in cardiovascular collapse. The act of positioning the patient in the Trendelenburg position can lead to the occurrence of a cardiac arrest. It is crucial to carefully evaluate the potential cephalad

spread of any hyperbaric solution and anticipate its impact on the patient's hemodynamic. In pregnant women, adopting a left lateral tilt position (5-10°) after spinal anaesthesia helps enhance venous return by reducing compression of the aorta and vena cava caused by the gravid uterus.[66,67,68]

FLUID LOADING

The choice and quantity of intravenous fluid must be carefully determined, taking into account the impact of the sympathetic block, the patient's volume status before the SA, and any fluid losses experienced intraoperatively and postoperatively. In obstetric patients undergoing caesarean section, fluid loading (FL) is commonly employed, either as a sole intervention (44%) or in combination with a vasopressor (53%). Extensive studies have investigated various types of IV fluid and the most appropriate timing for their administration. The administration of intravenous fluids should consider the impact of sympathetic block, the patient's volume status pre-spinal anaesthesia, and any fluid losses during surgery and recovery. Fluid loading is frequently used for obstetric patients undergoing caesarean section, either as a standalone measure (44%) or in conjunction with a vasopressor (53%). Extensive research has been conducted on the different types of intravenous fluids and their optimal timing in this context.[69,70]

Pre-spinal anaesthesia crystalloid preloading is not recommended due to its ineffectiveness and lack of clinical indication. Conversely, colloid preloading with hydroxyethyl starch (HES) has been proven to effectively decrease the occurrence and seriousness of maternal hypotension and lessen the requirement for vasopressors. HES co-loading, when promptly administered after giving spinal anaesthesia (within 5 to 10 minutes), is equally as effective as HES preloading. Despite being a more economical

option, the effectiveness of crystalloid co-loading seems to be reduced, particularly when given at slower infusion rates.[70,71].

In certain unique clinical situations, like patients with pre-eclampsia, fluid loading should only be carried out when absolutely essential (following the initiation of spinal anaesthesia) to prevent fluid overload. Research has indicated that using crystalloid co-loading can decrease the occurrence of hypotension during spinal anaesthesia for caesarean delivery. Crystalloids are preferred due to their widespread use and lack of drawbacks associated with colloids, such as allergic responses, alterations in coagulation, and cost implications.[73]

DRUGS USED IN SPINAL ANESTHESIA

Atropine

Despite its widespread use in treating bradycardia, there is a lack of data regarding the use of atropine in preventing spinal anaesthesia induced hypotension (SAIH). Lim et al conducted a study where atropine was administered intravenously 1 minute after SA, and the results showed that it led to an incremental rise in heart rate is observed, which is dependent on the dosage administered and a decrease in the requirement for vasopressors.[72] Preoperative administration of atropine is currently not advised for patients undergoing spinal anaesthesia. However, administering a small dose of atropine (5 μg/kg) may be beneficial for individuals with a low resting heart rate or those who experience hypotension and bradycardia following spinal anaesthesia.

Ephedrine

Ephedrine has long been recognized as the established vasoconstrictor for SAIH. It acts as a sympathomimetic amine, directly targeting both α- and β- adrenergic receptors, and

indirectly by increasing the release of endogenous catecholamine (norepinephrine). However, patients who undergo prolonged therapy with ACE inhibitors (or angiotensin II receptor antagonists) may experience a decrease in the storage of endogenous norepinephrine. Consequently, the efficacy of ephedrine in these patients may be diminished. Therefore, it is recommended that these patients could benefit from the use of a direct-acting sympathomimetic drug, such as phenylephrine or even adrenaline.[74]

Phenylephrine, a pure α-adrenergic agonist, is preferred as a vasoconstrictor due to its pharmacodynamics, absence of placental transfer, and negligible effect on fetal metabolism. However, ephedrine remains the recommended choice for treating patients exhibiting parasympathetic hyperactivity, which is characterized by bradycardia and hypotension.[74,75,76]

Phenylephrine

Phenylephrine, which acts directly as an α (1)-adrenergic receptor agonist, is primarily used to address intraoperative arterial hypotension. Although physiological studies suggest that α-adrenergic agonists can increase cardiac afterload and reduce venous compliance, the effect of phenylephrine on cardiac output is heavily dependent on the initial venous return condition.[77,78]

When the left ventricular function is preserved, an increase in left ventricular afterload will minimally affect cardiac output. Phenylephrine can aid in optimizing intravascular volume and improving venous return, thereby enhancing cardiac output according to Starling's law. However, if the patient already has heightened sympathetic tone, phenylephrine may raise arterial resistance and reduce cardiac output, especially in instances of impaired cardiac function.[79,80]

Several strategies have been assessed for the administration of phenylephrine. Administering phenylephrine prophylactically, immediately after the intrathecal injection of local anesthetic, to ensure a systolic blood pressure of at least 80% of baseline, has been found to result in increased phenylephrine usage and more effective prevention of arterial hypotension in parturient women and elderly patients, when compared to the rescue method involving a 100 μg bolus of phenylephrine when the systolic blood pressure drops below 80% of baseline.[71,81,82]

It is noteworthy that fixed-rate continuous infusions do not offer advantages over repeated boluses in prophylactic administration regimens. Conversely, utilizing a variable rate prophylactic infusion of phenylephrine has been proven to improve hemodynamic stability, decrease nausea and vomiting, and reduce the need for interventions to maintain blood pressure. Furthermore, research indicates that closed-loop computer-controlled continuous infusion of phenylephrine results in superior blood pressure management compared to manually controlled continuous infusion.[83-85]

The most effective way to minimize the decrease in blood pressure commonly occurs after the administration of SA. is to administer phenylephrine prophylactically through continuous, rate-adjusted intravenous infusion immediately after intrathecal injection. In cases where this is not possible, a fixed rate of 50 μg/minute (with additional boluses of 100 μg if blood pressure falls below 80% of baseline) can be considered. If neither of these methods is viable, clinicians may opt for prophylactic treatment with repeated boluses of phenylephrine.[71,77,84]

Noradrenaline:

Phenylephrine, a potent and selective αadrenergic receptor agonist devoid of any effect o n β-

adrenergic receptors, has been linked to a dosedependent decrease in heart rate and a pote ntial reduction in cardiac output, as discussed previously.In response, some scholars have proposed the utilization of norepinephrine, which also demonstrates mild βadrenergic rec eptor agonist activity. Recently, a randomized controlled trial was conducted by Ngan Ke e et al to compare the effects of norepinephrine and phenylephrine in maintaining arterial blood pressure in parturients undergoing elective caesarean sections under spinal anesthes ia.[86]

The automated continuous administration of norepinephrine (5 μg/mL) proved to be equally effective as phenylephrine in managing blood pressure, while also resulting in a reduced occurrence of bradycardia and a decline in CO (although neonatal outcomes remained unaffected) due to norepinephrine's impact on β-adrenergic receptors. An infusion rate of norepinephrine between 0.07 to 0.08 μg kg−1 min−1 could be considered ideal for commencing norepinephrine infusions to prevent SAH during caesarean section.[87,88]

Serotonin Antagonists

The relationship between arterial hypotension and bradycardia during spinal anesthesia has been attributed to the activation of cardioinhibitory receptors via the Bezold-Jarisch reflex (BJR) and chemoreceptors. Serotonin (5-hydroxytryptamine (5-HT)) released from circulating platelets has been implicated in this process. Studies on animals suggest that serotonin may contribute to the BJR during hypovolemia by stimulating peripheral Type

3 receptors on intracardial vagal nerve endings. In rat models, the activation of the BJR by serotonin was found to be reversible upon blocking NRS TTreceptors.[89,90]

An isolated incident of asystole during SA in humans was documented, and it was observed that the condition exhibited a favorable response to the administration of atropine and ondansetron, a serotonin 5-HT3 receptor antagonist. This observation prompted further investigation into the potential correlation between the BJR and the serotonergic and cholinergic receptors in the afferent and efferent fibers of this vagally mediated reflex during SAIH. Consequently, multiple authors have recommended the use of ondansetron to mitigate the hypotension and bradycardia caused by SA. [91]

It is crucial to highlight that although the administration of 4 to 8 mg of ondansetron just before spinal anesthesia has been effective in preventing a decrease in blood pressure, there is insufficient evidence to support a decrease in the incidence of SAIH. A meta-analysis carried out by Gao et al in 2015, involving 10 randomized controlled trials, suggested that the preventive use of ondansetron could offer potential benefits. However, subsequent research focusing on parturients and patients aged 70 and older has not been able to confirm these positive outcomes.[91-99]

Despite the improvement in arterial blood pressure values following the use of ondansetron, there is a noticeable inclination towards prioritizing the monitoring of easily measurable variables like blood pressure, over crucial yet non-measurable factors such as tissue oxygenation.[98] A recent investigation revealed that the administration of intravenous ondansetron 4 mg resulted in a 26% decrease in The effective dose (ED50) of a preventive phenylephrine infusion for patients undergoing caesarean delivery with combined spinal-epidural anesthesia is being investigated for its efficacy.[99]

Cafedrine and Theodrenaline

A bolus can be given for cafedrine/theodrenaline, while catecholamines usually require dilution and administration through syringe pumps. Bolus injection is faster, making it advantageous in emergencies. Theodrenaline increases vascular resistance, whereas cafedrine has an inotropic effect.[100]

Sakai et al conducted experiments on dogs and rats to determine the most effective ratio of cafedrine and theodrenaline when administered together. They found that a ratio of 20:1 resulted in the best combination for achieving a quick onset and sustained hypertensive effect.[101]

INFERIOR VENA CAVA ULTRASONOGRAPHY

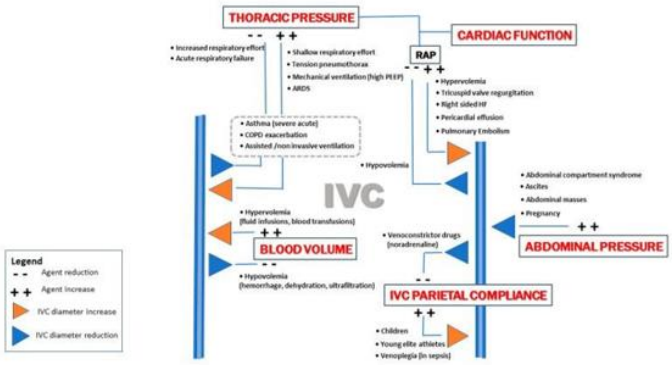
Pathophysiological assumptions of IVC ultrasonography for volume status evaluation

The inferior vena cava (IVC) is the largest diameter vessel in the venous system, located in the retroperitoneal area. It plays a crucial role in returning deoxygenated blood from the lower extremities and abdomen to the right atrium. As a major blood reservoir, the IVC contains 85% of the total plasma volume in the venous circulation. Changes in circulating volume can lead to variations in the diameter of the IVC. Notably, a flat vena cava trauma patients displaying an inferior vena cava (IVC) with a transverse diameter of less than 9 mm at various levels are likely to be experiencing significant hypovolemia.[3]

The position of the patient and the angle at which they are lying can have an impact on the amount of blood circulating in their body and the diameter of the inferior vena cava (IVC) due to the force of gravity. The size of the inferior vena cava (IVC) tends to be smaller when the patient is positioned on the left side, whereas it is larger when the patient is

positioned on the right side. Apart from the circulating volume, there are other significant factors that can cause variations in the diameter of the IVC during the respiratory cycle. These factors include the functioning of the right side of the heart and the difference in pressure between the thoracic and abdominal cavities (Figure 5).

FIGURE 5 Main determinants affecting inferior vena cava diameter



Chest pressure can act directly and/or indirectly (via the RAP) on IVC diameter. The clinical conditions within the grey dashed rectangle can correlate with both types of chest pressure variations.

(ARDS: acute respiratory distress syndrome, COPD: chronic obstructive pulmonary disease, HF: heart failure, IVC: inferior vena cava, PEEP: positive end-expiratory pressure, RAP: right atrial pressure).

INTRATHORACIC PRESSURE

When spontaneously breathing, patients experience an increase in abdominal pressure and a more negative pressure in the thoracic cavity during inhalation. This change in intrathoracic pressure causes a decrease in right atrial pressure. The interaction between the thoracic and abdominal regions has a hemodynamic effect, leading to an increase in blood flow from the inferior vena cava to the right atrium. As a result, the inferior vena cava temporarily decreases in size, resulting in an elevation in SV during inhalation. Conversely, venous return reduces and inferior vena cava widens during exhalation.[103.]

In situations where there is forced inspiration, respiratory distress., or worsening of COPD, the collapsibility of the inferior vena cava (IVC) may be amplified if the intrathoracic pressure (ITP) becomes significantly negative. This leads to an augmented venous return to the right atrium., Raising the ITP and reducing the pressure gradient between the abdominal and thoracic compartments using PEEP in ventilated patients can impede venous return during inspiration. This issue is crucial when managing pre-load dependency situations such as Rt ventricular dysfunction, PE, tamponade, or hypovolemia. Consequently, a sudden decrease in venous return can lead to hemodynamic instability. [105.]

INTRA-ABDOMINAL PRESSURE

The physiology of the inferior vena cava (IVC) can be influenced by intra-abdominal pressure (IAP) to a greater extent than intrathoracic pressure (ITP). This is because IAP affects both the diameter of the IVC and venous return. If the TMP of the inferior vena cava surpasses the crucial closing force in the region below diaphragm, the IVC will remain open. In such cases, elevation of abdominal pressure leads to a reduction in IVC diameter and a temporary increase in venous return. During this process, Acting as the direct source of blood supply, the liver plays a crucial role in circulation. In contrast, the IVC collapses when intrabdominal pressure rises, causing a drop in pressure below the critical closing pressure and leading to a marked decrease in venous return. Consequently, elevated intra-abdominal pressure during inhalation can produce different effects on total and regional IVC venous return

The venous return during the respiratory cycle can be influenced not only by (IAP) but also by the volume status. When there is hypervolaemia, the active descent of the diaphragm leads to a improves the overall IVC flow by improving the return of

splanchnic vein through the IVC Conversely, during hypovolemia, the increased pressure in the abdomen reduces the flow of blood returning to the heart, leading to a decrease in the flow of blood in the inferior vena cava at the level of the diaphragm.[107.]

So, the flow and diameter of the IVC can be influenced by two important factors, namely intra-abdominal pressure and volaemia. As a result, these factors have the potential to affect venous return. Regardless of the volume status, increased abdominal pressure consistently causes a decrease in venous return to the inferior vena cava (IVC), leading to a subsequent decline in cardiac output.[108]

CARDIAC FUNCTION

The cardiac cycle, function of Rt heart , and rhythm all influence the inferior vena cava wall movements .Thus, the hemodynamics of the right atrium (RA) can be observed via the wall movements of the IVC in various physiological and pathological conditions. This is due to the significant correlation between venous return and right atrial pressure. [109] During sinus rhythm, the maximum diameter of the IVC occurs in the presystolic and systolic phases. However, in the presence of AF, the filling of the IVC is affected, making it challenging to evaluate the correlation between the cardiac cycle and the dimensions of the IVC.

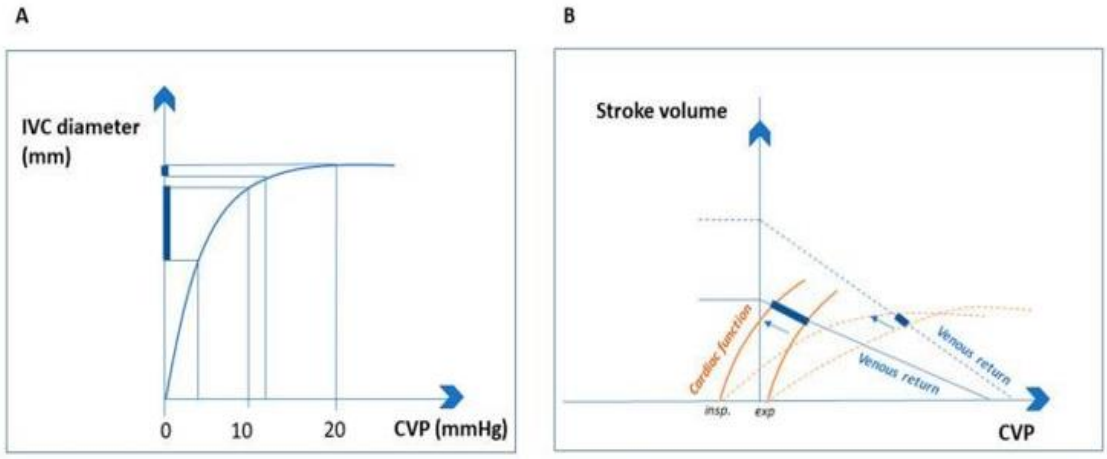
The assessment of the IVC diameter should take into account the physiology of venous return, the function of the right heart, and the interaction between the heart and lungs.

The collapsibility of the IVC during inspiration, in the presence of normal ITP, indicates the right heart's efficiency in reducing RAP. (see Figure 2B)[110].

The clear curvilinear association between IVC diameter and CVP is apparent in light of the heightened ITP (Figure 2A). This relationship exhibits an initial steep phase, wherein a

minimal rise in CVP results in a substantial expansion of IVC diameter, followed by a subsequent plateau phase, where a more increase in CVP leads to less or no dilation of the IVC[105]. In instances of pathological conditions, like acute circulatory failure, alterations in IVC diameter are influenced by both heart functional reserve and residual

FIGURE 6 The diameter of the inferior vena cava in relation to the residual compliance of the venous system.



venous compliance (Figure 6). Consequently, the behaviour of the IVC is determined by an intricate interplay involving the heart, blood volume, and respiratory mechanics operating concurrently in various clinical scenarios (Figure 6).

(A). 1. During the initial upward phase, a slight change in CVP leads to a notable increase in IVC diameter. Subsequent phase, IVC compliance diminishes, and a more substantial rise in CVP results in minimal IVC dilation.

Modified from[105,111]. correlation between IVC diameter and Heart functional reserve

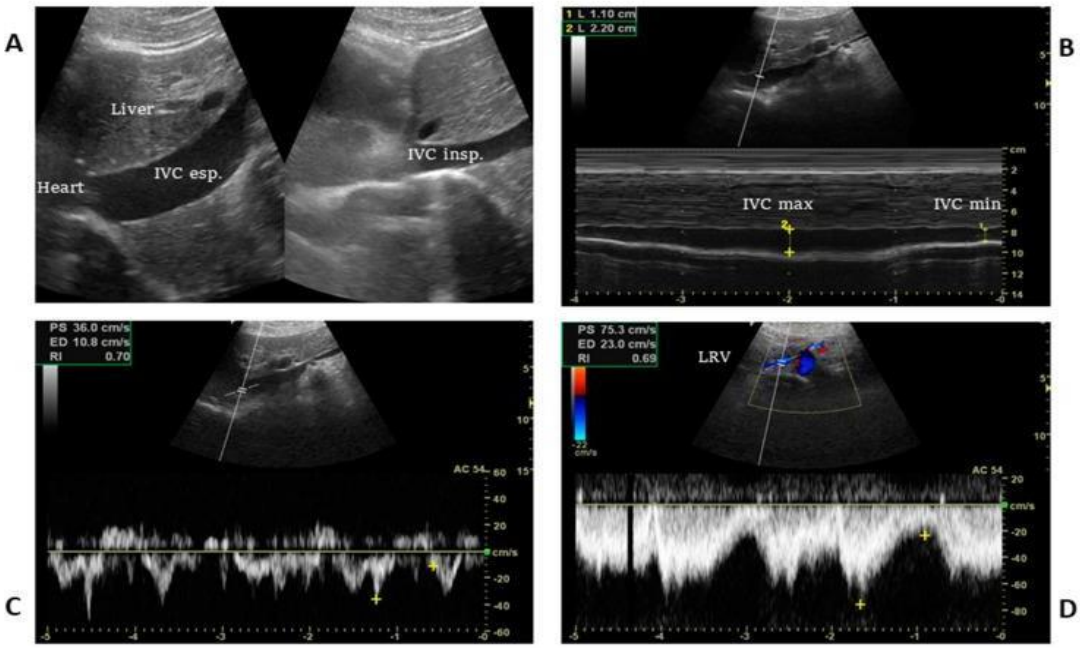
(B). 1. The meeting point of the heart function and venous return curves is shown for individuals with normal (solid lines) and impaired cardiac function (dotted lines). It is only in cases where cardiac function remains unaltered that inspiration can shift the

cardiac function curve to the left, causing a decrease in central venous pressure (CVP) and collapse of the inferior vena cava (IVC).[110]

Ultrasound Technique, Static and Dynamic IVC Indexes

To assess the inferior vena cava (IVC), an alternative approach involves using either a convex probe with a phased array transducer ranging from 2–8 MHz for cardiac evaluation or a lower frequency range of 2–5.5 MHz for abdominal assessment. Typically, the IVC is examined through a subcostal approach, which includes a longitudinal scan covering the veno-atrial junction and the right atrium (RA), allowing for a clear view of the inner walls. However, in situations where a suboptimal or inaccessible subcostal window is encountered, a coronal transhepatic scan along the posterior right axillary line can be employed as a suitable alternative. (Figure 7).

FIGURE 7 A longitudinal scan of the (Inferior vena cava) including the veno–atrial junction



(A) Rt coronal trans-hepatic scan along the posterior rt axillary line

(B) (B-mode (A) is used to identify the (inferior vena cava) and then the Doppler M-mode (B) is Implemented with the sweep velocity at 25 to 50 mm/s depending on the RR in order to include at least three respiratory cycles. The maximum and minimum IVC diameters are used to obtain the collapsibility index) (in the example, cIVC is 50%).

(C) PW Doppler in the IVC

(D) PW Doppler at the outlet of the lt renal vein

PW Doppler in the IVC at the outlet of the lt renal vein since the existence of continuous flow corresponds to low to normal central venous pressure, may offer more data for estimating CVP.

Even though there is no universal standardization, the precise location for measuring the diameter of the inferior vena cava (IVC) is of utmost importance. In patients who are breathing on their own, the alterations in IVC diameter are minimal near the Rt atrium, but they enlarge notably 2 cm below the hepatic vein inlet or at the level of the LRV.[112]. According to the majority of authors, it is recommended to obtain measurements within a distance of 1.5 cm from the junction of the inferior vena cava and the right atrium. [113].

B-mode is utilized to locate the IVC, followed by the application of M-mode Doppler with a sweep velocity ranging from 25 to 50 mm/s, adjusted according to the RR, in order to record a minimum of three respiratory cycles. When patients are breathing spontaneously, the minimum venous diameter may be influenced by their inspiratory effort. To accurately measure the maximal IVC diameter at the end of expiration, it is recommended to perform a maximal inspiration; sniffing manoeuvre.[114] The utilization of indexed IVC size (iIVC),

obtained by dividing IVCmax by body surface area, can enhance the precision of sample accuracy[115].

Furthermore, apart from these fixed parameters, it is consistently advantageous to conduct a dynamic evaluation by utilizing the IVC collapsibility index (cIVC), which can be computed using the subsequent equation:

$$\mathbf{cIVC = (IVCmax - IVCmin)/IVCmax}^{\mathbf{116}}\mathbf{.}$$

The IVC distensibility index, also known as dIVC, is a measure used in mechanically ventilated patients. It is calculated by subtracting the min IVC (IVC min) from the max IVC (IVC max), and then dividing the result by IVC min. Another measure, called ΔDIVC, represents the respiratory variations in IVC diameter. It is calculated by dIVC = (IVCmax − IVCmin)/IVCmin. Additionally, IVC-ADI (IVC Area Distensibility Index) can be used. This index is calculated by max IVC area-min IVC area)/min IVC area × 100%. These indices provide valuable information in assessing the distensibility and variations in the inferior vena cava in mechanically ventilated patients.[117].

Evaluation of the venous spectrometric wave using echo colour and pulsed wave Doppler, while not frequently done in the IVC, may provide valuable information in specific clinical scenarios like stenosis/thrombosis or congenital anomalies. [118].

The technical limitations of US-IVC encompass factors like obesity or pregnancy, the presence of chest or stomach tubes, and the potential for inter/intra-observer variability. Moreover, the lateral movement of the IVC during breathing can contribute to this variability. Consequently, these constraints have a detrimental impact on accurately determining the vein's center and achieving precise measurements in M-mode imaging.[119]

Volume Status Evaluation using IVC Ultrasonography

The association between inferior vena cava (IVC) diameter and its variations throughout the respiratory cycle, CVP, and fluid responsiveness has been extensively studied. In this context, the two aspects are discussed separately based on their pathophysiological basis.

Volume Status Evaluation in Spontaneously Breathing Patient

In general, (there was a statistically significant non-linear relationship observed between the sonographic dimensional parameters of the inferior vena cava (IVC) and central venous pressure (CVP)[32]. The majority of studies indicated a moderate correlation between measurements of IVC diameter or collapsibility and CVP or right atrial pressure (RAP)).[33]. The most accurate diagnostic predictions for a RAP above or below 10 mmHg were achieved with cut-off values of 2 cm diameter and cIVC of 40%. [120]

Based on the most recent American and European guidelines, if the IVC diameter is ≤ 2.1 cm and (there is a collapsibility of more than 50% during inspiration, it suggests a right atrial pressure (RAP) between 0-5 mm Hg. On the other hand, if the diameter is greater than 2.1 cm and there is less than 50% inspiratory collapse, it indicates a high RAP of 10-20 mmHg). In cases where the clinical presentation does not align with the suggested pattern, a mean pressure value of 8 mmHg is utilized.[121].

Standardization of the (IVC) diameter to the BSA was advised for outpatients undergoing haemodialysis. ((i.e., IVC diameter 2.1 cm if BSA > 1.61 m^2, IVC diameter 1.7 cm when BSA < 1.61 m^2)[122] 1. It is advised to standardize the inferior vena cava (IVC) diameter to body surface area (BSA) in outpatients receiving haemodialysis. An indexed IVC size (iIVC) ranging from ≥ 8 to ≤ 11.5 mm/m2 is deemed safe for excluding severe hyper or hypovolaemia in these patients)[115].

Adding pulsed wave Doppler to the inferior vena cava (IVC) can offer more data for estimating central venous pressure (CVP). (If there is a continuous flow from the IVC to the right atrium (RA), it suggests a CVP that is within the low to normal range. Conversely, an interrupted waveform indicates a high right atrial pressure (RAP), but only if it is

accompanied by other ultrasound indicators like the size of the IVC and collapsibility index of the cIVC). Additionally, it has been noted that the inferior vena cava diameter and collapsibility index of the inferior vena cava are associated with the amount of plasma volume removed via ultrafiltration in continuous and intermittent haemodialysis and blood donation. [123]

Volaemic Status Evaluation in Mechanically Ventilated Patients

IVC diameter should not be used for non-invasive CVP assessment in patients on mechanical ventilation, according to the available data. The association between CVP and IVC diameter was found to be modest to moderate in the remaining investigations, and statistically insignificant in 8 of the 16 research included in the latest meta-analysis 1. The results may result from the intricate interplay among ITP, RA pressure, and venous return often complicates the process of reaching definitive conclusions in many instances. [121]

The assessment of fluid responsiveness is dependent on the ability of the inferior vena cava (IVC) to expand by enlarging its diameter under positive pressure ventilation, transitioning from the steep to the plateau section of the IVC-to-central venous pressure (CVP) curve (Figure 2A).[111]. In a study involving 540 individuals experiencing acute circulatory failure from various causes revealed that respiratory changes in the inferior

vena cava diameter were not highly accurate in predicting fluid responsiveness. This could be attributed to factors such as abdominal hypertension and suboptimal mechanical ventilation settings, including low tidal volume, positive end expiratory pressure, respiratory rate, and driving pressures.[124].

Yao and colleagues recently introduced a novel distensibility index, known as VCI ADI, based on IVC area in mechanically ventilated individuals. This index demonstrated

superior sensitivity in predicting fluid responsiveness compared to dIVC, despite having a relatively low specificity of 40.0% (with a cut-off value of 10.2%).[115]

To summarize, when evaluating fluid responsiveness, dIVC demonstrates superior diagnostic performance compared to cIVC in patients who are breathing spontaneously. However, its clinical usefulness in mechanically ventilated patients is uncertain and should only be considered in cases where there is preserved biventricular heart function. Furthermore, the evidence currently available does not sufficiently support the use of dIVC in abdominal surgery, simultaneous abdominal HTN, patients undergoing protective mechanical ventilation, and the paediatric population due to its limited diagnostic accuracy[125]

IVCCI predicts anaesthesia-induced hypotension

IVCCI effectively anticipated post-anaesthesia hypotension in 10 research studies (pooled sensitivity 82%, specificity 81%) (Table 4)[126,127]. 1. In a randomized controlled trial involving 122 patients, utilizing pre-induction ultrasound-guided volume management with an IVCCI cut-off of 42% led to a notable 52% decrease in hypotension incidence, 56% reduction in vasopressor usage, and lower total volume administered

when compared to standard care. Another randomized controlled trial with 160 patients showed that the IVC ultrasound-guided volume management group experienced a 35% relative risk reduction for hypotension (P < .044) and a significantly decreased requirement for vasoactive medications.[128]

Table 1: IVCCI to predict hypotension with anaesthesia induction.

Reference	Type of anaesthesia	IVCCI cut-off pre-induction (%)	SN (%)	SP (%)
Bhimsaria 2022 [129]	General	>50	71	80
Purushothaman 2020 [130]	General	>43	87	94
Rose 2022 [127]	General	>37	90	87
Zhang 2016 [131]	General	>43	79	92
Elbadry 2022 [132]	Spinal	>33	85	93
Ni 2022 [133]	Spinal	>42	84	76
Salama 2019 [134]	Spinal	>45	84	77
Saranteas 2019 [135]	Spinal	>30	82	61
Arican 2019 [136]	Conscious sedation	>45	83	83
Xu 2021 [137]	Conscious sedation	>37	82	61
Pooled data	10 studies	>30 to >50	82	81

Based on pre-induction IVCCI, stroke volume variation, stroke volume, cardiac output, plethysmography variability index, and perfusion index, the risk of hypotension after general anaesthesia was assessed in 100 patients. Through multiple logistic regression

analysis, it was determined that IVCCI was the most crucial independent factor in predicting post-induction hypotension.

IVC ULTRASOUND PEARLS AND PITFALLS

Technical aspects of IVC ultrasound

The inferior vena cava is commonly seen in the long-axis from the subcostal view, although it can also be observed in the mid-axillary line. Enhancing inter-rater reliability can be achieved through proper training and gaining experience. [138]. If the intravascular volume depletion causes the IVC to collapse, there is a possibility of mistaking it for the aorta. However, both vessels can be distinguished by careful observation.

Factors that affect IVC diameter or collapsibility

Table 2 provides a summary of various factors that can lead to the alteration of IVC diameters and IVC CI, potentially causing either over-estimation or under-estimation of intravascular volume.[138] If the patient exhibits factors that contribute to IVC distension, but the IVC appears collapsing, it is improbable that intravascular hypervolemia is present, and vice versa.

Table 2: Factors that affect IVC diameter or collapsibility.

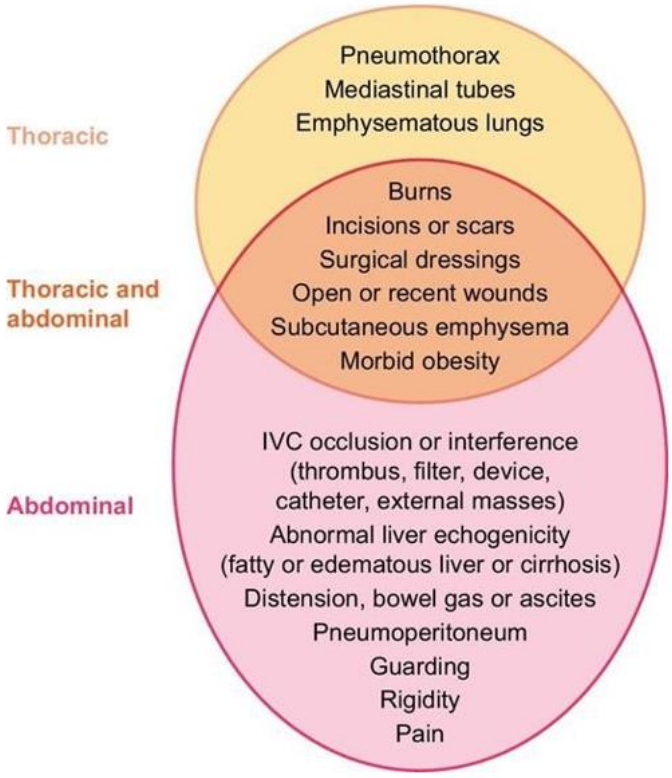
	IVC CI	IVCmax	Comments
Overestimate intravascular volume			
Cardiac tamponade	↓	↑	Blocks forward flow
Severe valvular stenosis	↓	↑	Blocks forward flow
Massive pulmonary embolism	↓	↑	Impairs LV filling
Right ventricular myocardial infarction	↓	↑	Impairs LV filling
Severe tricuspid regurgitation	↓	↑	Impairs LV filling
High PEEP	Minimal Δ	↑	Blocks forward flow
Decreased tidal volume	↓	No Δ	Decreased pressure changes
Decreased inspiratory effort/shallow breathing	↓	No Δ?	Decreased pressure changes. Highly collapsible IVC indicates not hypervolemic
Increased tidal volume (ventilated)	↑	No Δ?	Increased pressure changes
Increased inspiratory effort moving probe in and out of field (diaphragmatic breathing)	↑	No Δ	Not on centre of cylinder. Can try mid-axillary or cross-sectional views
Increased inspiratory effort/deep breathing (sniff)	↑	No Δ	Negative intrathoracic pressure pulls more blood forward into heart
Valsalva manoeuvre	↑	↓	Increased abdominal pressure decreases flow to IVC
Intra-abdominal hypertension	No Δ	↓	Large IVCmax with no collapse indicates not

			hypovolemic
Late term pregnancy, supine position	↑	↓	Gravid uterus compresses IVC and decreases venous return. IVC CI decreases, and IVCmax increases in 15° left lateral decubitus position compared with supine position
Off-centre scan (cylinder tangent effect) [138]	Minimal Δ	↓	Not on centre of cylinder. Try to maximize diameter
Extracorporeal blood replacement therapy	↑?	↓?	Decreased IVV during procedure which increases after blood is returned

Factors that affect IVC visibility

Suboptimal visualization of the subcostal IVC has been documented in 3% to 20% of studies, as outlined in Figure 8 due to various factors.[140].

FIGURE8: Factors that limit IVC visualization by ultrasound.



MATERIAL & METHODS

MATERIAL AND METHODS

Source of data

Study will be conducted non-obstetric patients without cardiac abnormalities/ diseases/complication undergoing spinal anaesthesia for elective surgeries at R. L. Jalappa Hospital, Tamaka, Kolar are selected after informed consent taken.

Method of collection

100 patients undergoing non-obstetric patients without cardiac abnormalities/ diseases/complication undergoing spinal anaesthesia for elective surgeries will be selected and informed consent will be obtained.

Inclusion criteria

- Patients aged 16 to 65 years
- American Society of Anesthesiologists (ASA) physical status grades I– II
- Patients scheduled for non-cardiovascular, non-obstetric surgery under spinal anaesthesia

Exclusion criteria

- Pre-existing hypotension Severe cardiovascular disease
- Decompensated heart failure
- Elevated pulmonary arterial pressure >40 mmHg
- Contraindication for spinal anaesthesia, canal stenosis, pregnant patients, body mass index (BMI) >30 kg/m2,
- Failure to perform SA.

METHODOLOGY

Sample size

Based on the previous literature for an outcome variable on prevalence of hypotension IVCCI guided group compared standard group, with minimum difference of 16.9%, Type I error is 5.0%, and power of the test is assumed to be minimum 90%, and Type II error (10%) the sample size of 100 is adequate for the study (pre-post study) for assessing the degree of relationship between preoperative IVCCI and incidence of hypotension.

Reference article : Ni TT, Zhou ZF, He B, Zhou QH. Inferior Vena Cava Collapsibility Index Can Predict Hypotension and Guide Fluid Management After Spinal Anesthesia. Frontiers in surgery. 2022;9.

Formula

Sample size estimation with two proportions, page 11

$$N = \frac{(Z_{\alpha/2}\sqrt{2\bar{p}(1-\bar{p})} + Z_{1/\beta}\sqrt{p_1(1-p_1)p_2(1-p_2)})^2}{(p_1 - p_2)^2}$$

Where

p_1 and p_2= the proportion of event of interest (outcome) for group I and group II,

$\bar{p} = \frac{(p_1 + p_2)}{2}$,

$Z_{\alpha/2}$ = normal deviate at a level of significance ,

Z_{1-b} = the normal deviate at 1-b% power with b% of type II error, normally type II error is considered 20% or less.

Sampling procedure

- Detailed clinical history of the patient will taken. Routine investigations to be done and checked.

- Prior to surgery, patients will be kept on standard fasting state (8 hours for solid and 2 hours for clear fluid)

- All IVC measurements are to be performed in the supine position right before shifting into operation theatres using an ultrasonography machine.

- All IVC measurements are to be performed by a single person to avoid human error.

- The IVC measurements to be done in paramedian long-axis view via subcostal approach as recommended by the American Society of Echocardiography.

- IVC will be identified using Doppler waveform and phasic collapse during respiration.

- In the long-axis subcostal view, using M-mode measurements of the IVC diameter to be obtained at 2 to 3 cm distal to the right atrium.

- For each patient, 3 scans of IVC and its diameter in various phases will be carried out, not exceeding 10 minutes in total.

- The maximum ($dIVC_{max}$) and the minimum ($dIVC_{min}$) anteroposterior diameters of the IVC at the end of expiration and inspiration are taken during the same respiratory cycle.

- The IVCCI will be calculated using the following formula:

- IVCCI = (dIVCmax –dIVCmin)/dIVCmax × 100%

- Upon shifting into operation theatre, routine monitoring (Heart rate, electrocardiogram, blood pressure measurements, and peripheral oximeter readings) done and noted. No fluid load to be administered to any of the patients before spinal anaesthesia.

- Heart rate (HR) and mean blood pressure (MBP) are to be measured three times before anaesthesia with an interval of 2 minutes between measurements, and the average values will be recorded.

- Under aseptic precautions, after confirming the L3-L4 interspace and proper positioning, spinal anaesthesia should be administered with a 25-gauge Quincke needle.

- The drug is to be slowly injected intrathecally over 10 seconds after free flow of cerebrospinal fluid was obtained. After injection, patients are immediately positioned in the supine position prior to surgery.

- At the same time, non-invasive blood pressure measurements along with other vital parameters will be observed and noted continuously post-spinal anaesthesia. The sensory block level to be determined with pinprick test aiming for a T8-T6 level blockade.

- A fall by more than 30% from the baseline blood pressure value or any recording of MAP <60 mmHg after spinal anaesthesia is to be considered as an episode of hypotension. When MAP is <55mmHg the episode is to be considered as severe hypotension.

- Episodes of hypotension are to be treated using 5 mL/kg of crystalloids infused within 15min.

- After 2 minutes of persistent hypotension or MAP <55 mmHg, appropriate vasoactive drug (ephedrine 5mg, phenylephrine 100 μg, atropine 0.5mg) should be administered to achieve blood pressure within the normal range. The type and dosage of drug administered besides time taken to normalise the blood pressure are also to be noted.

- The medical team will diligently monitor and address any possible complications that may occur during the surgery, including but not limited to nausea, vomiting, discomfort, shivering, or allergic reactions

- After obtaining ethical clearance, study will be registered with CTRI.

Statistical analysis

Study Design:

An observational single group prospective(pre-post) clinical study

Duration of study:

From September 2022 to December 2023

Sampling technique:

Systematic Random sampling

Statistical methods:

Chi-Square test, Fisher exact test, student t test or, or any other suitable method at the time of data analysis

Software's will be used:

SPSS 22.0, R Environment, MedCalc, MS Excel and Word

Randomization: NA

Reference:

Suresh K.P. and Chandrasekhar S (2012). Sample Size estimation and Power analysis for Clinical research studies. Journal Human Reproduction Science,5(1), 7-13.

RESULTS

RESULTS:

Table 3. Age

	Minimum	Maximum	Mean	Std. Deviation
Age (years)	19	76	43.86	13.279

Table 4. Gender

Gender	Frequency	Percent
Male	57	55.9
Female	45	44.1

Table 5. Height, Weight, BMI

	Minimum	Maximum	Mean	Std. Deviation
Height (cm)	152.0	176.0	162.872	5.9470
Weight (kg)	48	95	66.61	10.592
BMI	17.847	37.109	25.11237	3.802517

Table 6. ASA Grade

ASA Grade	Frequency	Percent
1	48	47.1
2	54	52.9

Table 7. Vital parameters

	Minimum	Maximum	Mean	Std. Deviation
Heart Rate	60	120	84.52	12.577
Systolic blood pressure	90	160	122.09	14.399
Diastolic blood Pressure	58	100	76.22	10.340
MAP	70.000	110.000	91.50654	9.930089
SpO2	95	100	98.96	1.024

Table 8. IVC measurements

	Minimum	Maximum	Mean	Std. Deviation
IVC (ins)	.2	3.0	1.375	0.6578
IVC (exp)	1.0	3.3	1.993	0.6009
IVCCI	4.167	80.000	32.37504	20.666019

Table 9. Blood pressure post-spinal anaesthesia

	Minimum	Maximum	Mean	Std. Deviation
Systolic blood pressure	50	130	98.09	16.326
Fall in SBP	0.04	0.60	0.2400	0.12467
Diastolic blood Pressure	32	88	63.47	9.899

Table 10. Incidence of hypotension based on fall in SBP

Incidence of hypotension based on fall in SBP	Frequency	Percent
Yes	47	46.1
No	55	53.9

Table 11. MAP post-spinal anaesthesia

	Minimum	Maximum	Mean	Std. Deviation
MAP	41.333	96.333	75.00980	10.889429

Table 12. Incidence of hypotension based on fall in MAP

Incidence of hypotension based on fall in MAP	Frequency	Percent
Yes	9	8.8
No	93	91.2

Table 13. Incidence of hypotension post-spinal anaesthesia

Incidence of hypotension	Frequency	Percent
Yes	50	49.0
No	52	51.0

Table 14. Association of incidence of hypotension with Age

Age (years)			P value
Hypotension	**Mean**	**Std. Deviation**	
Yes	46.12	13.549	0.092
No	41.69	12.769	

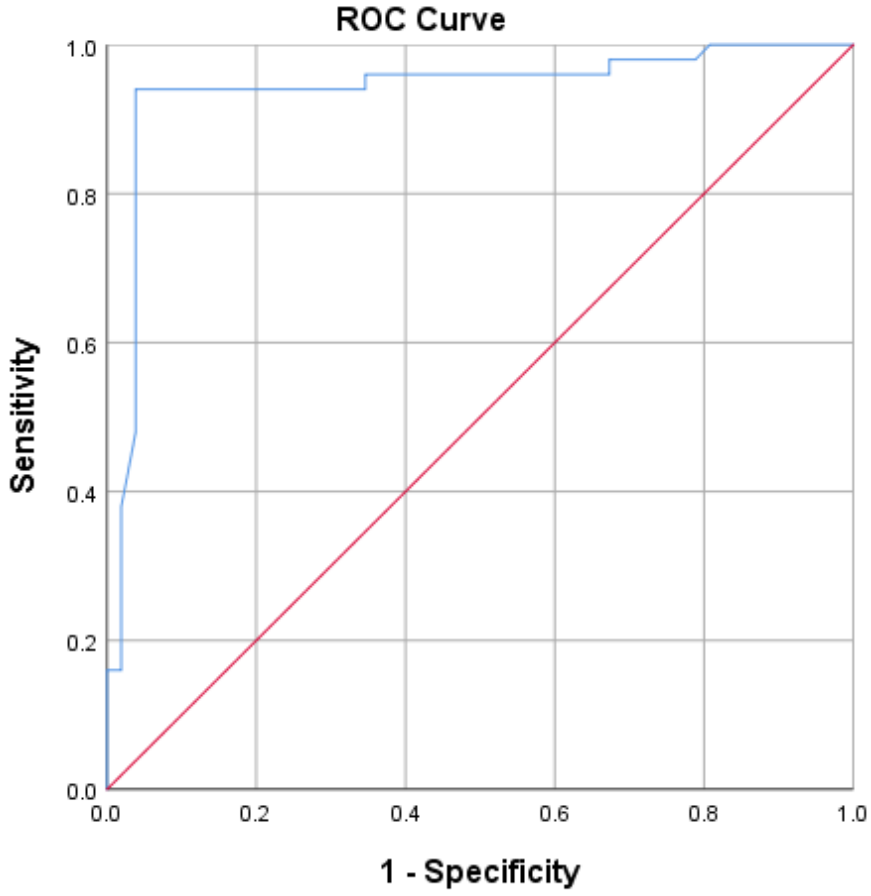
Table 15. Association of incidence of hypotension with IVC parameters

IVC parameters	Hypotension	Mean	S. D	P value
IVC (ins)	**Yes**	0.990	0.4595	**<0.001***
	No	1.746	0.6070	
IVC (exp)	**Yes**	1.924	.6026	0.257
	No	2.060	.5975	
IVCCI	**Yes**	49.32381	14.260156	**<0.001***
	No	16.07815	9.899110	

Table 16. Area Under the Curve- IVCCI in predicting hypotension

Area	Std. Error	P value	Asymptotic 95% CI	
			Lower Bound	Upper Bound
0.939	0.028	0.000	0.884	0.993

Coordinates of the Curve		
Positive if Greater Than or Equal To	**Sensitivity**	**Specificity**
9.10973	98%	78.8%
9.76190	98%	76.9%



Diagonal segments are produced by ties.

DISCUSSION

DISCUSSION

The objective of this investigation was to examine the effectiveness of the inferior vena cava collapsibility index (IVC-CI) in predicting hypotension following spinal anaesthesia, with the objective of guiding fluid management strategies.

The average age of individuals in our research sample was approximately 44 years, with a balanced distribution between males and females, a mean BMI of 25 kg/m^2, and belonged to ASA grades 1 and 2 in nearly equal proportions. Their mean vital parameters were within the normal range, and we found their IVCCI to be 32.4±20.7. The incidence of hypotension was seen to be about 46% and 9% based on the drop in SBP and MAP, respectively. The overall incidence of hypotension post-spinal anaesthesia was seen to be 49%. This incidence of hypotension was found to have a highly significant association with 2 out of 3 IVC parameters studied, which are IVC (inspiration) and IVC-CI values. Our study showed that IVC-CI is a test with very good performance (AUC = 0.94), a high sensitivity of 98%, and a specificity between 77% and 79%.

An important area of study and clinical interest in anaesthesia and critical care medicine is the prediction of hypotension and the use of the IVC-CI to guide fluid management during spinal anaesthesia. Spinal anaesthesia frequently results in hypotension from sympathetic blocking, which causes vasodilatation and a decrease in venous return. Effectively controlling this hypotension is essential to avoiding negative consequences like organ hypoperfusion and cardiovascular instability. In this context, the IVC-CI has shown promise as a tool for managing and predicting hypotension.

The inferior vena cava's (IVC) collapsibility during the respiratory cycle is evaluated using the non-invasive ultrasound measurement known as the IVC-CI. It offers important details regarding a patient's intravascular volume status and fluid response. Monitoring

IVC-CI in the setting of spinal anaesthesia may be able to assist medical professionals in optimising fluid administration, preventing the risk of consequences from both hypovolemia and overabundance of fluid. The usefulness of IVC-CI in anticipating hypotension after spinal anaesthesia has been the subject of numerous investigations.

Zhang et al. (2019) conducted a significant study that showed preoperative IVC-CI readings may accurately predict the incidence of hypotension following spinal anaesthesia. They discovered that patients with greater IVC-CIs had a more risk of hypotension, indicating that proactive fluid management techniques may be beneficial for these people. [141]

The precise definition of post-spinal hypotension (PSH) remains a topic of debate. A comprehensive analysis of existing literature revealed approximately 15 different interpretations of PSH. The most commonly used definitions included a reduction to 80% of the baseline systolic blood pressure (SBP), (as well as criteria such as an 80% decrease in SBP from the baseline or SBP<100 mmHg.[142] The occurrence of PSH has displayed significant variation across different research studies). The prevalence of PSH in the present study (34%) is consistent with findings from various other studies documented in the scientific literature.[143]

Preemptive therapies in clinical practice are made possible by the ability to predict hypotension before it occurs. For example, an anaesthesiologist can identify patients who are more likely to experience hypotension and adjust fluid supply accordingly based on IVCCI data. The objective of this technique is to minimise the occurrence of hypotension-related problems and preserve hemodynamic stability during the perioperative phase.

In addition, IVCCI-guided fluid management provides a more customised approach than conventional techniques that exclusively rely on blood pressure monitoring. Clinicians can obtain real-time insights into a patient's hemodynamic condition and fluid responsiveness by measuring dynamic changes in IVC diameter during respiration.

The determination of intravascular volume status in individuals undergoing mechanical ventilation can be effectively and non-invasively conducted through ultrasound examination of the inferior vena cava (IVC) and the IVC Collapsibility Index (IVC-CI). This method is highly valuable and provides useful insights. Intrathecal LA administration often leads to the occurrence of intraoperative hypotension. This study deems a reduction in average blood pressure exceeding 30% from the baseline level as the criterion for notable hypotension. It is based on the fact that this definition is commonly used in similar studies and because mean blood pressure provides a more accurate measure of tissue perfusion compared to systolic or diastolic blood pressure.[144-146]

Numerous studies have attempted to establish the IVCCI as a reliable tool for predicting fluid responsiveness in resuscitation and intensive care. [147-151] In the field of anaesthesia, the primary focus lies on optimising volume status. [141] Fluid responsiveness refers to a significant increase of 10% to 15% in cardiac output following the administration of a fluid bolus. However, it is worth noting that only a small number of anaesthesiologists regularly utilise cardiac output monitoring. [152,153] Thus, most anaesthesiologists rely on basic monitoring techniques such as blood pressure and heart rate to assess hemodynamics. This is precisely why incorporating bedside IVC ultrasound can be beneficial in identifying patients with depleted volume who require fluid optimisation.

Devi et al. conducted a study in 2021 in a tertiary hospital in southern India on ASA 1- 2 patients aged 18–60 years posted for orthopaedic surgeries and found that patients who

had an IVCCI of 40% before surgery and received fluid bolus showed a significantly lower incidence of post-spinal hypotension (PSAH) compared to the patients with no IVCCI assessment and fluid therapy prior to surgery. It was also concluded that the requirement for vasopressors was lower in these patients. The relative risk reduction of PSAH was seen to be 52% in this study. [154]

Ayyanagouda et al. demonstrated in their 2020 research a 40% decrease in the relative risk of post-spinal anaesthesia hypotension (PSAH) among patients who underwent inferior vena cava ultrasound (IVCUS)-guided volume optimisation prior to the procedure, in comparison to those who did not receive IVCUS assessment. Additionally, the study indicated a decreased need for vasopressors in the former group[155].

While Ni TT et al. (2021) found that the IVCCI exhibited a sensitivity of 83.9%, a specificity of 76.3%, and a positive predictive value of 84% in predicting the occurrence of hypotension following spinal anaesthesia, [156] Several adverse effects, including delirium and cardiac ischemia, can result from SIH. [157,158] Patients with cardiovascular risk factors may see significant differences in their prognosis from transient hypotension. Nonetheless, healthy individuals may usually manage it well, with the only side effects being nausea, vomiting, and dizziness. [159] In their retrospective investigation, Monk et al. found a correlation between The mortality rate within 30 days following non-cardiac surgery and occurrences of intraoperative hypotension.[160] Despite this data, the patient-adapted and monitoring-based approach to the administration of fluids and vasopressor medications for the prevention or treatment of SIH is frequently ignored. [161]

1. The IVCUS assessment surpasses MAP, HR, and CVP measurements for induction in the operating room setting when used as a perioperative screening and monitoring instrument. [162,163]

Nevertheless, a study conducted in 2021 by Chowdhury et al. yielded contradictory results. They determined that neither IVCCI nor CAPVV proved to be reliable indicators of post-spinal hypotension (PSH) in adult patients undergoing infra- umbilical surgery under spinal anaesthesia. However, the combination of ultrasound parameters and baseline mean blood pressure (MBP) showed potential for predicting PSH. It is worth noting that this disparity in findings could potentially be attributed to the limited sample size utilised in their study. [164]

Likewise, the 2021 study by Roy et al. yielded similar results, failing to establish the efficacy of IVC ultrasonography in predicting blood pressure changes following spinal anaesthesia in patients who were breathing spontaneously. The researchers determined that IVCCI lacks the same predictive capability for hypotension in spontaneously breathing patients undergoing spinal anesthesia compared to mechanically ventilated patients. [165]

IVCCI use before spinal anaesthesia is controversial, as per certain research, because it displays insufficient fluid reserve and induces sympathetic denervation. While one study did not find the IVCCI to be predictive in Patients post knee operation,[29] Another found it to be helpful tool for providing ultrasound-guided fluid treatment to reduce the severity of hypotension. [145] A more recent study discovered that the caval-aorta index was a more reliable predictor than the IVCCI. Therefore, this feature should be the focus of future research.[146]

In their study in 2019, (Jaremko et al. also determined that an elevation in IVC-CI does not aid in the detection of severe hypotension after spinal anaesthesia following the administration of normal saline in spontaneously breathing patients undergoing elective knee arthroplasty. The study found that the circulating blood volume and heart preload

remained unchanged after the intravascular administration of crystalloids during spinal anaesthesia).[167]

Another study conducted in 2018 also had comparable outcomes, leading to the conclusion that IVC-CI does not serve as a predictor for hypotension or bradycardia linked to SA. Moreover, the research findings concluded that the influence of SA on the variability of IVC does not have any effect on the circulating blood volume or heart preload. [168] However, discrepancies may have arisen due to variations in patient populations and anaesthesia practices across the studies.

In 2019, Saranteas et al. demonstrated that, in older patients, (the pre-operative dIVCmax/IVCCI ratio is a more reliable indicator of spinal-induced hypotension than IVCCI and other echocardiographic parameters. It was determined that the preoperative dIVCmax/IVCCI ratio is a more accurate predictor of spinal-induced hypotension compared to the IVCCI and other echocardiographic measurements). Furthermore, the dIVCmax/IVCCI ratio and the patients' age can be utilized as indicators for spinal- induced hypotension in the elderly population. [168]

In a prospective study of 2021 conducted on ASA 1-4 geriatric patients undergoing spinal anaesthesia by Aslan et al., it was concluded that, as expVCI is noninvasive, simple to use, and quick, it can be chosen over invasive procedures for predicting post- spinal hypotension in older persons. The diameter measurement of expVCI acquired through USG prior to spinal anesthesia has demonstrated its effectiveness in predicting the occurrence of post-spinal hypotension through the analysis of lactate and pH levels, which are crucial blood gas parameters.

As the expVCI determination is non-invasive, simple to use, and quick, it can be preferable to invasive procedures for predicting post-spinal hypotension in older persons.

The diameter value of the expVCI, determined through USG prior to SA, acts as a valuable indicator for post-spinal hypotension. This assessment considers the crucial blood gas parameters of lactate and pH.

However, the results of our study showed no significant association between IVC (exp) and the incidence of hypotension, and the difference can be attributed to the differences in the characteristics of our study population, which included individuals over the age of 18, including older adults with ASA grades 1 and 2. [169)]Another study published by Salama et al. in 2019 demonstrated that the pre-operative IVCCI and IVC:AO index serve as reliable indicators for the likelihood of PSAH. Nevertheless, the IVC:AO index proves to be a stronger predictor compared to the IVCCI. [145]

IVCCI, however, has potential, and putting it into regular practice would need careful thought. IVCCI findings can be impacted by variables such as patient-specific traits (like respiratory mechanics) and operator variability in ultrasonography measures. Standardisation of methods and procedures is necessary to guarantee the precision and consistency of IVCCI measurements in various contexts.

In conclusion, an emerging paradigm in perioperative treatment is the prediction of hypotension using IVCCI to direct Fluid control and supervision following SA. In management of spinal anaesthesia-induced hypotension, doctors can improve patient safety and outcomes by utilising ultrasound technology and dynamic fluid responsiveness indices. To fully realise the clinical benefits of this technique, more study and development of IVCCI-based procedures are required.

Despite the encouraging results, there are a few drawbacks to take into account. The findings of our study may not be widely applicable due to the limited sample size. To validate our findings, larger cohort studies are needed in the future. Furthermore, the

prognostic efficacy of IVC-CI in a particular surgical context—spinal anaesthesia— was the main emphasis of this investigation. IVC-CI's suitability as a general predictor of hypotension would be strengthened by additional research conducted on a variety of surgical procedures and patient groups. This was a single-centre study, and it is advised to conduct multicentre studies to determine the ideal cut-off point for these factors.

CONCLUSION

CONCLUSION

Our research indicates that pre-spinal anaesthesia IVC-CI measurements demonstrate high predictive accuracy for post-spinal hypotension. Higher IVC-CI levels have been significantly linked to hypotension, which implies that IVC-CI may be a helpful tool for identifying patients who are at risk. This result is in line with a number of other studies that show IVC-CI can predict hemodynamic changes and reflect intravascular volume status.

It is important to give considerable thought to incorporating IVC-CI into regular practice. Although our research indicates that IVC-CI could improve risk classification and direct decisions regarding fluid management, putting it into practice might require funding and personnel training. Further studies should examine the long-term effects and cost-effectiveness of integrating IVCCI into clinical algorithms.

In conclusion, this study's findings highlight IVCCI's potential as a prognostic tool for post-spinal hypotension. Through the utilisation of this non-invasive metric,medical professionals can customise fluid management tactics to meet the specific requirements of each patient, which could potentially lower the frequency of hypotensive episodes and enhance perioperative care. In order to validate and improve these results and create individualised, evidence-based perioperative treatment plans, more research is necessary.

SUMMARY

SUMMARY

Hypotension is a common adverse reaction associated with spinal anaesthesia. Administering pre-emptive intravenous fluid loading before spinal anaesthesia may pose a risk of volume overload, despite its potential benefits. The IVC-CI assessment is widely used in emergency and critical care settings due to its non-invasive nature ease of teaching, and performance, as well as its strong validation. It is a good tool for perioperative screening and treatment monitoring in the surgery theatre and induction area. Even for patients who are at high risk, the majority of anaesthesiologists employ relatively rudimentary hemodynamic monitoring during surgery, mostly measuring blood pressure and heart rate. It is crucial to remember that TTE can quickly and painlessly monitor IVC and that these measurements can accurately forecast intraoperative spinal-induced hemodynamic disturbances.

Numerous researches have addressed the prediction of fluid responsiveness., and a number of dynamic markers of fluid responsiveness, including IVCCI, have been carefully examined.[170,171] Administration of fluids guided by pre-spinal IVC ultrasound has been proven to optimise patient volume status, leading to a reduced occurrence of hypotension after spinal anaesthesia. This, in turn, decreases the necessity for interventions to manage hypotension. [145]

Accordingly, our study's findings added to the body of evidence by demonstrating that IVCCI is a test with a high sensitivity and specificity for post-spinal hypotension as well as a good predictive value. In our investigation, we observed a 49% occurrence rate of hypotension after administering spinal anesthesia. Among the three IVC parameters that

were analyzed, the incidence of hypotension was shown to be substantially correlated with the IVC (inspiration) and IVCCI values.

Optimising perioperative care is one of the clinical implications of integrating IVC-CI into fluid management procedures. Clinicians can reduce the risk of hypotension by proactively adjusting fluid administration tactics in patients with greater preoperative IVC-CI levels. Clinicians can utilise IVCCI to direct customised fluid resuscitation techniques rather than using a one-size-fits-all approach to fluid delivery. Prior to administering SA, it is advisable to perform an IVC ultrasound examination for high-risk patients, the elderly, and individuals suspected of hypovolemia as part of POCUS.

This method is consistent with the principle of goal-directed fluid treatment, which bases its interventions on tailor-based dynamic measures of fluid responsiveness. This tailored approach may result in better patient outcomes and a decrease in the morbidity brought on by perioperative hypotension. However, to determine the viability and long-term economic viability of IVC-CI as a screening and therapeutic tool, as well as its effect on postoperative hemodynamic problems, more research is necessary.

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ANNEXURES

PROFORMA

"Prediction of Hypotension Using Inferior Vena Cava Collapsibility Index to Guide Fluid Management After Spinal Anaesthesia."

Investigators: Dr. Nagasobbanaa Manukaran / Dr. Ravi M

IP No. of the patient: **Age/Sex:** **ASA grade:**

- **General physical examination:**

Height:		Weight:	
Pulse rate:		Blood pressure:	

Pallor/icterus/cyanosis/clubbing/lymphadenopathy/edema

- **Surgery:**

- **Baseline vitals:**

HR:	BP:	MAP:	SpO2:

- **Measurements:**

Minimum diameter of IVC	Maximum diameter of IVC	IVCCI

Readings immediately prior to SAB

HR:	BP:	MAP:	Fall in BP (mmHg)

Readings immediately after SAB

Minutes after SAB	HR	BP	MAP	Fall in BP (mmHg)
1st minute				
2nd minute				
3rd minute				
4th minute				
5th minute				
10th minute				
15th minute				

PATIENT INFORMATION SHEET

"Prediction of Hypotension Using Inferior Vena Cava Collapsibility Index to Guide Fluid Management After Spinal Anaesthesia."

Investigators: Dr. Nagasobbanaa Manukaran / Dr Ravi M

Study location: R L Jalappa Hospital and Research Centre attached to Sri Devaraj Urs Medical College, Tamaka, Kolar.

Details: **N**on-obstetric patients without cardiac abnormalities/diseases/complication undergoing spinal anaesthesia for elective surgeries at R. L. Jalappa Hospital, Tamaka, Kolar are included in the study. Patients who meet exclusion criteria will be excluded from the study.

This study aims to assess the degree of relationship between preoperative IVCCI and incidence of hypotension and to determine the ideal volume of crystalloid to be administered intraoperatively for optimum fluid correction based on preoperative IVCCI to prevent post-spinal hypotension. Patient and the attenders will be completely explained about the procedure being done under ultrasound guidance. Throughout the study if any form of payment/purchase is necessary, it will be completely borne by the investigator. No extra cost will charged for patient throughout the study.

Please read the above mentioned information and discuss with your family members. You can ask any question regarding the study. If you agree to participate in the study we will collect required information and relevant history will be taken. The collected information will be used only for dissertation and publication.

All information collected from you will be kept confidential and will not be disclosed to any outsider. Your identity will not be revealed. There is no compulsion to agree to this study. The care you will get will not change if you do not wish to participate.

You are required to sign/ provide thumb impression only if you voluntarily agree to participate in this study.

For further information contact:

Dr. Nagasobbanaa Manukaran
(Principal investigator)
Contact number: 9791859349

ರೋಗಿಯ ಮಾಹಿತಿ ಹಾಳೆ

ಕೆಳಮಟ್ಟದ ವೆನಾ ಕಾವಾ ಕೊಲ್ಯಾಪ್ಸಿಬಿಲಿಟಿ ಸೂಚ್ಯಂಕವು ಹೈಪೊಟೆನ್ಷನ್ ಅನ್ನು ಊಹಿಸಬಹುದು ಮತ್ತು ಬೆನ್ನುಮೂಳೆಯ ಅರಿವಳಿಕೆ ನಂತರ ದ್ರವ ನಿರ್ವಹಣೆಗೆ ಮಾರ್ಗದರ್ಶನ ನೀಡುತ್ತದೆ

ತನಿಖಾಧಿಕಾರಿಗಳು: ಡಾ. ನಾಗಸೊಬ್ಬಣ್ಣ ಮನುಕರನ್ / ಡಾ ರವಿ ಎಂ

ಅಧ್ಯಯನ ಸ್ಥಳ: ಆರ್ ಎಲ್ ಜಾಲಪ್ಪ ಆಸ್ಪತ್ರೆ ಮತ್ತು ಸಂಶೋಧನಾ ಕೇಂದ್ರವು ಶ್ರೀ ದೇವರಾಜ್ ಅರಸ್ ವೈದ್ಯಕೀಯ ಕಾಲೇಜು, ಟಮಕ, ಕೋಲಾರ.

ವಿವರಗಳು: ಕೋಲಾರದ ಟಮಕಾದ ಆರ್.ಎಲ್.ಜಾಲಪ್ಪ ಆಸ್ಪತ್ರೆಯಲ್ಲಿ ಐಚ್ಛಿಕ ಶಸ್ತ್ರಚಿಕಿತ್ಸೆಗಾಗಿ ಬೆನ್ನುಮೂಳೆಯ ಅರಿವಳಿಕೆಗೆ ಒಳಗಾಗುವ ಹೃದಯ ವೈಪರೀತ್ಯಗಳು/ರೋಗಗಳು/ಸಂಕೋಚನವಿಲ್ಲದ ಪ್ರಸೂತಿ-ಅಲ್ಲದ ರೋಗಿಗಳನ್ನು ಅಧ್ಯಯನದಲ್ಲಿ ಸೇರಿಸಲಾಗಿದೆ. ಹೊರಗಿಡುವ ಮಾನದಂಡಗಳನ್ನು ಪೂರೈಸುವ ರೋಗಿಗಳನ್ನು ಅಧ್ಯಯನದಿಂದ ಹೊರಗಿಡಲಾಗುತ್ತದೆ.

- ಈ ಅಧ್ಯಯನವು ಪೂರ್ವ-ಪೂರ್ವ ivcci ಮತ್ತು ಹೈಪೋಟೆನ್ಷನ್ ನ ನಡುವಿನ ಸಂಬಂಧದ ಮಟ್ಟವನ್ನು ನಿರ್ಣಯಿಸುವ ಗುರಿಯನ್ನು ಹೊಂದಿದೆ ಮತ್ತು ಪ್ರಿಸೈಆಪರೇಟಿವ್ ivcci ಯ ಆಧಾರದ ಮೇಲೆ ಗರಿಷ್ಠ ದ್ರವದ ತಿದ್ದುಪಡಿಗಾಗಿ ಇಂಟರ್ಪಲ್ಡಿಕೇಟೆಡ್ ಇಂಟ್ರಾಲ್ಡ್ ಅನ್ನು ನಿರ್ಣಯಿಸುವುದು. ರೋಗಿಗಳು ಮತ್ತು ಹಾಜರಾಗುವವರು ಅಲ್ಟ್ರಾಸೌಂಡ್ ಮಾರ್ಗದರ್ಶನದಲ್ಲಿ ನಡೆಯುವ ಕಾರ್ಯವಿಧಾನದ ಬಗ್ಗೆ ಸಂಪೂರ್ಣವಾಗಿ ವಿವರಿಸಲಾಗುತ್ತದೆ. ಯಾವುದೇ ರೀತಿಯ ಪಾವತಿ / ಖರೀದಿ ಅಗತ್ಯವಿದ್ದರೆ, ಅದನ್ನು ಸಂಪೂರ್ಣವಾಗಿ ತನಿಖೆದಾರನು ಭರಿಸಲಿದೆ. ಅಧ್ಯಯನದ ಉದ್ದಕ್ಕೂ ರೋಗಿಗೆ ಯಾವುದೇ ಹೆಚ್ಚುವರಿ ವೆಚ್ಚವಿಲ್ಲ.
- ದಯವಿಟ್ಟು ಮೇಲಿನ ಮಾಹಿತಿಯನ್ನು ಓದಿ ಮತ್ತು ನಿಮ್ಮ ಕುಟುಂಬ ಸದಸ್ಯರೊಂದಿಗೆ ಚರ್ಚಿಸಿ. ಅಧ್ಯಯನಕ್ಕೆ ಸಂಬಂಧಿಸಿದಂತೆ ನೀವು ಯಾವುದೇ ಪ್ರಶ್ನೆಯನ್ನು ಕೇಳಬಹುದು. ನೀವು ಅಧ್ಯಯನದಲ್ಲಿ ಭಾಗವಹಿಸಲು ಒಪ್ಪಿದರೆ ನಾವು ಅಗತ್ಯವಿರುವ ಮಾಹಿತಿಯನ್ನು ಸಂಗ್ರಹಿಸುತ್ತೇವೆ ಮತ್ತು ಸಂಬಂಧಿತ ಇತಿಹಾಸವನ್ನು ತೆಗೆದುಕೊಳ್ಳುತ್ತೇವೆ. ಸಂಗ್ರಹಿಸಿದ ಮಾಹಿತಿಯನ್ನು ಪ್ರಬಂಧ ಮತ್ತು ಪ್ರಕಟಣೆಗೆ ಮಾತ್ರ ಬಳಸಲಾಗುತ್ತದೆ.
- ನಿಮ್ಮಿಂದ ಸಂಗ್ರಹಿಸಲಾದ ಎಲ್ಲಾ ಮಾಹಿತಿಯನ್ನು ಗೌಪ್ಯವಾಗಿ ಇರಿಸಲಾಗುತ್ತದೆ ಮತ್ತು ಯಾವುದೇ ಹೊರಗಿನವರಿಗೆ ಬಹಿರಂಗಪಡಿಸಲಾಗುವುದಿಲ್ಲ. ನಿಮ್ಮ ಗುರುತನ್ನು ಬಹಿರಂಗಪಡಿಸಲಾಗುವುದಿಲ್ಲ. ಈ ಅಧ್ಯಯನವನ್ನು ಒಪ್ಪಿಕೊಳ್ಳಲು ಯಾವುದೇ ಒತ್ತಾಯವಿಲ್ಲ. ನೀವು ಭಾಗವಹಿಸಲು ಬಯಸದಿದ್ದರೆ ನೀವು ಪಡೆಯುವ ಕಾಳಜಿಯು ಬದಲಾಗುವುದಿಲ್ಲ.

ಈ ಅಧ್ಯಯನದಲ್ಲಿ ಭಾಗವಹಿಸಲು ನೀವು ಸ್ವಯಂಪ್ರೇರಣೆಯಿಂದ ಸಮ್ಮತಿಸಿದರೆ ಮಾತ್ರ ನೀವು ಸಹಿ/ಹೆಬ್ಬೆರಳಿನ ಗುರುತನ್ನು ಒದಗಿಸಬೇಕಾಗುತ್ತದೆ.

ಹೆಚ್ಚಿನ ಮಾಹಿತಿಗಾಗಿ ಸಂಪರ್ಕಿಸಿ:

ಡಾ. ನಾಗಶೋಬನಾ ಮನುಕರಣ್

(ಪ್ರಧಾನ ತನಿಖಾಧಿಕಾರಿ)

ಸಂಪರ್ಕ ಸಂಖ್ಯೆ: 9791859349

INFORMED CONSENT FORM

SL no: Date:

"Prediction of Hypotension Using Inferior Vena Cava Collapsibility Index to Guide Fluid Management After Spinal Anaesthesia."

I, _____ aged _____ ,have been explained in an understandable language about the purpose of the study; *to assess the degree of relationship between preoperative IVCCI and incidence of hypotension and to determine the ideal volume of crystalloid to be administered intraoperatively for optimum fluid correction based on preoperative IVCCI to prevent post-spinal hypotension*, which will be done under ultrasound guidance and spinal anaesthesia, also the risks and complications of the procedure, such *intraoperative hypotension, volume overloading, electrolyte imbalance and etc*. Hence, hereby I give my valid written informed consent without any force or prejudice to be part of the study.

The nature and risks involved have been explained to me to my understanding and satisfaction. I have been explained in detail about the study being conducted. I have read the patient information sheet and I have had the opportunity to ask any question. Any question that I have asked, have been answered to my satisfaction. I understand throughout the study if any form of payment/purchase is necessary, it will be completely borne by the investigator. I will not be charged any extra cost throughout the study.

I consent voluntarily to take part as a participant in this research. I hereby give my full valid consent to provide my history, undergo physical examination, undergo the procedure, undergo investigations and provide its results and documents etc to the doctor / institute etc. For academic and scientific purpose, the operation / procedure etc may be videographed or photographed.

All the data may be published or used for any academic purpose. I will not hold the doctors / institute etc responsible for any untoward consequences during the procedure / study.

A copy of this Informed Consent Form and Patient Information Sheet has been provided to the participant.

Patient's consent (Signature/Thumb impression): _____

Name of patient: _____

Research/Study conducting Doctor's signature: _____

Name of Doctor : Dr. Nagasobbanaa Manukaran (Principal investigator)

ಮಾಹಿತಿ ನೀಡಿದ ಒಪ್ಪಿಗೆ ನಮೂನೆ

ಎಸ್‌ಎಲ್ ಸಂಖ್ಯೆ: ದಿನಾಂಕ:

ಕೆಳಮಟ್ಟದ ವೆನಾ ಕಾವಾ ಕೊಲ್ಯಾಪ್ಸಿಬಿಲಿಟಿ ಸೂಚ್ಯಂಕವು ಹೈಪೊಟೆನ್ಷನ್ ಅನ್ನು ಊಹಿಸಬಹುದು ಮತ್ತು ಬೆನ್ನುಮೂಳೆಯ ಅರಿವಳಿಕೆ ನಂತರ ದ್ರವ ನಿರ್ವಹಣೆಗೆ ಮಾರ್ಗದರ್ಶನ ನೀಡುತ್ತದೆ

ನಾನು, _____ ವಯಸ್ಸು _____ , ಅಧ್ಯಯನದ ಉದ್ದೇಶದ ಬಗ್ಗೆ ಅರ್ಥವಾಗುವ ಭಾಷೆಯಲ್ಲಿ ವಿವರಿಸಿದ ನಂತರ; ಶಸ್ತ್ರಚಿಕಿತ್ಸೆಯ ನಂತರದ IVCCI ಮತ್ತು ಹೈಪೊಟೆನ್ಷನ್ ಸಂಭವಿಸುವಿಕೆಯ ನಡುವಿನ ಸಂಬಂಧದ ಮಟ್ಟವನ್ನು ನಿರ್ಣಯಿಸಲು ಮತ್ತು ಶಸ್ತ್ರಚಿಕಿತ್ಸೆಯ ನಂತರದ IVCCI ಯ ಆಧಾರದ ಮೇಲೆ ಗರಿಷ್ಠ ದ್ರವದ ತಿದ್ದುಪಡಿಗಾಗಿ ಕ್ರಿಸ್ಟಲಾಯ್ಡ್ನ ಆದರ್ಶ ಪರಿಮಾಣವನ್ನು ಇಂಟ್ರಾಆಪರೇಟಿವ್ ಆಗಿ ನಿರ್ಧರಿಸಲು ನಂತರದ ಬೆನ್ನುಮೂಳೆಯ ಹೈಪೊಟೆನ್ಷನ್ ಅನ್ನು ತಡೆಗಟ್ಟಲು ಮತ್ತು ಕಾರ್ಯವಿಧಾನದ ಅಪಾಯಗಳು ಮತ್ತು ತೊಡಕುಗಳು, ಇಂಟ್ರಾಆಪರೇಟಿವ್ ಹೈಪೊಟೆನ್ಷನ್, ವಾಲ್ಯೂಮ್ ಓವರ್‌ಲೋಡಿಂಗ್, ಎಲೆಕ್ಟ್ರೋಲೈಟ್ ಅಸಮತೋಲನ ಮತ್ತು ಇತ್ಯಾದಿ, ಈ ಮೂಲಕ ನನ್ನ ಮಾನ್ಯವಾದ ಲಿಖಿತ ತಿಳುವಳಿಕೆಯನ್ನು ಯಾವುದೇ ಬಲ ಅಥವಾ ಪೂರ್ವಾಗ್ರಹವಿಲ್ಲದೆ ಅಧ್ಯಯನದ ಭಾಗವಾಗಿ ನೀಡುತ್ತೇನೆ.

ಒಳಗೊಂಡಿರುವ ಸ್ವಭಾವ ಮತ್ತು ಅಪಾಯಗಳನ್ನು ನನಗೆ ತೃಪ್ತಿಪಡಿಸಲು ವಿವರಿಸಲಾಗಿದೆ. ನಡೆಸುತ್ತಿರುವ ಅಧ್ಯಯನದ ಬಗ್ಗೆ ನನಗೆ ವಿವರವಾಗಿ ವಿವರಿಸಲಾಗಿದೆ. ನಾನು ರೋಗಿಯ ಮಾಹಿತಿ ಹಾಳೆಯನ್ನು ಓದಿದ್ದೇನೆ ಮತ್ತು ಯಾವುದೇ ಪ್ರಶ್ನೆಯನ್ನು ಕೇಳಲು ನನಗೆ ಅವಕಾಶವಿದೆ. ನಾನು ಕೇಳಿದ ಯಾವುದೇ ಪ್ರಶ್ನೆಗೆ ನನ್ನ ತೃಪ್ತಿಗೆ ಉತ್ತರಿಸಲಾಗಿದೆ, ಯಾವುದೇ ರೀತಿಯ ಪಾವತಿ/ಖರೀದಿ ಅಗತ್ಯವಿದ್ದರೆ, ಅದನ್ನು ತನಿಖಾಧಿಕಾರಿಯು ಸಂಪೂರ್ಣವಾಗಿ ಭರಿಸುತ್ತಾನೆ ಎಂದು ನಾನು ಅಧ್ಯಯನದ ಉದ್ದಕ್ಕೂ ಅರ್ಥಮಾಡಿಕೊಂಡಿದ್ದೇನೆ. ಅಧ್ಯಯನದ ಉದ್ದಕ್ಕೂ ನನಗೆ ಯಾವುದೇ ಹೆಚ್ಚುವರಿ ವೆಚ್ಚವನ್ನು ವಿಧಿಸಲಾಗುವುದಿಲ್ಲ.

ಈ ಸಂಶೋಧನೆಯಲ್ಲಿ ಪಾಲ್ಗೊಳ್ಳುವವರಾಗಿ ಭಾಗವಹಿಸಲು ನಾನು ಸ್ವಯಂಪ್ರೇರಣೆಯಿಂದ ಸಮ್ಮತಿಸುತ್ತೇನೆ. ನನ್ನ ಇತಿಹಾಸವನ್ನು ಒದಗಿಸಲು, ದೈಹಿಕ ಪರೀಕ್ಷೆಗೆ ಒಳಗಾಗಲು, ಕಾರ್ಯವಿಧಾನಕ್ಕೆ ಒಳಗಾಗಲು, ತನಿಖೆಗೆ ಒಳಗಾಗಲು ಮತ್ತು ಅದರ ಫಲಿತಾಂಶಗಳು ಮತ್ತು ದಾಖಲೆಗಳನ್ನು ಇತ್ಯಾದಿಗಳನ್ನು ವೈದ್ಯರು / ಸಂಸ್ಥೆ ಇತ್ಯಾದಿಗಳಿಗೆ ಒದಗಿಸಲು ನಾನು ಈ ಮೂಲಕ ಒಪ್ಪಿಗೆ ನೀಡುತ್ತೇನೆ. ಶೈಕ್ಷಣಿಕ ಮತ್ತು ವೈಜ್ಞಾನಿಕ ಉದ್ದೇಶಕ್ಕಾಗಿ ಕಾರ್ಯಾಚರಣೆ / ಕಾರ್ಯವಿಧಾನ, ಇತ್ಯಾದಿಗಳನ್ನು ವೀಡಿಯೊಗ್ರಾಫ್ ಮಾಡಬಹುದು ಅಥವಾ ಛಾಯಾಚಿತ್ರ ಮಾಡಬಹುದು.

ಎಲ್ಲಾ ಡೇಟಾವನ್ನು ಪ್ರಕಟಿಸಬಹುದು ಅಥವಾ ಯಾವುದೇ ಶೈಕ್ಷಣಿಕ ಉದ್ದೇಶಕ್ಕಾಗಿ ಬಳಸಬಹುದು. ಕಾರ್ಯವಿಧಾನ / ಅಧ್ಯಯನದ ಸಮಯದಲ್ಲಿ ಯಾವುದೇ ಅಹಿತಕರ ಪರಿಣಾಮಗಳಿಗೆ ನಾನು ವೈದ್ಯರು / ಸಂಸ್ಥೆ ಇತ್ಯಾದಿಗಳನ್ನು ಹೊಣೆಗಾರರನ್ನಾಗಿ ಮಾಡುವುದಿಲ್ಲ.

ಈ ತಿಳುವಳಿಕೆಯುಳ್ಳ ಒಪ್ಪಿಗೆ ನಮೂನೆಯ ಪ್ರತಿಯನ್ನು ಮತ್ತು ರೋಗಿಯ ಮಾಹಿತಿ ಹಾಳೆಯನ್ನು ಭಾಗವಹಿಸುವವರಿಗೆ ಒದಗಿಸಲಾಗಿದೆ.

ರೋಗಿಯ ಒಪ್ಪಿಗೆ (ಸಹಿ/ಹೆಬ್ಬೆರಳಿನ ಗುರುತು): _____

ರೋಗಿಯ ಹೆಸರು: _____

ಸಂಶೋಧನೆ/ಅಧ್ಯಯನ ನಡೆಸುವ ವೈದ್ಯರ ಸಹಿ: _____

ವೈದ್ಯರ ಹೆಸರು: **ನಾಗಶೋಬನಾ ಮನುಕರಣ್**

KEY TO MASTERCHART

Num	Serial number
IP number	In-patient number
ASA grade	American Society of Anaesthesiologists' classification of Physical Health
BMI	Body mass index
SBP	Systolic blood pressure
DBP	Diastolic blood pressure
MAP	Mean arterial pressure
SpO_2	Saturation of peripheral oxygen
IVC (ins)	Inferior vena cava measurement during inspiration in cm
IVC (exp)	Inferior vena cava measurement during expiration in cm
IVCCI	Inferior vena cava collapsibility index

MASTER CHART

Num.	IP number	Age (years)	Gender	ASA Grade	Height (cm)	Weight (kg)	BMI	Heart Rate	Systolic blood pressure	Diastolic blood Pressure	MAP	SpO2	IVC (ins)	IVC (exp)	IVCCI	Systolic blood pressure	Diastolic blood Pressure	MAP	Based on MAP	SBP fall	Based on SBP	Overall Hypotension
1	55918	24	Female	1	158	65	26.03749399	98	112	58	76	99	0.7	1.9	63.15789474	80	50	60	No	0.32	Hypotension	Hypotension
2	131646	53	Female	2	155	59	24.55775234	72	130	80	96.66666667	98	1.5	1.9	21.05263158	118	67	84	No	0.12	No	No
3	149775	35	Male	2	176	68	21.95247934	84	130	90	103.3333333	100	1.3	1.7	23.52941176	116	83	94	No	0.14	No	No
4	151576	60	Male	2	169	78	27.30996814	89	122	64	83.33333333	98	0.9	1.8	50	90	58	68.66666667	No	0.32	Hypotension	Hypotension
5	169471	45	Female	2	164	60	22.30814991	88	122	80	94	100	2	2.3	13.04347826	111	65	80.33333333	No	0.11	No	No
6	170469	50	Male	2	162	58	22.10028959	84	110	72	84.66666667	99	0.6	2.1	71.42857143	80	54	62.66666667	No	0.3	Hypotension	Hypotension
7	171860	23	Male	1	160	78	30.46875	120	142	68	92.66666667	98	0.8	1.9	57.89473684	101	60	73.66666667	No	0.41	Hypotension	Hypotension
8	175879	57	Male	2	160	68	26.5625	96	90	60	70	97	0.9	2.1	57.14285714	60	32	41.33333333	Hypotension	0.3	Hypotension	Hypotension
9	176877	57	Male	2	160	66	25.78125	96	90	60	70	96	2	2.3	13.04347826	80	55	63.33333333	No	0.1	No	No
10	176887	57	Male	1	160	82	32.03125	96	160	78	105.3333333	100	0.5	1	50	130	63	85.33333333	No	0.3	Hypotension	Hypotension
11	183265	53	Male	2	172	85	28.73174689	88	140	90	106.6666667	100	0.9	1	10	113	88	96.33333333	No	0.27	No	No
12	185742	55	Male	2	157	56	22.7189744	90	100	62	74.66666667	100	2.1	2.3	8.695652174	78	45	56	Hypotension	0.22	No	Hypotension
13	188652	32	Female	1	157	70	28.398718	92	130	90	103.3333333	100	1.3	1.4	7.142857143	113	83	93	No	0.17	No	No
14	189012	49	Male	1	168	75	26.57312925	92	130	90	103.3333333	99	0.9	1.5	40	98	53	68	No	0.32	Hypotension	Hypotension
15	189183	55	Female	2	154	62	26.14268848	84	130	80	96.66666667	99	0.9	1.1	18.18181818	111	65	80.33333333	No	0.19	No	No
16	190403	36	Male	1	168	80	28.3446712	110	120	68	85.33333333	99	2.8	3	6.666666667	103	64	77	No	0.17	No	No
17	191469	59	Male	2	168	86	30.47052154	84	120	90	100	99	1.2	1.3	7.692307692	116	84	94.66666667	No	0.04	No	No
18	193949	30	Female	1	152	64	27.70083102	84	120	70	86.66666667	99	1.2	2.1	42.85714286	90	66	74	No	0.3	Hypotension	Hypotension
19	194132	56	Female	2	154	48	20.23950076	74	110	70	83.33333333	99	0.9	1.7	47.05882353	65	46	52.33333333	Hypotension	0.45	Hypotension	Hypotension
20	194304	26	Male	1	157	57	23.12467037	86	110	70	83.33333333	100	2.1	2.6	19.23076923	99	58	71.66666667	No	0.11	No	No
21	194312	28	Male	1	167	63	22.58955144	70	122	70	87.33333333	98	2.3	2.5	8	100	66	77.33333333	No	0.22	No	No
22	194364	32	Male	1	161	55	21.21831719	102	120	80	93.33333333	100	0.8	1.8	55.55555556	88	60	69.33333333	No	0.32	Hypotension	Hypotension
23	194378	52	Female	2	159	55	21.75546853	70	110	80	90	100	2	2.4	16.66666667	100	66	77.33333333	No	0.1	No	No
24	194379	52	Female	1	155	55	22.89281998	70	110	68	82	99	2.6	2.9	10.34482759	96	60	72	No	0.14	No	No
25	194860	50	Male	2	162	60	22.86236854	90	120	70	86.66666667	98	1.3	2.5	48	80	62	68	No	0.4	Hypotension	Hypotension
26	195097	76	Female	2	160	64	25	70	150	90	110	100	0.8	1.7	52.94117647	110	78	88.66666667	No	0.4	Hypotension	Hypotension
27	195740	35	Female	2	158	72	28.84153181	78	120	70	86.66666667	98	1.2	2.3	47.82608696	88	56	66.66666667	No	0.32	Hypotension	Hypotension
28	195935	26	Male	1	165	71	26.07897153	84	130	90	103.3333333	99	1	1.3	23.07692308	119	72	87.66666667	No	0.11	No	No
29	196142	75	Female	2	162	52	19.81405274	94	140	80	100	99	1.1	1.4	21.42857143	122	68	86	No	0.18	No	No
30	196434	41	Female	2	168	88	31.17913832	88	134	76	95.33333333	99	0.7	1.3	46.15384615	95	65	75	No	0.39	Hypotension	Hypotension
31	196440	62	Male	2	176	80	25.82644628	98	130	92	104.6666667	99	1.1	2.1	47.61904762	100	64	76	No	0.3	Hypotension	Hypotension
32	196476	75	Male	2	165	58	21.30394858	68	110	80	90	98	0.8	1.3	38.46153846	50	40	43.33333333	Hypotension	0.6	Hypotension	Hypotension
33	196680	45	Male	2	167	76	27.25088745	98	100	70	80	100	1.8	2.3	21.73913043	88	62	70.66666667	No	0.12	No	No
34	196962	22	Female	1	160	65	25.390625	80	110	78	88.66666667	98	2	2.1	4.761904762	96	72	80	No	0.14	No	No
35	197364	58	Female	2	156	54	22.18934911	82	122	90	100.6666667	100	2.5	2.8	10.71428571	116	76	89.33333333	No	0.06	No	No
36	198866	46	Male	1	165	60	22.03856749	90	140	90	106.6666667	100	0.6	1.3	53.84615385	102	76	84.66666667	No	0.38	Hypotension	Hypotension
37	199092	32	Female	1	156	75	30.81854043	78	120	76	90.66666667	100	1.9	2.4	20.83333333	111	65	80.33333333	No	0.09	No	No
38	199441	39	Female	1	160	95	37.109375	80	120	76	90.66666667	100	1.6	2.1	23.80952381	110	70	83.33333333	No	0.1	No	No

39	199482	39	Female	1	158	59	23.63403301	80	132	78	96	99	1.4	1.7	17.64705882	121	64	83	No	0.11	No	No
40	200039	40	Male	1	164	63	23.42355741	88	110	88	95.33333333	100	0.3	1.4	78.57142857	78	56	63.33333333	No	0.32	Hypotension	Hypotension
41	200305	21	Male	1	164	52	19.33372992	90	110	64	79.33333333	100	3	3.2	6.25	94	58	70	No	0.16	No	No
42	200506	20	Male	1	160	52	20.3125	114	100	60	73.33333333	98	1.5	1.8	16.66666667	91	55	67	No	0.09	No	No
43	200841	60	Male	1	170	77	26.64359862	105	100	92	94.66666667	97	1	3.2	68.75	60	43	48.66666667	Hypotension	0.4	Hypotension	Hypotension
44	200907	45	Male	2	170	65	22.49134948	64	140	90	106.6666667	98	1.2	1.5	20	122	83	96	No	0.18	No	No
45	201261	58	Male	2	168	64	22.67573696	76	144	92	109.3333333	98	0.9	1.5	40	106	65	78.66666667	No	0.38	Hypotension	Hypotension
46	201976	42	Female	2	160	69	26.953125	84	110	80	90	100	0.5	1.1	54.54545455	78	56	63.33333333	No	0.32	Hypotension	Hypotension
47	202356	50	Female	2	165	62	22.77318641	68	110	60	76.66666667	98	1.6	1.8	11.11111111	78	54	62	No	0.32	Hypotension	Hypotension
48	202412	37	Female	2	162	48	18.28989483	80	130	80	96.66666667	99	0.7	1.8	61.11111111	110	60	76.66666667	No	0.2	No	No
49	202546	48	Female	2	165	68	24.97704316	66	120	70	86.66666667	100	1.3	2.2	40.90909091	90	62	71.33333333	No	0.3	Hypotension	Hypotension
50	203119	55	Female	2	156	52	21.36752137	75	120	90	100	99	1.5	1.8	16.66666667	113	78	89.66666667	No	0.07	No	No
51	203312	50	Female	2	153	62	26.48553975	80	130	90	103.3333333	99	0.9	1.6	43.75	100	71	80.66666667	No	0.3	Hypotension	Hypotension
52	203430	51	Male	1	173	62	20.71569381	110	110	90	96.66666667	98	0.2	1	80	72	55	60.66666667	No	0.38	Hypotension	Hypotension
53	203434	50	Male	2	168	60	21.2585034	62	140	76	97.33333333	98	1.5	2	25	128	74	92	No	0.12	No	No
54	206020	58	Female	2	158	58	23.23345618	84	100	60	73.33333333	100	1.9	2.1	9.523809524	89	58	68.33333333	No	0.11	No	No
55	206460	56	Female	2	163	65	24.4646016	78	100	78	85.33333333	99	1.8	2	10	88	67	74	No	0.12	No	No
56	208198	44	Female	1	155	48	19.97918835	75	130	70	90	100	0.5	1	50	100	62	74.66666667	No	0.3	Hypotension	Hypotension
57	208320	44	Female	2	157	64	25.96454217	75	130	70	90	99	1.8	2.3	21.73913043	117	65	82.33333333	No	0.13	No	No
58	208737	68	Female	2	155	67	27.88761707	76	130	76	94	98	0.9	2.1	57.14285714	99	67	77.66666667	No	0.31	Hypotension	Hypotension
59	209155	38	Male	1	164	58	21.56454491	60	120	70	86.66666667	100	1	2.1	52.38095238	90	59	69.33333333	No	0.3	Hypotension	Hypotension
60	209656	48	Female	2	167	68	24.38237298	78	146	78	100.6666667	99	0.9	1.8	50	106	65	78.66666667	No	0.4	Hypotension	Hypotension
61	211234	59	Male	1	161	63	24.30461788	65	110	70	83.33333333	99	1.2	1.5	20	100	66	77.33333333	No	0.1	No	No
62	211519	35	Male	1	176	85	27.44059917	93	110	80	90	98	1.6	2	20	105	64	77.66666667	No	0.05	No	No
63	211897	39	Female	1	161	80	30.86300683	69	112	70	84	99	2.9	3.1	6.451612903	99	68	78.33333333	No	0.13	No	No
64	212279	30	Male	1	162	64	24.38652644	65	120	70	86.66666667	98	2.8	3.2	12.5	103	58	73	No	0.17	No	No
65	212475	58	Male	1	165	72	26.44628099	86	110	76	87.33333333	98	1	2.3	56.52173913	78	60	66	No	0.32	Hypotension	Hypotension
66	212934	43	Female	2	156	74	30.40762656	120	140	68	92	99	1.5	1.7	11.76470588	118	60	79.33333333	No	0.22	No	No
67	213176	22	Female	1	163	60	22.58270917	80	90	60	70	100	2.6	3.2	18.75	68	55	59.33333333	Hypotension	0.22	No	Hypotension
68	213607	37	Female	1	155	80	33.29864724	96	120	72	88	99	1.2	2.2	45.45454545	89	65	73	No	0.31	Hypotension	Hypotension
69	213930	36	Male	2	170	58	20.06920415	72	110	60	76.66666667	99	2.7	3	10	100	53	68.66666667	No	0.1	No	No
70	215660	68	Male	2	170	55	19.03114187	70	140	80	100	99	1	1.9	47.36842105	110	77	88	No	0.3	Hypotension	Hypotension
71	216015	55	Male	2	165	89	32.69054178	83	110	64	79.33333333	96	1.2	2.5	52	80	67	71.33333333	No	0.3	Hypotension	Hypotension
72	216767	25	Male	1	163	55	20.70081674	64	120	70	86.66666667	100	2	3.3	39.39393939	89	60	69.66666667	No	0.31	Hypotension	Hypotension
73	216839	52	Male	2	163	60	22.58270917	98	144	60	88	98	1.7	2	15	123	54	77	No	0.21	No	No
74	217315	33	Female	1	154	70	29.51593861	94	110	70	83.33333333	100	0.8	1.6	50	92	66	74.66666667	No	0.18	No	No
75	219076	38	Male	2	172	95	32.11195241	80	130	100	110	99	2.1	2.3	8.695652174	112	79	90	No	0.18	No	No
76	219105	46	Male	2	166	65	23.58832922	80	150	80	103.3333333	99	0.3	1.5	80	90	65	73.33333333	No	0.6	Hypotension	Hypotension
77	219537	22	Female	1	162	55	20.95717116	86	100	60	73.33333333	97	1	3.2	68.75	60	48	52	Hypotension	0.4	Hypotension	Hypotension
78	219848	27	Female	1	165	70	25.71166208	96	120	80	93.33333333	100	1.2	1.5	20	97	68	77.66666667	No	0.23	No	No

79	219945	30	Male	1	174	70	23.1206236	88	120	90	100	100	1.2	2	40	90	60	70	No	0.3	Hypotension	Hypotension
80	220028	48	Male	2	165	80	29.38475666	90	110	90	96.66666667	98	0.8	1	20	88	72	77.33333333	No	0.22	No	No
81	220191	44	Male	2	163	64	24.08822312	102	130	70	90	99	1.9	2.3	17.39130435	119	59	79	No	0.11	No	No
82	220531	56	Male	1	162	71	27.05380277	79	130	90	103.3333333	98	0.6	1.3	53.84615385	100	77	84.66666667	No	0.3	Hypotension	Hypotension
83	220639	58	Male	2	167	82	29.4022733	80	120	70	86.66666667	100	2	2.5	20	114	63	80	No	0.06	No	No
84	220878	40	Female	2	155	63	26.2226847	92	130	80	96.66666667	100	1.6	3.1	48.38709677	95	59	71	No	0.35	Hypotension	Hypotension
85	221083	19	Male	1	164	48	17.84651993	77	110	80	90	99	2.2	2.5	12	100	71	80.66666667	No	0.1	No	No
86	221563	41	Male	2	165	75	27.54820937	100	130	60	83.33333333	100	1.5	2.3	34.7826087	98	56	70	No	0.32	Hypotension	Hypotension
87	221596	52	Male	2	167	64	22.94811574	90	140	90	106.6666667	100	0.5	1.4	64.28571429	92	78	82.66666667	No	0.48	Hypotension	Hypotension
88	221755	45	Male	2	175.2	70	22.80498739	70	120	70	86.66666667	99	1.3	2.3	43.47826087	92	42	58.66666667	Hypotension	0.28	No	Hypotension
89	221946	29	Female	1	152	75	32.46191136	80	120	80	93.33333333	99	1	1.2	16.66666667	98	65	76	No	0.22	No	No
90	222056	29	Female	1	155	75	31.21748179	80	120	80	93.33333333	100	0.9	1.7	47.05882353	88	65	72.66666667	No	0.32	Hypotension	Hypotension
91	222063	31	Male	2	165	75	27.54820937	100	130	60	83.33333333	100	2.1	2.4	12.5	122	64	83.33333333	No	0.08	No	No
92	222178	40	Female	1	159.7	54	21.17307459	82	130	80	96.66666667	98	1	1.8	44.44444444	99	67	77.66666667	No	0.31	Hypotension	Hypotension
93	222278	45	Female	1	159	68	26.89767019	82	130	80	96.66666667	100	1.2	1.4	14.28571429	118	65	82.66666667	No	0.12	No	No
94	222279	32	Male	1	166	64	23.22543185	86	122	70	87.33333333	99	2.3	2.4	4.166666667	110	65	80	No	0.12	No	No
95	222346	38	Female	1	164	54	20.07733492	80	110	60	76.66666667	98	1.6	2.7	40.74074074	78	45	56	Hypotension	0.32	Hypotension	Hypotension
96	222486	50	Male	2	167	76	27.25088745	90	140	90	106.6666667	100	0.8	1.3	38.46153846	110	76	87.33333333	No	0.3	Hypotension	Hypotension
97	222515	35	Male	2	176	70	22.5981405	90	120	70	86.66666667	99	2	2.3	13.04347826	98	63	74.66666667	No	0.22	No	No
98	223298	52	Male	1	159	68	26.89767019	99	154	78	103.3333333	99	0.8	1.5	46.66666667	124	62	82.66666667	No	0.3	Hypotension	Hypotension
99	224731	27	Female	1	156	57	23.42209073	82	118	90	99.33333333	98	2.9	3.1	6.451612903	105	76	85.66666667	No	0.13	No	No
100	224808	60	Female	2	160	58	22.65625	72	130	80	96.66666667	95	1.3	1.5	13.33333333	115	63	80.33333333	No	0.15	No	No
101	225053	43	Male	1	166	78	28.30599506	64	110	70	83.33333333	99	1.5	1.6	6.25	100	64	76	No	0.1	No	No
102	226992	25	Male	1	174	79	26.0932752	104	145	88	107	100	0.6	1.2	50	86	70	75.33333333	No	0.59	Hypotension	Hypotension