"TO COMPARE THE EFFECTIVENESS OF SEQUENTIAL COMPRESSION DEVICE VS CRYSTALLOID PRELOAD IN REDUCING POST SPINAL HYPOTENSION IN CAESAREAN SECTION"

By

Dr. REVATHI ASHOK

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

In partial fulfillment of the requirements for the degree of

DOCTOR OF MEDICINE

IN ANAESTHESIOLOGY

Under the Guidance of

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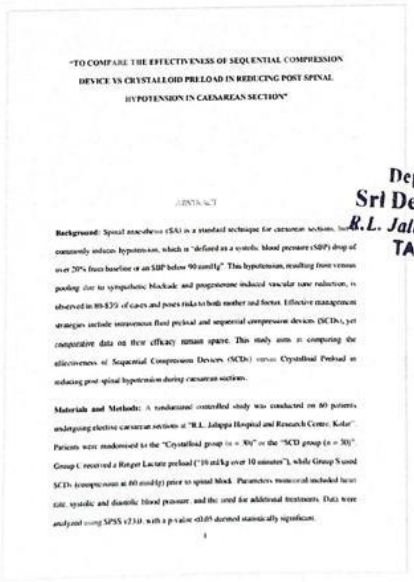

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ABBREVIATIONS

BJR	Bezold-Jarisch Reflex
CNS	Central Nervous System
CO2	Carbon Dioxide
CSF	Cerebrospinal Fluid
IV	Intravenous
ICP	Intracranial Pressure
L1	First Lumbar Vertebral Body
L2	Second Lumbar Vertebral Body
L3	Third Lumbar Vertebral Body
LDH	Lactate Dehydrogenase
mm/Hg	Millimeters of Mercury
NS	Normal Saline
PDPH	Post-Dural Puncture Headache
SA	Spinal Anesthesia
SAIH	Spinal Anesthesia-Induced Hypotension
SBP	Systolic Blood Pressure

SCD	Sequential Compression Devices
T1	First Thoracic Vertebral Body
T3	Third Thoracic Vertebral Body
T6	Sixth Thoracic Vertebral Body
T12	Twelfth Thoracic Vertebral Body
S2	Second Sacral Vertebral Body
S3	Third Sacral Vertebral Body

ABSTRACT

Background: Spinal anaesthesia (SA) is a standard technique for caesarean sections, but it commonly induces hypotension, which is defined as a systolic blood pressure (SBP) drop of over 20% from baseline or an SBP below 90 mmHg. This hypotension, resulting from venous pooling due to sympathetic blockade and progesterone-induced vascular tone reduction, is observed in 80-83% of cases and poses risks to both mother and foetus. Effective management strategies include intravenous fluid preload and sequential compression devices (SCDs), yet comparative data on their efficacy remain sparse. This study aims at comparing the effectiveness of Sequential Compression Devices (SCDs) versus Crystalloid Preload in reducing post-spinal hypotension during caesarean sections.

Materials and Methods: A randomized controlled study was conducted on 60 patients undergoing elective caesarean sections at R.L. Jalappa Hospital and Research Centre, Kolar". Patients were randomised to the Crystalloid group (n = 30) or the SCD group (n = 30). Group C received a Ringer Lactate preload (10 ml/kg over 10 minutes), while Group S used SCDs (compression at 60 mmHg) prior to spinal block. Parameters monitored included heart rate, systolic and diastolic blood pressure, and the need for additional treatments. Data were analyzed using SPSS v23.0, with a p-value <0.05 deemed statistically significant.

Results: The mean age of participants was 26.8 ± 3.5 years, with nil significant age differences between the groups. Heart rate changes also showed no differences between the two groups. Systolic blood pressure (SBP) dropped significantly from the 6th to 12th minute in the Crystalloid group compared to the SCD group ($p < 0.05$). Diastolic blood pressure (DBP) was significantly lower in the SCD group at the 55th and 60th minutes post-intervention ($p < 0.05$).

The Crystalloid group required significantly more additional treatments at the 6th minute ($p < 0.05$).

Conclusion: Sequential Compression Devices (SCDs) are more effective than Crystalloid Preload in mitigating early post-spinal hypotension during caesarean sections. SCDs provide superior stability of systolic blood pressure and reduce the need for additional treatments shortly after anaesthesia administration, making them a preferable option in managing post-spinal hypotension in caesarean sections.

Keywords: Spinal anesthesia, Caesarean section, Hypotension, Sequential Compression Device (SCD), Crystalloid Preload, Post-spinal hypotension.

TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

INTRODUCTION

INTRODUCTION

Patients undergoing lower abdominal surgeries including caesarean section require spinal anaesthesia to perform the surgical procedure.

Spinal anaesthesia (SA) is a frequently employed method for caesarean section. Hypotension is defined by a systolic pressure drop of more than 20% of the baseline, or a systolic blood pressure (SBP) less than 90 mm/Hg. There is an 80-83% incidence of spinal anaesthesia induced hypotension. It is because of venous pooling due to sympathetic blockade and also due to the vascular tone being reduced by progesterone, and poses a risk to the mother, foetus, and infant. The degree of hypotension depends upon various factors like height of the block, position of the patient, volume and concentration of the drug and type of surgery. Post spinal hypotension can be managed effectively by relieving aortocaval compression, loading the patient with intravenous fluids, sequential compression devices (SCD), vasopressors, ionotropic agents.[1,2]

Early detection can reduce adverse events such as reduced uterine blood flow and foetal acidosis.[1,3] Hypotension is one of the most frequent side effect of spinal anaesthesia. It is due to loss of vascular tone due to sympatholysis which leads to reduced venous return to the heart. This is more pronounced in the obstetric patients due to the presence of the gravid uterus and effects of progesterone.

There are many studies found in literature with regard to effectiveness of crystalloid preloading in reducing post spinal hypotension. But there are not many studies done to prove the effectiveness of SCD in reducing post spinal hypotension. Therefore, we aim to evaluate the effectiveness of crystalloid preload and SCD in lowering hypotension following spinal anaesthesia in elective caesarean sections.

Preloading with crystalloid solution or the use compression devices can be done in order to reduce the hypotension. We intend to study the effectiveness of both crystalloid preloading and sequential compression device in reducing spinal induced hypotension in caesarean section.

AIMS & OBJECTIVES

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- To determine the effectiveness of sequential compression devices in reducing incidence of hypotension after spinal anesthesia in elective caesarean section.

- To compare and assess the role of crystalloid preload in reducing incidence of hypotension after spinal anesthesia with sequential compression devices.

REVIEW OF LITERATURE

REVIEW OF LITERATURE

Spinal anaesthesia

The first step towards the development of local anaesthesia was the isolation of local anaesthetics, with cocaine being the only one that occurs naturally. The very first regional anaesthetic method employed was spinal anaesthesia, and August Bier conducted the first operation using spinal anaesthetic in 1898 in Germany. Prior to this, the only local anaesthetic treatments available were topical eye anaesthetic and infiltration anaesthesia.[4]

The central nervous system (CNS) consists the brain and spinal cord. When local anaesthetic is administered in or around the CNS, it is termed neuraxial anaesthesia. Spinal anaesthesia falls under the category of neuraxial anaesthesia, involving the direct injection of a local anaesthetic into intrathecal region, also known as the subarachnoid space. This space contains sterile cerebrospinal fluid (CSF), which surrounds and protects the brain and spinal cord, An adult typically harbors approximately 130 to 140 mL of CSF, which circulates throughout the day. Approximately 500 mL of CSF is generated daily.[4]

Epidural and caudal anaesthesia are other neuraxial methods, each with its own set of indications.

Anatomy and physiology[5]

A good grasp of neuraxial anatomy and appropriate placement are essential for administering spinal anaesthesia. The goal is to administer the anaesthetic at the ideal dosage into the intrathecal (subarachnoid) region.

The spinal column consists of seven cervical vertebrae, twelve thoracic vertebrae, five lumbar vertebrae, and five fused sacral vertebrae. These vertebrae are named based on their relative

positions and anatomical characteristics. They are arranged in a stacked manner, featuring articulating joints, ligaments, and a central hollow space known as the spinal canal, which houses the spinal cord. The pedicles of neighbouring vertebrae have lateral openings via which the spinal nerves leave the spinal canal.

As previously mentioned, spinal anaesthesia is typically administered in the lumbar region, ideally below second lumbar vertebrae to minimize the risk of spinal cord injury. The conus medullaris, which is usually located close to the lower margin of the first or second lumbar vertebral body, indicates the caudal end of the spinal cord.

In pediatric patients, it is often found lower, around the level of L3. In adults, the average location of the conus is at the lower part of L1 (with a range extending from middle third of T12 to the upper third of L3). Conus position variations follow a normal distribution and does not show significant differences between gender or with increasing age.[6]

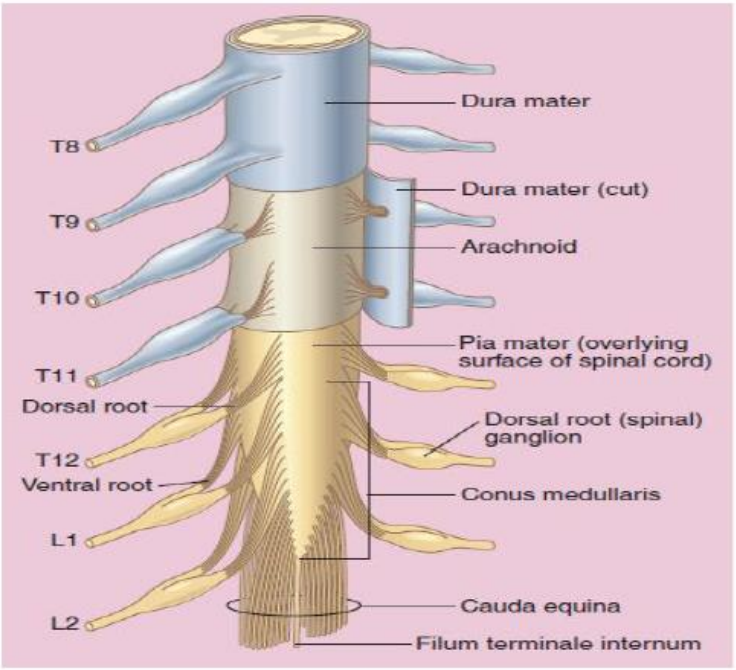

Figure 1: Covering of spinal cord

Typically, the dural sac extends to S2/3. These factors make the L3/4 or L4/5 interspace a common choice for the spinal needle insertion. When adopting higher interspaces, spinal cord injuries is more likely, especially in obese people.[7]

Indications

Indications

Epidural anaesthesia is frequently used in conjunction with general anaesthesia or as the only anaesthetic for thoracic surgery. As mentioned earlier, surgeries involving the lower limbs, pelvis, lower abdomen, and perineum—especially those done below the umbilicus, spinal anaesthesia is commonly employed.

Patients should be explained about the procedure, advantages and associated complications and obtain informed consent before the procedure. Since surgeries are often performed on awake or lightly sedated patients, discussions regarding the rationale for spinal anesthesia, potential risks, benefits, and alternative treatments can help alleviate anxiety. Spinal anaesthesia is most suitable for brief procedures, while general anaesthesia is typically preferred for longer surgeries or those that could compromise respiration.[8]

Contraindications[5]

There are various well-documented contraindications to neuraxial anaesthesia.

Absolute contraindications include

- patient's refusal,
- a cerebral mass that frequently causes an increase in intracranial pressure (ICP), and
- an infection at the surgical site that could result in meningitis
- thrombocytopenia, coagulopathy

There are several relative contraindications:[9,10]

- Pre-existing neurological conditions (particularly those that wax and wane, e.g., multiple sclerosis)
- Risk factors for hypotension, which causes severe dehydration (hypovolemia), include ageing beyond 40 to 50 years, emergency surgery, obesity, long-term alcohol usage, and chronic hypertension.

- Severe mitral and aortic stenosis, along with left ventricular outflow obstruction seen in hypertrophic obstructive cardiomyopathy, represent relative contraindications.

The American Society of Regional Anaesthesia (ASRA) offers updated guidelines regarding the timing of neuraxial anaesthesia for patients on oral anticoagulants, antiplatelets, thrombolytic therapy, unfractionated heparin, or low molecular weight heparin.[11,12]

Preparation of patients[5]

Before initiating neuraxial anaesthesia, it is essential to obtain a comprehensive history and perform physical examination. Understanding the patient's prior exposure to anaesthesia, reviewing allergies, and noting any family history of anaesthesia-related issues are all crucial aspects of the medical history.[13]

The physical assessment focuses on identifying the appropriate site for spinal anaesthesia placement. A thorough examination of the back is conducted, including a pre-procedural neurological assessment to evaluate strength and sensation. Additionally, it is important to evaluate and record the existence of any spinal abnormalities, including spina bifida, spinal stenosis, scoliosis, prior back surgery, and a history of tethered cord.

Drugs used

- 0.5 percent levobupivacaine heavy
- 0.75 percent ropivacaine heavy

Complications

Appropriate patient selection and treatment should be developed to assist and avoid typical neuraxial anaesthetic problems. While many of the consequences are quite rare, it is important to be aware of them. Severe problems are thought to be exceedingly rare, although their occurrence is likely underestimated. [14–16]

Some examples include;

- Backache (more common with epidural anaesthesia)
- Post dural-puncture headache (PDPH; as high as 25 percent in some studies) A non-cutting needle should be used for patients who are at high risk of PDPH, and it is recommended to utilize the smallest gauge needle available.[17]
- Vomiting and nausea
- Hypotension
- High and total spinal anaesthesia
- Hematoma of the spine
- Neurological damage
- Arachnoiditis[18]
- Transient neurological syndromes (especially with lidocaine)

Spinal anaesthesia induced hypotension[19]

Maintaining adequate arterial blood pressure is crucial for ensuring appropriate organ

perfusion. Although spinal anaesthesia (SA) offers numerous advantages over general anaesthesia, it often leads to arterial hypotension.

The administration of local anaesthetic drug into the subarachnoid space induces significant and comprehensive physiological effects, with the cardiovascular system experiencing the most pronounced changes. Autonomic denervation combined with more widespread neural blocking and vagal innervation involvement causes these results. However, the cardiovascular effects of spinal anaesthesia cannot be fully attributed to the presence of local anaesthetics because their concentrations in the cerebrospinal fluid (CSF) in the ventricles are not high enough to directly depress medullary vasomotor centers.

Even in cases of cervical-level anaesthesia, the concentrations of local anaesthetics in the cisternal CSF remain below the threshold necessary to affect medullary vasomotor centers when administered directly to the brainstem. Anaesthetic plasma levels during spinal anaesthesia are also inadequate to directly affect peripheral vascular smooth muscles or the heart.

Since sympathetic denervation plays a significant role in the cardiovascular changes during spinal anaesthesia, it is essential to take into account spinal anesthesia's effects on the sympathetic nervous system prior to talking about how it affects cardiovascular reactions.

Effect on sympathetic system[19]

The degree of cardiovascular responses to spinal anaesthesia is dependent on the degree of sympathetic denervation, indicating that more severe neural blockade results in more pronounced alterations to hemodynamic parameters.

In cases of partial sympathetic blockade, sympathetic activity increases reflexively in regions where sympathetic function remains intact. This results in vasoconstriction, which aims to counterbalance the peripheral vasodilation observed in sympathetically denervated areas.

Changes in the upper extremity cutaneous blood flow and arterial pressure waveforms are evident at low or mid-thoracic sensory levels of spinal anaesthesia. Furthermore, keep in mind that the majority of cephalad preganglionic sympathetic fibers exit the spinal cord at the T1 level.

Therefore, circulatory changes with mid-cervical sensory levels of anaesthesia are not more apparent than those at the T1 level since sympathetic denervation is complete at the" T1 level.

The arterial hypotension mechanism

The degree of the ensuing sympathetic block can differ greatly amongst people, while the cardiovascular effects of SA are directly associated with the degree of the sympathetic block.

Effect on resistive and capacitive vascular system[19]

Spinal anaesthesia (SA)-induced sympathetic blockade causes accelerated vasodilation in the arteries and arteriolar regions that are affected, which raises sympathetic arterial vascular tone in areas that are not affected by the blockade through a baroreflex mechanism. In younger patients, these compensating mechanisms tend to work efficiently.[20]

SA's hemodynamic implications include a sympathetic block of the venous reservoir, which causes blood to pool in the lowermost capacitance veins. Vasopressors can mobilise up to 20% of the circulating blood volume when the level of sensory block is greater than or equal to T6, causing pooling in the hepatosplanchnic region.[20,21]

Effect on cardiovascular system

Because spinal anaesthesia (SA) creates an imbalance between sympathetic and parasympathetic tones in favor of parasympathetic activity, it causes bradycardia and a drop in blood pressure. Both a disruption of cardiovascular function and an adaptive reaction intended to extend diastole to assist ventricular filling may be indicated by this bradycardia and hypotension.

It is well-documented that arterial blood pressure is regulated by the cardioinhibitory receptors of the Bezold-Jarisch reflex (BJR) in conjunction with aortic and carotid baroreceptors. In cases of mild hypovolemia, BJR activity diminishes while the baroreflex is stimulated, resulting in an elevation of arterial blood pressure. On the other hand, an acute decrease in venous return during profound hypovolemia—such as in situations of considerable blood loss—causes a paradoxical activation of the BJR, which results in prolonged hypotension and bradycardia. This bradycardia may serve as an adaptive mechanism to ensure adequate diastolic filling time.[22]

Bradycardia occurs in about 13% of non-obstetrical patients during SA; if corrective action is taken right away, there are no major consequences. But since severe bradycardia brought on by SA can swiftly proceed to asystole, it should always be regarded as a warning indication of an imminent hemodynamic collapse.[9,22,23]

Arterial hypotension[19]

The onset of arterial hypotension is a result of the effects of SA on the cardiovascular system. Patient mortality and morbidity have been associated to intraoperative arterial hypotension.[24,25] The most widely used definition for spinal anaesthesia induced hypotension(SAIH) in the literature is a systolic arterial blood pressure less than 80% of the

baseline. The prevalence of SAIH rises with age, with 36% of younger patients affected and 75% of individuals over the age of 50 affected.[26] Even at modest dosages (7.5 mg bupivacaine), the prevalence of SAIH in elderly people remained significant.[27] Moreover, disruption of homeostasis mechanisms might exacerbate SAIH; this is particularly the case in patients with irregular neuro-humoral regulation of heart rate, rapid block extension, or concomitant sedation (increased dysautonomia).

Cardiac output

Preload plays a crucial role in determining cardiac output. In individuals with normal blood volume levels, cardiac output remains unchanged even with high doses of spinal anaesthesia that induce complete sympathetic denervation, provided that the legs are positioned above the level of the heart.

Effect on heart rate

In the absence of medications affecting autonomic activity, spinal anaesthesia typically leads to a decrease in heart rate. One of the contributing factors to the bradycardia is the inhibition of preganglionic cardiac accelerator fibers, which originate from T1 to T4 during deep spinal anesthesia. A significant decrease in right atrial pressure as well as the pressure in the major arteries as they enter the right atrium both have an impact on the bradycardic response.

A patient's heart rate is raised when they are positioned slightly head-down because this promotes venous return.

The right atrium and surrounding major arteries include intrinsic stretch receptors, often referred to as chronotropic stretch receptors, which are involved in the direct correlation between heart rate and right atrial pressure during high spinal anaesthesia.

The average reduction in heart rate due to complete sympathetic denervation is typically moderate, ranging from 10% to 15%. These cardiovascular responses are mediated by the Bezold-Jarisch reflex mechanism.

Effect on cerebral blood flow

In humans, cerebral blood flow remains stable despite significant fluctuations in mean arterial pressure due to the cerebrovascular autoregulatory mechanisms. These systems ensure the maintenance of cerebral blood flow within a narrow range. Unlike other vascular beds, the cerebral circulation is minimally influenced by the sympathetic nervous system.

Impact on local blood flow

Spinal anaesthesia induces minor reduction of approximately 10% in hepatic blood flow, but it does not lead to any significant alterations in other hemodynamic variables or arterial and venous oxygen tensions.

Hypotension management during SA

It is now known that, even in the presence of mild hypotension, normal people receiving spinal anaesthesia maintain appropriate oxygenation in vital organs including the heart and brain. As a result, maintaining normal blood pressure during spinal anaesthesia is no longer regarded as desirable or required. On the other hand, severe hypotension can happen when decreases in myocardial demands and cerebrovascular resistance are not sufficient to make up for reductions in cerebral and coronary perfusion pressures. Vasopressors are no longer commonly utilized to manage hypotension during spinal anaesthesia. Methoxamine and phenylephrine are examples of alpha receptor agonists that can cause afterload to increase to the point where an increase in workload causes a greater demand for oxygen in the left ventricle than there is for oxygen in the myocardium due to increased coronary perfusion

pressure.

Moreover, reduced preload and cardiac output during spinal anaesthesia usually leads to severe hypotension; alpha agonists do not increase either of these parameters.

An increase in heart rate and cardiac output caused by positive inotropic drug use to raise blood pressure may cause a corresponding increase in myocardial oxygen demands than myocardial oxygen supply. Positive inotropic drugs, which enhance cardiac output by boosting myocardial contractility, may also be ineffective during spinal anaesthesia since myocardial contractility is unaffected. Increasing cardiac contractility when end-diastolic volumes are diminished due to lower preload may be counterproductive.

A vasopressor that causes venoconstriction selectively and doesn't change heart rate, afterload, or myocardial contractility is ideal and it would effectively address hypotension during spinal anaesthesia by targeting its root cause that is reduced preload. However, such an ideal vasopressor does not currently exist. Therefore, managing hypotension during spinal anaesthesia is best approached through physiological rather than pharmacological means.

Physiological management of hypotension during spinal anaesthesia focuses on increasing venous return to restore cardiac output. By putting the patient in a head-down position and encouraging internal auto-transfusion, this is accomplished efficiently.

In those with normovolemia, these interventions increase cardiac output and venous return, which causes blood pressure to come back to near normal levels.

The slight remaining decrease in blood pressure is attributed to reduced afterload resulting from arterial and arteriolar vasodilation.

Large amounts of electrolyte solutions are rapidly infused intravenously as an additional

method of blood pressure restoration. However, the primary goal in addressing hypotension during spinal anaesthesia is not solely to raise blood pressure but to restore tissue oxygenation. Vasopressors can increase blood pressure; however, they should only be used in limited circumstances in modern medicine due to their well-known effects on the balance between the supply and demand of oxygen in the heart.

Role of crystalloids and colloids[28]

Crystalloid solutions are classified as balanced hypotonic or hypertonic salt solutions and are comprised of water and electrolytes. They are employed to sustain intravascular fluid volume and supply essential water and electrolytes. The distribution of these solutions is 1:1:4 between interstitial fluid and plasma. Although standard saline (0.9% NaCl solution) is frequently used, other formulations can provide better therapeutic advantages for specific patient populations.

Other commercially available crystalloid fluids include:

Lactated Ringer's/Hartman's solution (lactate buffered solution)

Acetate and lactate buffered solution

Acetate buffered solution

0.45% NaCl (hypotonic solution)

Acetate and gluconate buffered solution

5% Dextrose in water

3% NaCl (hypertonic solution)

10% Dextrose in water

Mechanism of action

Aqueous solutions comprising mineral salts and other small molecules that dissolve in water are referred to as crystalloid fluids. Most commercially available of these are isotonic with human plasma, meaning they have similar solute concentrations to plasma but do not, in vivo, exhibit osmotic effects. These fluids expand the intravascular volume without markedly affecting the distribution of fluid between the intracellular, intravascular, and interstitial compartments or the ion concentrations.

Fluid shifts occur when solutions that are hypertonic, like 3% saline solutions, have solute concentrations higher than those of human serum and are osmotically active.

They are primarily used for urgent serum solute replacement, such as in cases of severe hyponatremia with neurological symptoms.

Compounds in buffered solutions break down in the body to produce bicarbonate, which aids in preserving the optimum physiological pH of the plasma. For this, substances like lactate, acetate, and gluconate are frequently utilised.

The liver converts lactate and gluconate into bicarbonate, and the skeletal muscle metabolises acetate.[28]

Ringer lactate[29]

Ringer's lactate solution, also called lactated ringer, is an isotonic crystalloid that can be buffered or balanced, and it is used to replenish lost fluid. In Ringer's lactate, sodium, potassium, calcium, chloride, and lactate in the form of sodium lactate are all mixed to produce a solution with a pH of around 6.5 and an osmolarity of 273 mOsm/L.

Mechanism of action

The compensatory base for lactic acid is lactate. Pyruvate is produced during normal aerobic glucose metabolism and is used in cellular respiration. However, some anaerobic metabolism

always occurs, leading to the conversion of pyruvate to lactate via lactate dehydrogenase (LDH), sustaining NAD+ levels for continued glycolysis.

Carbon dioxide (CO_2), water (H_2O), and ATP are eventually produced during cellular respiration, which maintains a balanced NADH/NAD+ ratio through the transfer of protons and electrons.

If the aerobic system fails, protons accumulate. To stabilize the NADH/NAD+ ratio, lactate is produced and transported out of cells, serving as a buffer by absorbing H+ ions and generating lactic acid. Through the action of LDH and cellular respiration, lactate can be converted back into pyruvate, yielding H_2O and CO_2. Carbonic anhydrase converts CO_2 and H_2O into carbonic acid (H_2CO_3), which quickly dissociates to produce bicarbonate (HCO^{3-}). Bicarbonate can also be produced by breaking down lactate.[30]

Indication

- Dehydration
- Moderate metabolic acidosis
- Restoration of normal fluid balance

Contraindication

- Addison’s disease
- Impaired lactate metabolism

Adverse effects

Ringer's lactate solution is thought to increase the risk of hyperkalemia and lactic acidosis. However, it's important to consider that Ringer's lactate contains only 4 mEq/L of potassium. Although it might seem sense that giving a patient who is hyperkalemic extra potassium would make their situation worse, this isn't always the case. Potassium equilibrates between

the intracellular and extracellular compartments because it has a greater volume of distribution within the body than the extracellular compartment.

Even in patients with renal failure, administering 4 mEq/L of potassium does not have a cumulative effect.[31] In addition, patients' hyperkalemia may deteriorate as a result of metabolic acidosis. Thus, after receiving a large-volume IV infusion of normal saline, individuals may develop hyperchloremic non-anion gap metabolic acidosis. With regard to Ringer's lactate, this is untrue.[32]

Because it could worsen lactic acidosis, ringer's lactate is typically avoided in septic patients. But since Ringer's lactate contains sodium lactate instead of lactic acid, this worry is unwarranted. The body produces extra lactate as a result of the Ringer's lactate administration, which the body uses as fuel. Nevertheless, blood lactate level monitoring is frequently highly valued in modern medical practice.[33]

Ringer's lactate allergic responses can cause a wide range of symptoms, from localized reactions like redness and itching to more severe ones including widespread symptoms, local infections, and even regional cellulitis. If left untreated, these local infections can progress to systemic infections. But rather than the Ringer's lactate itself, a lot of these symptoms can be related to the intravenous site access. Adhesive dressings used for IV access are more likely to cause allergic responses. IV infiltration is another possible problem that may result in localized discomfort, redness, and edema.

This is typically managed conservatively and by replacing the IV access. Depending on the infection's severity, treatment options for infections might range from systemic antibiotic therapy to local treatment.

Toxicity

The toxicity associated with Ringer's lactate is primarily attributed to volume overload

resulting from the intravenous administration of fluid, rather than the composition of Ringer's lactate itself. Excessive infusion of IV fluids can lead to fluid overload, manifesting in symptoms such as peripheral edema or, in severe cases, respiratory distress due to pulmonary edema. Patients experiencing symptoms should receive diuretic medication, and their serum electrolyte levels should be regularly monitored. Intubation or non-invasive positive pressure ventilation may be necessary for those experiencing severe respiratory distress. It is possible to manage asymptomatic fluid overload with fluid restriction and regular monitoring.[29]

Sequential Compression Devices (SCDs)

By delivering intermittent pressure to the limbs, sequential compression devices (SCDs) are medical devices intended to prevent deep vein thrombosis (DVT) and enhance blood circulation in the legs. These devices are commonly used in hospitals, particularly for patients who are immobile or at risk of developing blood clots due to surgery, trauma, or other medical conditions.

Mechanism of Action:

The way sequential compression devices deliver intermittent pressure to both the limbs. It consists of cuffs or inflatable sleeves that are tied on the lower limbs of the patient. These cuffs are connected to sleeves which controls the pressure and the process of inflation and deflation.

It has three phases:

1) Inflation: The inflation starts from the lower end of the limb(distal) and then ascends to the other end(proximal). This will help in pushing the blood from the feet upwards.
2) Sustained Compression: In this phase, after inflation, the pressure will be kept constant on the limbs and there will be no pooling in the peripheries.

3) Deflation: In this third phase, the cuff deflates all at once causing the blood to flow. This cycle then repeats again.

Physiological Effects:

This artificial compression acts like a muscle contraction that occurs in people when they walk. This helps in improving blood flow, reduces peripheral edema and prevents blood stasis.

Clinical Evidence:

Various studies have shown the efficacy of sequential compression devices in preventing DVT and improving blood circulation.

A study published in the Journal of Vascular Surgery (Meyer et al., 2018) compared the use of SCDs with pharmacological prophylaxis for preventing DVT in patients undergoing hip or knee replacement surgery. The results showed that SCDs were as effective as pharmacological agents in reducing the risk of DVT without increasing the risk of bleeding complications.

Another study published in the American Journal of Medicine (Amin et al., 2014) evaluated the use of SCDs in critically ill patients admitted to the intensive care unit (ICU). The researchers found that SCDs significantly reduced the incidence of DVT and pulmonary embolism (PE) in this high-risk population.

Clinical evidence supports their use as part of a comprehensive strategy for thromboprophylaxis in various clinical settings.

Various articles discussing the effectiveness of sequential compression device and crystalloid preload in reducing post spinal hypotension in caesarean section;

According to a study by Panigrahi BP et al. (2011), the SCD approach lowers the degree of hypotension by mobilizing more blood to the central [active] blood volume without the need for medication or intravascular fluids.[34]

In a study conducted by Sujata NA et al., (2013) to assess the sequential compression mechanical pump to prevent hypotension during elective caesarean section under spinal anesthesia. In Group M(mechanical pump), hypotension occurred in 12 out of 47 patients (25.5%), whereas in Group C(crystalloid), it occurred in 27 out of 45 patients (60%), indicating a significant difference (P = 0.001) The median ephedrine dose was higher in Group C (12 [range: 0–24] mg) compared to Group M (0 [range: 0–12] mg) (P < 0.001) Nonetheless, there was no noteworthy distinction between the groups regarding the duration until the development of hypotension. In conclusion, the incidence and severity of hypotension after spinal anaesthesia for a caesarean delivery were both decreased by the use of a sequential compression mechanical pump that senses venous refilling and modifies accordingly.[35]

In order to evaluate crystalloid preloading and co-loading to avoid spinal anesthesia-induced hypotension, Farid Z et al. (2016) did a study. There were 74 elective C-sections performed, with group P (preloading group) mean age of 28.38±5.07 years and group C(co-loading) mean age of 28.27±5.07 years. Eighteen patients (48.6%) in group-C and twenty-three patients (62.2%) in group-P had hypotension; there was nil statistically significant difference between the two groups (p=0.242). Overall, preloading and co-loading with 15 ml/kg of Hartmann's solution (lactated Ringer's solution) separately are ineffective in preventing hypotension in obstetric patients having spinal anaesthesia.[36]

According to a study by Zadeh FJ et al. (2017), SCD lowers significant variations in diastolic blood pressure and the frequency of nausea and vomiting after spinal anaesthesia for caesarean sections. The off-pump group exhibited significantly higher changes in heart rate, mean arterial pressure (MPA), diastolic arterial pressure (DAP), and systolic arterial pressure (SAP) at baseline and the first minute ($p<0.05$) compared to the control groups. However, differences in these changes between the off-pump group and control groups were significant at other times. Using a Sequential compression device (SCD) after spinal anaesthesia for a caesarean section effectively lowers hypotension, according to the research findings.

Additionally, it suggests that this method may lead to a decrease in the required dosage of vasopressors to increase blood pressure. However, further studies with larger sample sizes are needed to confirm its effectiveness definitively.[3]

Prajith KR et al., (2020) compared the elastic wrapping and pneumatic compression of lower limb and studied hemodynamic changes after spinal anaesthesia. The occurrence of hypotension was notably reduced in Group 2 and 3 compared to the control group. Likewise, there was a significant increase in the need for ephedrine in the control group compared to both compression band (CB) and pneumatic compression device groups (PCD). The occurrence of hypotension was lower in the CB group compared to the PCD group. Additionally, the time taken to administer the first dose of ephedrine was significantly shorter in the control group (7.37 ± 4.94 min) compared to CB (10 ± 2.8 min) and PCD (13.88 ± 9.23 min). In conclusion, employing leg-wrapping with CB proves to be a cost-effective, non-invasive, non- pharmacological, and efficient method to decrease the occurrence of hypotension following spinal anaesthesia in pregnant individuals.[1]

In a study conducted by Javaherforooshzadeh F et al., (2020) to assess the post spinal

hemodynamic changes in sequential compression device group for caesarean section, there were no notable distinctions between the groups in terms of patient characteristics, maximum sensory block, duration from skin incision to delivery, spinal anaesthesia to delivery time (in minutes), and total surgical duration. In relation to alterations in heart rate, repeated measures ANOVA revealed a significant variance attributed to time, groups, and the interaction between these factors ($P < 0.0001$, $P < 0.0001$, and $P < 0.0001$, respectively). Post hoc Tukey analysis indicated that three minutes following spinal anaesthesia, the SCD group exhibited notably higher diastolic blood pressure compared to the control group ($P < 0.05$). Furthermore, the SCD group displayed significantly reduced incidences of nausea (P 0.005) and vomiting ($P = 0.001$) compared to the control group. Furthermore, the SCD group demonstrated a substantially lower average ephedrine dosage per patient (4.1 mg versus 17.1 mg, $P = 0.001$). However, there were no significant differences observed between the groups concerning neonatal Apgar scores at 1 and 5 minutes. In conclusion, this investigation highlights that the use of SCD can mitigate substantial fluctuations in diastolic blood pressure, an important hemodynamic parameter, and decrease the occurrence of nausea and vomiting. Consequently, SCD could be incorporated into spinal anaesthesia protocols for elective caesarean sections.[37]

In a study by Bhardwaj N et al., (2020) to assess the crystalloid preloading and co-loading for prevention of hypotension. This study has a total of 50 participants. Both the P (preloading) and C (co-loading) groups had 25 patients each. After spinal anaesthesia, systolic blood pressure decreased in both groups, although the decline was greater in the P group (p0.001). The P group also required more mephentermine than the C group. For the prevention of maternal hypotension following subarachnoid block, co-loading of crystalloid fluid was considered superior than preloading of crystalloids.[38]

In a study by Nizar ND et al., (2020), the aim was to assess and compare the effect of preloading with gelatine, hydroxyethyl starch solution before spinal anaesthesia. SBP (P = 0.011), DBP (P = 0.002), and MAP (P = 0.001) were all significantly lower with both fluids. Heart rate was also significantly reduced over time (P 0.001). Between the two groups, there was no statistically significant difference in ephedrine use. Volulyte 6% and Gelaspan 4% did not cause significant changes in acid-base status. Using 500 mL of Volulyte 6% or Gelaspan 4% as pre-loading fluids did not substantially diminish the frequency of hypotension following spinal anaesthesia following orthopaedic lower limb surgery; however, both were beneficial in maintaining normal acid-base balance.[39]

Kishnani P et al., (2021) assessed the preloading and relative efficacy of ringer lactate (group R) and pentastarch (group P) before spinal anaesthesia in lower abdominal and lower limb surgeries. The frequency of hypotension was higher in group R than in group P. HR and SpO2 levels did not differ significantly across groups. In both groups, the difference in mean basal SBP and DBP was statistically insignificant. After that, group R experienced a higher decline in SBP and DBP at all time intervals, and there was a difference of statistical significance between the two groups. (p value 0.001). The amount of ephedrine utilized in group P was lower than in group R. Shivering and vomiting were minor consequences that were statistically insignificant. The fluids utilized caused no adverse responses. Volume preloading continues to play an important role in minimizing the occurrence and severity of hypotension in patients undergoing spinal anesthesia. Pentastarch 6% has been demonstrated to be a superior preloading agent as its both safe and effective in reducing post spinal hypotension in patients.[40]

A study by Sultan A et al., (2021) was done to assess the effect of colloid versus crystalloid preload on hemodynamic stability of lower limb surgeries under spinal anaesthesia. More

than a 20% reduction in systolic blood pressure occurred in 45% of crystalloid individuals compared to 15% of colloid patients. This difference was statistically significant ($p<0.01$). In terms of the need for ephedrine to address hypotension, (45% of patients in the crystalloid group required Ephedrine compared to 15% in the colloid group). This was statistically significant as well ($p<0.01$). In terms of managing blood pressure, ephedrine needs, and heart rate fluctuations, colloid solution outperformed crystalloid solution.[41]

According to a study done by Agarwal A et al., (2022) on use of SCD for preventing spinal induced hypotension in patients undergoing caesarean section concluded that usage of SCD is more effective than preload in preventing post spinal hypotension. Significant variances were noted in heart rate, systolic blood pressure, diastolic blood pressure, and mean arterial pressure. In the crystalloid group, there was a significantly greater drop in systolic blood pressure from the starting level ($p=0.043$). In conclusion, utilizing the sequential compression device proves beneficial in averting hypotension among expectant mothers undergoing elective caesarean section with spinal anesthesia.

MATERIALS & METHODS

MATERIAL & METHOD

SOURCE OF DATA:

Study Design: A Randomized control study

Study Duration: From September 2022 to December 2023

Study Participants: This study was conducted on patients posted for Elective Caesarean sections at R. L. Jalappa Hospital and Research Centre, Tamaka, Kolar.

Sampling Method: Computer generated random sequence of numbers concealed by closed envelope technique.

Sample size: 46(23 in each group)

SAMPLE SIZE CALCULATION:

FORMULA:

$n = (Z_{1-\alpha/2}+Z1-\beta)^2 \times ((P_1\times(1-P_1)+P_2\times(1-P_2)) / (P_1-P_2)^2$

Where:

n = Sample size,

$Z_{1-\alpha/2} = 1.96$ when $\alpha = 5\%$ for two-tailed hypothesis,

$Z_{1-\beta} = 0.842$ when $\beta = 20\%$ (test power = 80%),

P = Probability of the main outcome.

INCLUSION CRITERIA

- Patients more than 18 years of age with American Society of Anaesthesiologists (ASA) physical status 2 posted for elective caesarean section under spinal anaesthesia.

EXCLUSION CRITERIA

- Allergic to Local anaesthetic
- Coagulopathy- platelet count < 80,000/mm^3, INR > 1.5, PT > 4 sec control and APTT > 10 sec control
- Infection at the site of injection
- Neurological deficits like paraplegia and paresis of lower limb

SAMPLING PROCEDURE:

- The study was started after Institutional Ethical Clearance (IEC). Patients included in the study after obtaining written, informed consent. Study was conducted on patients more than 18 years of age undergoing elective caesarean section. Necessary investigations like Platelet count, PT, INR and APTT are done prior to surgical procedure.
- Peripheral Intravenous cannula was secured and IV fluids (Ringer's Lactate) was initiated.
- Patients in both groups were premedicated with the following medications 15minutes before the procedure.

1. Inj. Ranitidine 50mg IV
2. Inj. Ondansetron 4mg IV.

- Patients were divided into two groups using computer generated random number table

  Group S: Sequential compression device group

  Group C: Crystalloid Group

- Group C – Patients were preloaded with Ringer Lactate (RL) at 10ml/kg over 10 minutes.
- After preloading the patient, subarachnoid block was given and crystalloid (RL) continued at 2ml/kg/hour during the intraoperative period.
- Group S – Sequential compression device was applied and compressions initiated at 60mmHg prior to the subarachnoid block. 2ml/kg/hour of maintenance fluid was continued during the surgical procedure.
- In the Operation Theatre, routine monitors like pulse-oximeter, electrocardiogram, non-invasive blood pressure (NIBP), temperature monitoring was connected and monitored throughout the procedure.
- In the sitting position, L3-L4 interspace was identified and 2% Xylocaine was used for skin infiltration. Subarachnoid block was performed using 25-gauge Quincke's needle. After confirming the subarachnoid space and free flow of clear CSF, 2ml of 0.5% Hyperbaric Bupivacaine was injected intrathecally.
- Following the procedure, patients were positioned supine with 15-20 degree left lateral tilt. Level of blockade was assessed using pinprick test.
- Oxygen with face mask (3-5 liter/minute) was given, if the patient saturation is <95%.
- NIBP was measured every 3 minutes until delivery and thereafter every 5 minutes during the intraoperative period.
- In the event of maternal hypotension (drop in SBP by 20% of the baseline or SBP < 90mmHg) was treated with Inj. Mephentermine 3mg boluses.

- If bradycardia occurs (Heart rate <60 beats per minute), patients were treated with Inj. Glycopyrrolate 0.2mg i.v.
- After delivery Inj. Oxytocin 3 IU i.v stat was given and infusion was started at the rate of 10 IU/hour for 2hours.
- At the end of the surgery, sequential compression device was removed from patients in Group S and the level of block were reassessed.

PARAMETERS OBSERVED

- Heart rate
- SBP and mean arterial pressure (MAP)
- Oxygen saturation
- Electrocardiogram
- Temperature (Skin temperature)
- Urine Output

STATISTICAL ANALYSIS

All the data were entered in excel sheet and analyzed using SPSS v23.0 operating on windows 10. The data were summarized as mean, standard deviation, frequency and percentage. The summarized data were represented using tables, figures, bar diagram and pie chart. The mean difference between continuous data were compared using unpaired t-test and categorical data using chi-square test. For all statistical purpose, a p-value of <0.05 was considered statistically significant.

RESULTS

RESULTS

In present study total 60 patients fulfilling inclusion criteria are included with overall mean age of 26.8±3.5yrs. patients were divided into two groups as per the procedure of study criteria and grouped as 30 patients in crystalloid and other 30 patients SCD.

Table 1: Comparison of the mean age between the groups

	Crystalloid		SCD		p-value
	Mean	SD	Mean	SD	
Age yrs	27.3	3.8	26.3	3.4	0.369

The mean age of patients between the group were comparable with no significant difference noted.

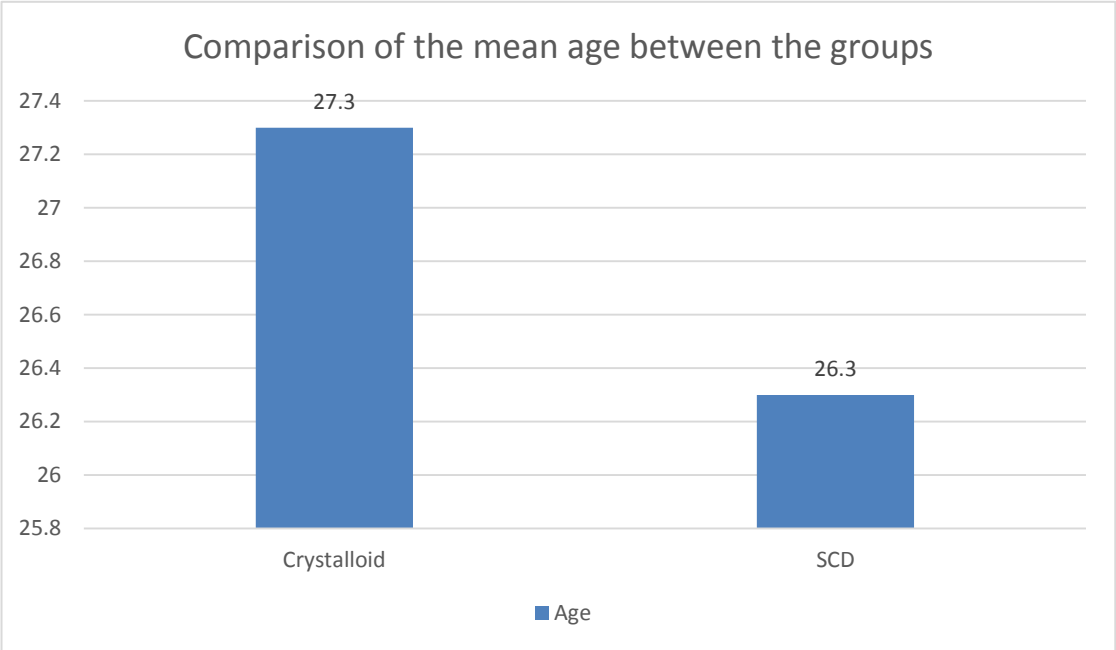

Figure 2: Comparison of the mean age between the groups

Table 2: Comparison of the heart rate between the groups

HR	Crystalloid		SCD		p-value
	Mean	SD	Mean	SD	
0min	102.9	12.4	111.5	12.0	0.08
3min	105.1	15.5	103.8	15.2	0.74
6min	94.6	19.4	98.5	19.3	0.43
9min	92.4	16.0	89.5	18.5	0.52
12min	90.6	15.0	92.8	18.6	0.62
15min	85.1	16.4	92.5	16.0	0.079
20min	92.5	13.8	90.7	13.2	0.615
25min	85.5	11.6	89.2	14.2	0.26
30min	88.0	13.4	88.0	11.5	1.0
35min	87.4	13.0	89.1	10.7	0.56
40min	90.4	13.1	87.5	11.4	0.36
45min	88.8	11.1	88.3	12.2	0.77
50min	88.0	9.1	86.1	10.5	0.45
55min	87.1	8.0	84.9	8.1	0.32
60min	86.7	6.6	83.0	7.3	0.07
65min	84.1	7.9	87.1	7.6	0.27
70min	84.7	4.3	82.0	8.5	0.58
75min	84.0	.	82.0	2.8	0.667

On assessment of the heart rate, there is no significant difference noted in mean heart rate changes between the group at various interval of time.

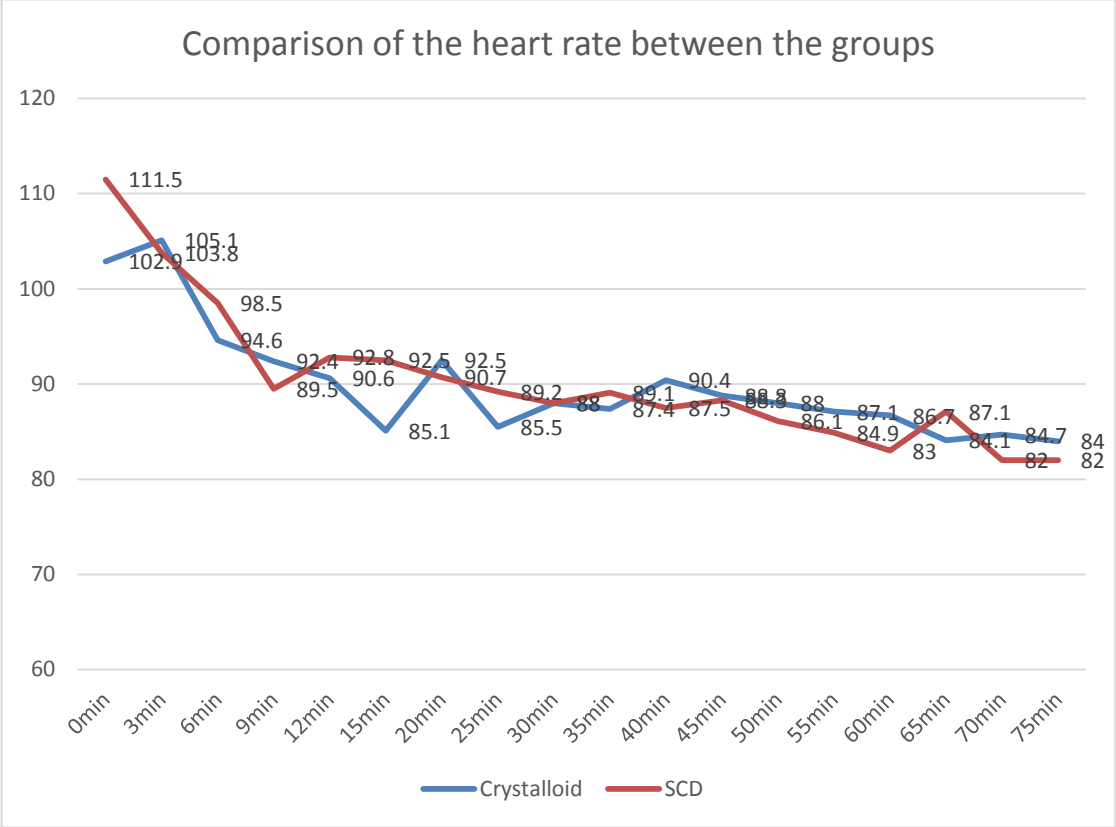

Figure 3: Comparison of the heart rate between the groups

Table 3: Comparison mean systolic blood pressure between the groups.

SBP	Crystalloid		SCD		p-value
	Mean	SD	Mean	SD	
0min	119.4	9.2	119.1	9.2	0.88
3min	104.5	11.9	106.8	9.5	0.40
6min	101.6	11.0	107.2	8.2	0.03*
9min	102.4	9.9	107.6	8.9	0.03*
12min	101.7	9.7	109.1	7.5	0.02*
15min	105.0	9.1	109.0	6.5	0.054
20min	106.6	8.9	110.1	8.0	0.11
25min	108.9	8.5	108.7	7.0	0.92
30min	105.8	9.8	106.7	7.4	0.70
35min	105.0	8.6	106.5	7.3	0.47
40min	104.5	7.7	106.8	6.4	0.21
45min	108.4	6.8	108.4	6.8	0.98
50min	106.8	5.7	108.2	5.3	0.32
55min	107.7	7.3	109.4	6.9	0.37
60min	108.2	6.0	110.6	5.4	0.16
65min	112.3	7.6	110.9	6.3	0.55
70min	113.7	8.5	113.0	6.2	0.87

On assessment of the systolic blood pressure, there is no significant difference noted in mean

heart rate changes between the group at various interval of time. However, there is significant drop in systolic blood pressure at 6^{th} to 12^{th} min of interval in crystalloid group compared to SCD.($p<0.05$)

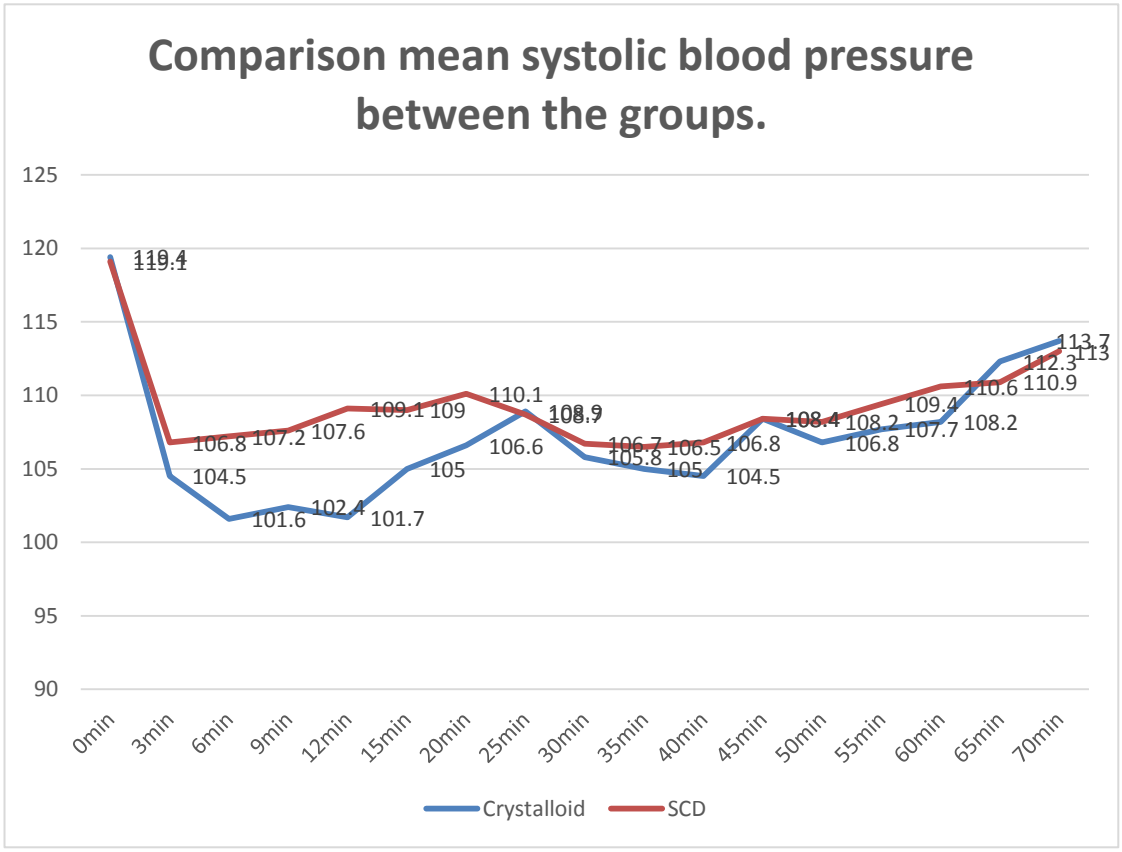

Figure 4: Comparison mean systolic blood pressure between the groups.

Table 4: Comparison of mean diastolic blood pressure between the groups

DBP	Crystalloid		SCD		p-value
	Mean	SD	Mean	SD	
0min	82.4	9.2	73.7	9.8	0.01*
3min	65.7	5.6	63.9	7.1	0.25
6min	64.6	7.8	65.8	6.1	0.27
9min	65.6	7.3	64.4	6.1	0.53
12min	67.1	5.1	67.5	6.4	0.50
15min	68.8	5.0	70.4	5.1	0.80
20min	67.1	5.1	67.5	4.2	0.21
25min	67.2	7.4	66.4	3.6	0.76
30min	68.1	3.8	67.3	3.4	0.59
35min	66.8	4.6	66.6	5.3	0.39
40min	68.3	6.1	66.3	5.1	0.86
45min	68.1	3.5	66.4	5.3	0.16
50min	68.7	4.8	66.0	4.6	0.15
55min	70.1	5.4	65.4	6.1	0.02*
60min	67.3	6.8	65.9	5.3	0.04*
65min	70.2	8.8	67.1	9.4	0.44
70min	70.3	5.5	70.3	12.3	0.34
75min	80.0	.	67.0	.	

On assessment of the diastolic blood pressure, there is no significant difference in the mean level between the group at various interval of time. However, there is significant lower mean DBP in group SCD at 55^{th} and 60^{th} min. ($p<0.05$)

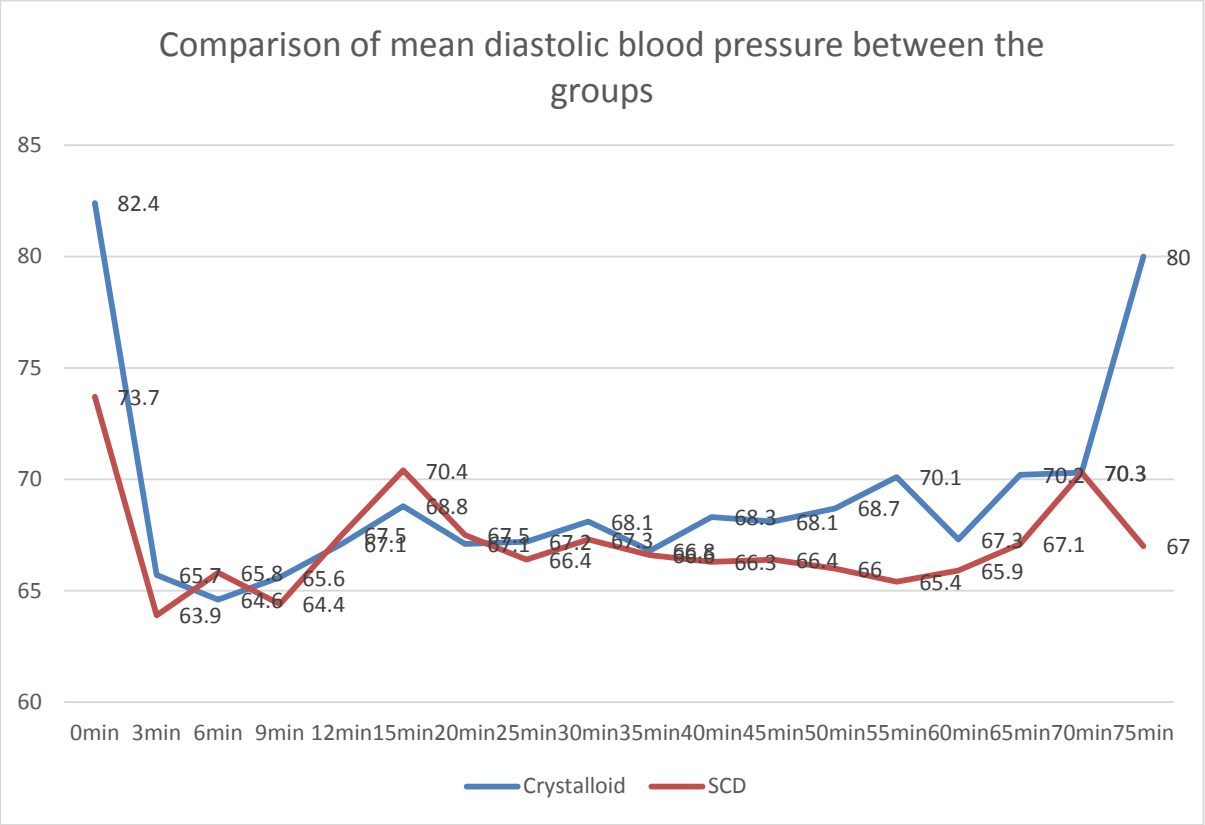

Figure 5: Comparison of mean diastolic blood pressure between the groups

Table 5: Comparison of the additional treatment required between the groups

Additional treatment		Crystalloid		SCD		Chi-square
		Count	N %	Count	N %	(p-value)
3 min	Nil	23	76.7%	28	93.3%	3.26 (0.07)
	3mg +200ml bolus	7	23.3%	2	6.7%	
6 min	Nil	22	73.3%	29	96.7%	6.4 (0.01)*
	3mg +200ml bolus	8	26.7%	1	3.3%	
9 min	Nil	25	83.3%	25	83.3%	1.11 (0.15)
	3mg	1	3.3%	0	0.0%	
	3mg +200ml bolus	4	13.3%	5	16.7%	
12 min	Nil	25	83.3%	30	100.0%	5.44 (0.065)
	100ml bolus	1	3.3%	0	0.0%	
	3mg +200ml bolus	4	13.3%	0	0.0%	
15 min	Nil	26	86.7%	30	100.0%	4.28 (0.11)
	3mg	1	3.3%	0	0.0%	
	3mg +200ml bolus	3	10.0%	0	0.0%	
20 min	Nil	28	93.3%	30	100.0%	2.06 (0.15)
	3mg +200ml bolu	2	6.7%	0	0.0%	
25 min	Nil	29	96.7%	29	96.7%	0.1 (0.99)
	3mg +200ml bolu	1	3.3%	1	3.3%	

30 min	Nil	29	96.7%	28	93.3%	0.35 (0.554)
	3mg +200ml bolus	1	3.3%	2	6.7%	
35 min	Nil	29	96.7%	29	96.7%	2.00 (0.368)
	3mg +100ml bolus	1	3.3%	0	0.0%	
	3mg +200ml bolus	0	0.0%	1	3.3%	
40 min	Nil	28	93.3%	30	100.0%	2.06 (0.355)
	3mg +100ml bolus	1	3.3%	0	0.0%	
	3mg +200ml bolus	1	3.3%	0	0.0%	

On assessment of additional treatment, there is no much significant difference in the distribution at different interval of time. However, there was significant higher incidence of requirement of the additional treatment in crystalloid group at 6^{th} min compared to SCD group.

DISCUSSION

DISCUSSION

Post-spinal hypotension is a prevalent complication during caesarean sections, posing significant risks to both the mother and foetus, including compromised placental perfusion and foetal distress. Various strategies are employed to mitigate this issue, primarily focusing on maintaining adequate blood pressure levels. Among these, the use of Crystalloid preload and Sequential compression devices (SCDs) are two common approaches. Crystalloid preload involves administering intravenous fluids to increase circulating blood volume before the onset of anaesthesia, aiming to prevent the anticipated drop in blood pressure. Conversely, Sequential compression devices (SCDs) mechanically enhance venous return by intermittently compressing the lower limbs, thereby supporting cardiovascular stability during anaesthesia. This study compares the effectiveness of these two interventions in managing post-spinal hypotension during caesarean sections, assessing their impacts on hemodynamic stability and the need for additional treatments.

The current study includes 60 patients in total who match the inclusion criteria. having a mean age of 26.8±3.5 years overall. According to the study's protocol, the patients were split into two groups: thirty patients were classified as SCD patients and the remaining thirty as crystalloid patients. There was no discernible variation in the mean age of the patients in the group.

Assessments of blood pressure and heart rate show no discernible differences between the groups. However, there is significant drop in systolic blood pressure at 6^{th} to 12^{th} min of interval in crystalloid group compared to SCD ($p<0.05$).

Similar to the current study, Agarwal A et al., documented the drop in systolic blood pressure from the initial level was notably higher in the crystalloid group ($p=0.043$).

In conclusion, sequential compression device use proves beneficial in averting hypotension among expectant mothers undergoing elective caesarean section with spinal anaesthesia.[2]

In a different trial by Sultan A et al., 45% of patients receiving crystalloid saw a systolic blood pressure reduction of more than 20%, compared to 15% of patients receiving colloids. There was a statistically significant difference ($p<0.01$). When it comes to the requirement for ephedrine to treat hypotension, 45% of patients in the crystalloid group and 15% in the colloid group, respectively, needed it. Additionally, this was statistically significant ($p<0.01$). Individuals receiving colloid were able to better control their heart rate swings, blood pressure, and demand for ephedrine than individuals getting crystalloid. [41]

Bhardwaj N et al., found that the prevention of maternal hypotension following subarachnoid block, co-loading of crystalloid fluid is superior than preloading of crystalloids.[38] Study by Javaherforooshzadeh F et al., found that three minutes following spinal anaesthesia, the SCD group exhibited notably higher diastolic blood pressure when compared to the control group ($P < 0.05$). [37]

According to a study by Sujata Na et al., the frequency and severity of hypotension following spinal anaesthesia for caesarean delivery were found to be reduced by using a mechanical sequential compression pump that detects venous refilling and adjusts accordingly. [35]

On assessment of additional treatment, there is no much significant difference in the distribution at different interval of time. However, there was significant higher incidence of requirement of the additional treatment in crystalloid group at 6^{th} min compared to SCD group.

In line study by Javaherforooshzadeh F et al., documented with the SCD group displayed significantly reduced incidences of nausea ($P = 0.005$) and vomiting ($P =0.001$) compared to

the control group. Furthermore, the SCD group demonstrated a substantially lower average ephedrine dosage per patient (4.1 mg versus 17.1 mg, P = 0.001).[37] In study by Zadeh FJ et al, the research indicates that employing Sequential Compression Device (SCD) effectively reduces hypotension following spinal anaesthesia for caesarean section. Additionally, it suggests that this method may lead to a decrease in the required dosage of vasopressors to increase blood pressure. However, further studies with larger sample sizes are needed to confirm its effectiveness definitively. [3]

LIMITATION

LIMITATIONS OF THE STUDY

1. Smaller sample size
2. Non consideration of analgesics given to the patient in wards before getting shifted to the operating room.
3. Patients receiving pre operative analgesics were not taken into consideration in this study.

CONCLUSION

CONCLUSION

In conclusion, Sequential Compression Devices appear to be more effective than Crystalloid Preload in mitigating early post-spinal hypotension during caesarean sections, making them a preferable option in this clinical scenario. Further research might focus on the long-term outcomes and patient comfort associated with each method to optimize perioperative care in caesarean sections.

SUMMARY

SUMMARY

In the current study a total 60 patients satisfying the inclusion criteria are selected with overall mean age of 26.8±3.5yrs. Patients were categorized into two groups according on the study criteria, with thirty patients in the crystalloid group and the remaining thirty in the SCD group.

- The mean age of patients between the group were comparable with no significant difference noted.
- Throughout different intervals of time, no discernible variation in heart rate has been seen between the groups.
- On assessment of the systolic blood pressure, there is no significant difference noted in mean systolic blood pressure changes between the groups at various interval of time. However, there is significant drop in systolic blood pressure at 6th to 12th min of interval in crystalloid group compared to SCD($p<0.05$).
- When it comes to diastolic blood pressure, there is no discernible variation in the group's mean level over time. However, there is significant lower mean DBP in group SCD at 55^{th} and 60^{th} min. ($p<0.05$)
- On assessment of additional treatment, there is no much significant difference in the distribution at different interval of time. However, there was significant higher incidence of requirement of the additional treatment in crystalloid group at 6^{th} min compared to SCD group.

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ANNEXURE

ANNEXURE

PROFORMA

TO COMPARE THE EFFECTIVENESS OF SEQUENTIAL COMPRESSION DEVICE VS CRYSTALLOID PRELOAD IN REDUCING POST SPINAL HYPOTENSION IN CAESAREAN SECTION

Personal Details:

NAME: AGE: SEX:
ADDRESS:
OCCUPATION:
SOCIOECONOMIC STATUS:
GESTATIONAL AGE:
PREVIOUS CAESAREAN SECTIONS:

HEIGHT: IBW:
TELEPHONE NO: UHID NO:
ASA GRADING:

Co-Morbidities:

Systemic examination:

RS - CVS -
CNS - P/A -

Investigations:

INVESTIGATION	PATIENT	CONTROL
1. Platelet count(th/mm^3)		
2. PT(sec)		
3. APTT(sec)		
4. INR		

Clinical Diagnosis:

Proposed operation:

Group:

Level of sensory block:

Time of delivery:

Vitals:

TIME (min)	HR (bpm)	BP (mmHg)	MAP (mmHg)	SPO_2(%)	RR (cpm)
0 min					
3min					
6min					
9min					
12min					
15min					
20min					
25min					
30min					
35min					
40min					
45min					
50min					
55min					
60min					
65min					
70min					
75min					
80min					
85min					
90min					

INFORMATION SHEET

TITLE: TO COMPARE THE EFFECTIVENESS OF SEQUENTIAL COMPRESSION DEVICE VS CRYSTALLOID PRELOAD IN REDUCING POST SPINAL HYPOTENSION IN CAESAREAN SECTION

I, Dr. Revathi Ashok post graduate in the department of Anaesthesiology, Sri Devaraj Urs Medical College, Kolar. We are carrying out above mentioned study at RLJH, Tamaka, Kolar. The study has been reviewed and approved by the institutional ethical review board. We will be studying the effectiveness of sequential compression devices in reducing hypotension after spinal anaesthesia in patients undergoing elective caesarean section.

Participation in this study doesn't involve any added cost to the patient. There is no compulsion to participate in this study and you will not be affected with regard to patient care, if you wish not to be part of this study.

All the information collected from the patient will be kept confidential and will not be disclosed to any outsider, unless compelled by the law. The information collected will be used only for this study. I request your kind self to give consent for the above-mentioned research project.

For any further clarification you are free to contact,

Dr. REVATHI ASHOK

Mobile no: 7259003715.

INFORMED CONSENT FORM

TO COMPARE THE EFFECTIVENESS OF SEQUENTIAL COMPRESSION DEVICE VS CRYSTALLOID PRELOAD IN REDUCING POST SPINAL HYPOTENSION IN CAESAREAN SECTION

Date:

I, _____ aged _____ ,after being explained in my own vernacular language about the purpose of the study, risks and complications of the procedure, hereby give my valid written informed consent without any force or prejudice for using sequential compression device and crystalloid preload. The nature and risks involved have been explained to me to my 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 consent voluntarily to participate in this research. I hereby give consent to provide my history, undergo physical examination, investigations, procedure and provide its results and documents to the doctor / institute etc. All the data may be published or used for any academic purpose. I will not hold the doctors / institute 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.

_____

(Name & Signature of Patient / patient Attendant)

Witness 1:

Witness 2:

_____

(Signature & Name of Research person /doctor)

ಮಾಹಿತಿ ಮತ್ತು ಒಪ್ಪಿಗೆ ನಮೂನೆ

ಸಿಸೆರಿಯನ್ ವಿಭಾಗದಲ್ಲಿ ಬೆನ್ನುಮೂಳೆಯ ನಂತರದ ಹೈಪೊಟೆನ್ಷನ್ ಅನ್ನು ಕಡಿಮೆ ಮಾಡುವಲ್ಲಿ ಅನುಕ್ರಮ ಸಂಕೋಚನ ಸಾಧನದ VS ಕ್ರಿಸ್ಟಲಾಯ್ಡ್ ಪ್ರೀಲೋಡ್ನ ಪರಿಣಾಮಕಾರಿತ್ವವನ್ನು ಹೋಲಿಸಲು

ದಿನಾಂಕ:

ನಾನು,_____

_____ , ಅಧ್ಯಯನದ ಉದ್ದೇಶ ಮತ್ತು ಕಾರ್ಯವಿಧಾನದ ಅಪಾಯಗಳು ಮತ್ತು ತೊಡಕುಗಳ ಬಗ್ಗೆ ನನ್ನದೇ ಆದ ಸ್ಥಳೀಯ ಭಾಷೆಯಲ್ಲಿ ವಿವರಿಸಿದ ನಂತರ, ಅನುಕ್ರಮ ಸಂಕುಚಿತ ಸಾಧನವನ್ನು ಬಳಸುವುದಕ್ಕಾಗಿ ಯಾವುದೇ ಬಲ ಅಥವಾ ಪೂರ್ವಾಗ್ರಹವಿಲ್ಲದೆ ನನ್ನ ಮಾನ್ಯ ಲಿಖಿತ ತಿಳುವಳಿಕೆಯನ್ನು 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. 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 ವೀಡಿಯೊ ಗ್ರಾಫ್ ಅಥವಾ ಛಾಯಾಚಿತ್ರ ಮಾಡಬಹುದು . ಎಲ್ಲಾ ಡೇಟಾವನ್ನು ಪ್ರಕಟಿಸಬಹುದು ಅಥವಾ ಯಾವುದೇ ಶೈಕ್ಷಣಿಕ ಉದ್ದೇಶಕ್ಕಾಗಿ ಬಳಸಬಹುದು. ಕಾರ್ಯವಿಧಾನ / ಅಧ್ಯಯನದ ಸಮಯದಲ್ಲಿ ಯಾವುದೇ ಅಹಿತಕರ ಪರಿಣಾಮಗಳಿಗೆ ನಾನು ವೈದ್ಯರು / ಸಂಸ್ಥೆ ಇತ್ಯಾದಿಗಳನ್ನು ಜವಾಬ್ದಾರರನ್ನಾಗಿ ಮಾಡುವುದಿಲ್ಲ.

ಭಾಗವಹಿಸುವವರಿಗೆ ಈ ತಿಳುವಳಿಕೆಯುಳ್ಳ ಒಪ್ಪಿಗೆ ನಮೂನೆ ಮತ್ತು ರೋಗಿಗಳ ಮಾಹಿತಿ ಹಾಳೆಯ ಪ್ರತಿಯನ್ನು ಒದಗಿಸಲಾಗಿದೆ.

_____

(ರೋಗಿ / ರೋಗಿಯ ಪರಿಚಾರಕರ ಸಹಿ ಮತ್ತು ಹೆಸರು)

ಸಾಕ್ಷಿ 1: _____

ಸಾಕ್ಷಿ 2: (ಸಂಶೋಧನಾ ವ್ಯಕ್ತಿ/ವೈದ್ಯರ ಸಹಿ ಮತ್ತು ಹೆಸರು)

KEY TO MASTER CHART

SBP	Systolic Blood Pressure
DBP	Diastolic Blood Pressure
HR	Heart Rate
ASA	American Society of Anesthesiologists
MIN	Minutes
Group C	Crystalloid group
Group S	Sequential compression device group

MASTER CHART

MASTERCHART

SL NO.	Group	UHID NUMBER	AGE(yrs)	GENDER	ASA	HR_0min	HR_3min	HR_6min	HR_9min	HR_12min	HR_15min	HR_20min	HR_25min	HR_30min	HR_35min	HR_40min	HR_45min	HR_50min	HR_55min	HR_60min	HR_65min	HR_70min	HR_75min
1	Crystalloid	213037	28	F	2	86	82	90	74	88	98	100	98	99	105	106	97	93	91				
2	Crystalloid	241996	30	F	2	101	108	124	112	120	127	122	118	119	120	115	123	99					
3	Crystalloid	328188	27	F	2	112	80	66	70	80	82	88	90	82	78	80	104	92	94	88	82	80	
4	Crystalloid	228397	27	F	2	120	110	102	108	100	98	92	96	88	80	84	90	92	80	78	70		
5	Crystalloid	228900	21	F	2	133	120	112	90	96	99	94	94	86	104	100	106	98	90				
6	Crystalloid	192715	33	F	2	100	96	90	88	86	70	66	68	72	70	76	80	81					
7	Crystalloid	225042	30	F	2	88	66	64	62	68	60	78	76	84	88	82	90	92	90	90	92		
8	Crystalloid	226523	28	F	2	98	100	108	98	92	102	104	90	88	70	76	78	76	80	82			
9	Crystalloid	228179	24	F	2	106	66	60	68	70	68	82	84	82	84	88	90	100	98	92	86	84	84
10	Crystalloid	192715	33	F	2	110	108	80	72	78	82	80	78	76	70	74	80	84	82	82			
11	Crystalloid	225042	30	F	2	78	108	60	72	78	70	72	76	70	74	78	84	80	88	94	90		
12	Crystalloid	230261	28	F	2	110	100	105	98	88	84	120	70	78	82	88	90	82	78	72			
13	Crystalloid	231165	24	F	2	104	110	100	90	120	100	96	70	110	102	100	98	90	100	80	80	82	
14	Crystalloid	231802	31	F	2	106	115	97	99	102	88	96	90	92	90	100	88	82	84				
15	Crystalloid	208237	29	F	2	122	110	86	90	92	75	96	70	78	77	75	74	72	70	88	92	90	
16	Crystalloid	229613	34	F	2	126	118	116	110	112	88	88	78	90	92	82	84	78	80	89	77		

17	Crystalloid	238985	29	F	2	98	125	128	110	88	62	90	84	90	92	98	86	90	102				
18	Crystalloid	237062	22	F	2	90	122	109	108	100	78	92	94	80	88	74	78	84	80	88			
19	Crystalloid	242055	25	F	2	88	119	121	110	80	65	92	88	74	78	84	80	88	90	82	70		
20	Crystalloid	242521	34	F	2	102	110	110	100	90	93	84	98	82	88	90	82	78	80	88	80	82	
21	Crystalloid	244319	29	F	2	108	122	87	102	88	69	110	68	80	82	88	90	82	78	80			
22	Crystalloid	218958	30	F	2	104	110	85	90	72	65	84	98	100	98	92	96	88	80	84	100		
23	Crystalloid	247596	27	F	2	94	108	110	120	90	86	84	80	88	70	80	88	82	84	86	82		
24	Crystalloid	244752	21	F	2	96	100	98	99	105	106	97	93	60	72	78	70	72	90	100			
25	Crystalloid	249479	25	F	2	100	123	118	119	120	115	123	99	105	98	88	84	110	100	90	86		
26	Crystalloid	245930	24	F	2	92	96	94	86	104	100	106	78	100	90	120	100	96	84	98	80		
27	Crystalloid	252083	20	F	2	102	105	68	72	70	76	80	80	97	99	102	88	96	84	80	90		
28	Crystalloid	246294	28	F	2	110	110	76	84	88	82	90	80	86	90	92	75	96	97	93	90		
29	Crystalloid	243764	24	F	2	112	116	90	88	70	76	78	88	116	110	112	88	88	92				
30	Crystalloid	254996	25	F	2	90	89	84	82	84	88	90	90	88	80	110	102	100	92	90	82	90	
31	SCD	222532	33	F	2	110	121	100	98	99	100	111	112	114	100	98	90	98	84	80	88		
32	SCD	228197	32	F	2	130	110	122	100	103	88	78	74	90	98	96	95	90	92	86	82	90	84
33	SCD	230236	28	F	2	100	112	102	100	90	94	92	90	88	76	75	70	76	78	76			
34	SCD	231165	24	F	2	88	80	76	66	68	72	74	70	68	77	79	72	70	84	82	80		
35	SCD	231803	25	F	2	108	110	104	90	98	110	111	120	98	90	94	100	102	98				

36	SCD	328188	27	F	2	120	122	118	109	108	110	98	70	76	80	82	88	78	76	70			
37	SCD	228397	27	F	2	130	110	90	82	66	68	74	70	84	89	74	70	68	80				
38	SCD	228749	26	F	2	98	102	90	76	70	75	74	70	68	80	82	84	88	74	76			
39	SCD	222532	33	F	2	88	80	65	64	68	100	102	98	90	86	89	90	81					
40	SCD	228197	32	F	2	122	108	108	110	100	102	98	90	84	86	92	94	88	86	78	78		
41	SCD	231222	26	F	2	112	100	62	60	68	82	88	90	92	102	98	94	96	84	80	88	78	
42	SCD	228179	24	F	2	130	110	100	102	104	100	98	78	80	82	88	80	76	78	70	86		
43	SCD	231803	25	F	2	120	115	123	99	105	98	78	77	75	74	72	70	82	78				
44	SCD	232242	26	F	2	104	100	106	78	100	90	90	92	82	84	78	80	88	84	88	78	76	80
45	SCD	240142	23	F	2	118	119	120	115	123	99	105	98	86	104	100	106	78	100	90	100		
46	SCD	237985	27	F	2	116	110	112	88	88	92	84	88	90	90	88	80	110	102	100	92	90	
47	SCD	243564	26	F	2	120	100	96	70	110	102	100	98	90	100	80	84	80	88	90	82	70	
48	SCD	226449	24	F	2	118	118	120	115	123	99	105	98	88	84	110	100	90	86				
49	SCD	246115	22	F	2	122	110	112	108	100	98	96	90	86	84	66	76	82	80	76			
50	SCD	229404	23	F	2	100	78	92	94	80	88	74	78	84	80	88	96	84	80	90	98		
51	SCD	158874	24	F	2	115	97	99	102	88	96	90	92	90	100	88	82	80	104	92	94	88	
52	SCD	248793	24	F	2	101	108	124	112	120	127	122	118	119	120	115	120	70	76	80	81		
53	SCD	250455	21	F	2	110	108	80	72	78	82	80	78	72	70	74	80	84	82	88			
54	SCD	239053	31	F	2	10	11	85	90	72	65	84	98	10	98	92	96	88	80	84	98		

						4	0							0									
55	SCD	252558	28	F	2	98	115	123	99	105	98	88	84	98	86	104	100	106	78				
56	SCD	252577	23	F	2	120	100	96	70	110	102	100	98	90	100	80	80	82					
57	SCD	248937	22	F	2	121	110	80	65	92	88	74	74	78	84	80	88	96	84	80	82		
58	SCD	253203	28	F	2	100	66	68	70	64	68	76	102	98	94	96	84	80	82	78			
59	SCD	255565	25	F	2	123	118	119	120	115	123	99	105	98	88	84	110	100	90	86			
60	SCD	255603	30	F	2	98	66	64	62	68	60	78	76	84	88	82	90	92	90	88			

SL NO.	Group	SBP_0min	SBP_3min	SBP_6min	SBP_9min	SBP_12min	SBP_15min	SBP_20min	SBP_25min	SBP_30min	SBP_35min	SBP_40min	SBP_45min	SBP_50min	SBP_55min	SBP_60min	SBP_65min	SBP_70min	SBP_75min
1	Crystalloid	113	124	118	92	92	85	97	95	78	78	87	101	100	102				
2	Crystalloid	111	121	110	118	118	118	115	128	109	108	111	115	110					
3	Crystalloid	132	105	92	110	112	117	126	123	108	105	109	111	102	124	121	125	129	
4	Crystalloid	110	86	90	92	99	101	105	111	112	107	109	113	118	117	122	121		
5	Crystalloid	122	104	100	99	102	105	110	111	114	120	109	112	110	109				
6	Crystalloid	126	110	88	100	98	92	96	102	106	103	107	110	112					
7	Crystalloid	130	84	90	92	98	100	102	101	104	99	100	103	105	110	106	111		
8	Crystalloid	108	90	92	98	102	104	110	116	90	102	104	105	113	109	110			
9	Crystalloid	121	102	100	103	99	102	104	110	107	112	109	121	104	110	114	116	107	122
10	Crystalloid	109	92	94	98	99	104	108	110	88	92	98	100	106	105	110			
11	Crystalloid	140	122	110	108	109	112	116	108	120	121	111	120	113	122	108	111		
12	Crystalloid	115	108	100	103	102	110	100	101	108	99	104	100	105	98	100			
13	Crystalloid	112	100	86	90	96	101	102	100	102	101	106	107	98	96	102	100	108	
14	Crystalloid	110	100	98	97	90	102	105	100	98	99	96	102	101	102				
15	Crystalloid	125	120	118	118	88	100	90	110	108	100	106	108	112	102	100	108	110	
16	Crystalloid	124	120	121	118	109	119	110	108	112	118	109	108	110	111	106	107		
17	Crystalloid	106	99	102	104	110	107	112	109	121	104	110	114	101	102				
18	Crystalloid	116	99	104	108	110	88	92	98	100	106	105	110	102	105	100			
19	Crystalloid	112	109	112	116	108	120	121	111	120	113	122	108	100	104	110	108		
20	Crystalloid	122	101	89	88	94	108	99	104	100	105	98	100	119	110	108	112	118	
21	Crystalloid	127	96	101	102	100	102	101	106	107	98	96	102	107	112	109			
22	Crystalloid	110	101	105	111	112	107	109	113	118	117	104	100	105	98	100	100		
23	Crystalloid	115	105	110	100	74	90	109	112	110	109	83	107	98	96	102	120		
24	Crystalloid	130	121	110	118	118	118	115	128	109	108	111	115	110	106	110			
25	Crystalloid	118	92	83	110	112	117	126	123	108	105	109	111	102	109	108	120		
2	Crystall	12	10	10	10	11	10	10	10	99	10	10	10	10	11	10	10		

6	oid	5	0	3	2	0	0	1	8		4	0	3	5	0	6	6		
27	Crystalloid	122	86	90	96	101	102	100	102	101	106	104	105	113	109	110	112		
28	Crystalloid	139	98	97	90	102	105	100	98	99	96	109	121	104	110	114	120		
29	Crystalloid	110	118	118	108	100	104	110	108	100	106	98	100	106	105	113	121		
30	Crystalloid	122	121	118	82	88	110	108	112	118	109	111	120	113	122	108	104	110	
31	SCD	117	96	101	102	100	102	101	106	107	101	103	102	107	112	109	106		
32	SCD	118	107	108	100	104	110	108	100	106	98	100	106	105	113	121	118	109	107
33	SCD	126	108	120	121	111	120	113	122	108	100	104	100	103	102	110			
34	SCD	139	122	110	92	109	112	116	109	101	121	111	120	113	122	108	116		
35	SCD	120	83	110	112	117	119	101	102	105	102	101	106	104	105				
36	SCD	121	110	118	118	108	100	104	110	108	100	106	98	100	106	105			
37	SCD	101	99	102	104	110	107	112	109	101	104	110	111	112	108				
38	SCD	131	121	110	119	118	114	117	128	109	108	111	115	110	107	113			
39	SCD	112	102	101	108	99	104	100	103	105	110	106	106	109					
40	SCD	110	108	112	118	109	111	120	113	122	108	104	110	110	108	100	106		
41	SCD	128	101	89	110	108	112	118	109	108	110	111	106	107	109	112	124	121	
42	SCD	125	118	109	108	111	115	110	110	111	114	120	109	112	110	109	111		
43	SCD	118	108	100	104	110	108	100	106	90	88	98	101	106	107				
44	SCD	112	118	109	111	120	113	122	108	104	110	102	101	106	104	105	113	109	110
45	SCD	122	111	110	108	106	110	108	112	118	109	111	120	113	122	108	104		
46	SCD	111	112	107	109	113	118	117	104	100	105	98	100	119	110	108	112	109	
47	SCD	139	98	97	90	103	107	101	98	99	101	109	121	104	110	114	120	121	
48	SCD	108	102	110	88	92	98	100	106	105	110	99	102	104	110				
49	SCD	120	118	118	109	100	97	110	108	100	106	108	112	102	100	108			
50	SCD	112	107	109	113	118	117	104	100	105	98	100	100	103	105	110	106		
51	SCD	110	108	100	106	98	100	106	105	113	109	112	116	108	120	121	111	109	
52	SCD	118	82	88	110	108	112	118	109	111	120	104	110	116	90	102	104		
53	SCD	125	100	103	102	110	100	101	108	94	104	100	103	105	110	116			

54	SCD	112	116	108	120	121	111	120	105	111	112	107	109	113	118	117	104		
55	SCD	121	110	118	118	118	115	128	109	108	98	100	106	105	110				
56	SCD	110	108	109	112	116	108	120	121	111	120	113	122	108					
57	SCD	120	113	122	109	102	105	110	108	119	113	120	108	100	104	110	108		
58	SCD	134	101	97	90	102	105	100	98	99	103	109	111	107	115	109			
59	SCD	122	110	108	109	112	116	108	120	121	111	120	113	122	108	111			
60	SCD	110	107	112	109	121	104	110	114	101	102	107	109	113	118	117			

SL NO.	Group	DBP_0min	DBP_3min	DBP_6min	DBP_9min	DBP_12min	DBP_15min	DBP_20min	DBP_25min	DBP_30min	DBP_35min	DBP_40min	DBP_45min	DBP_50min	DBP_55min	DBP_60min	DBP_65min	DBP_70min	DBP_75min
1	Crystalloid	81	78	74	68	64	54	62	62	53	51	58	69	70	70				
2	Crystalloid	84	68	51	65	68	68	67	98	76	70	80	76	81					
3	Crystalloid	82	61	60	72	70	71	82	78	66	62	62	68	66	82	81	78	81	
4	Crystalloid	82	54	52	55	62	70	69	71	70	69	63	72	73	75	76	72		
5	Crystalloid	84	70	66	58	68	64	60	68	70	72	69	70	71	68				
6	Crystalloid	90	70	50	56	68	66	62	68	70	64	70	64	68					
7	Crystalloid	88	60	62	60	66	68	62	68	68	66	70	74	78	76	72	90		
8	Crystalloid	90	60	62	66	70	80	71	72	66	60	68	64	69	70	72			
9	Crystalloid	82	60	70	68	70	66	69	62	69	65	67	68	71	72	80	72	70	80
10	Crystalloid	96	70	50	56	68	66	62	68	70	70	80	71	72	66	71			
11	Crystalloid	86	60	62	60	66	68	62	68	68	68	70	64	70	64	72			
12	Crystalloid	84	60	62	66	70	80	71	72	66	68	68	66	70	74				
13	Crystalloid	79	60	70	68	70	66	69	62	69	66	62	62	68	66	62	64	65	
14	Crystalloid	89	68	66	62	68	70	64	69	71	70	69	63	72	73				
15	Crystalloid	95	66	68	62	68	68	66	60	68	70	72	69	70	71	60	66	68	
16	Crystalloid	77	70	80	71	72	66	60	62	68	70	64	70	64	68	62	60		
17	Crystalloid	99	70	66	69	62	69	65	62	68	68	66	70	72	69				
18	Crystalloid	82	71	72	80	72	70	80	71	72	66	60	68	64	70	68			
19	Crystalloid	89	60	68	66	62	68	70	64	70	64	68	62	66	70	70	68		
20	Crystalloid	93	70	66	68	62	68	68	66	70	74	80	71	60	68	56	60	68	
21	Crystalloid	80	60	70	80	71	72	66	60	68	64	66	69	72	66	60			
22	Crystalloid	75	72	70	66	69	62	69	65	67	68	70	72	62	69	65	67		
23	Crystalloid	87	70	50	56	54	66	62	68	70	64	54	64	68	55	60	80		
24	Crystalloid	72	72	69	71	82	78	66	62	62	68	66	66	62	69	70			
25	Crystalloid	64	64	56	70	69	71	70	69	63	72	73	70	69	70	64	68		
26	Crystallo	66	66	70	64	60	68	70	72	69	70	71	70	72	78	68	66		

	id																		
27	Crystallo id	67	60	68	66	62	68	70	64	70	64	68	70	64	69	66	60		
28	Crystallo id	63	72	69	68	62	68	68	66	70	74	78	68	66	66	60	80		
29	Crystallo id	82	64	70	80	71	72	66	60	68	64	69	66	60	66	72	81		
30	Crystallo id	84	66	70	50	68	72	66	60	68	64	69	66	72	82	60	62	70	
31	SCD	99	70	66	69	62	69	65	62	68	68	66	70	72	69	70			
32	SCD	82	54	52	55	62	70	69	71	70	66	70	74	78	76	72	90	88	67
33	SCD	93	70	66	68	62	68	68	66	70	74	80	71	60	68	56			
34	SCD	82	70	66	58	60	68	66	62	68	70	64	66	64	62	61	67		
35	SCD	64	46	72	69	71	82	78	66	62	62	68	66	66	62				
36	SCD	62	68	68	66	70	74	78	68	66	66	60	80	63	72	69			
37	SCD	68	64	69	63	72	73	70	69	70	64	68	68	70	64				
38	SCD	68	68	66	60	68	70	72	69	70	71	60	66	68	56	63			
39	SCD	65	56	68	64	60	68	70	72	69	70	71	68	63					
40	SCD	62	69	65	62	68	68	66	70	72	69	68	70	64	70	64	68		
41	SCD	74	60	66	68	51	65	68	68	67	68	64	69	70	72	62	68	68	
42	SCD	66	60	68	64	69	66	60	66	68	64	60	68	70	72	69	70		
43	SCD	68	67	68	64	69	70	65	66	67	54	56	55	60	59				
44	SCD	62	68	70	64	70	64	68	62	66	69	62	69	65	67	68	56	57	
45	SCD	78	62	68	70	64	70	64	68	62	66	66	60	62	68	64	58		
46	SCD	68	68	66	70	74	80	71	60	68	56	69	63	72	73	70	69	70	
47	SCD	84	72	66	60	68	64	69	66	60	66	72	68	65	70	73	78	81	
48	SCD	67	55	66	51	68	68	66	70	74	80	71	60	68					
49	SCD	60	68	66	62	68	70	64	70	64	68	70	64	69	66	60			
50	SCD	71	70	69	63	72	73	70	69	70	68	70	72	69	70	71	70		
51	SCD	62	68	68	66	68	72	66	60	68	64	54	56	55	60	59	64	58	
52	SCD	75	47	49	68	62	68	68	66	70	74	65	67	68	50	53	51		
53	SCD	78	66	62	62	68	66	66	62	69	70	68	66	66	60	70			
54	SCD	77	70	80	69	72	66	60	62	68	69	65	67	68	60	68	64		
55	SCD	81	62	65	66	70	80	71	72	66	59	68	58	61	59				
56	SCD	72	68	69	71	82	78	66	62	62	68	64	60	68					
57	SCD	82	54	52	55	62	70	69	71	60	68	64	69	62	68	66	66		
58	SCD	86	61	58	54	60	62	60	66	68	62	68	68	65	62	68			
59	SCD	72	72	69	71	82	78	66	66	69	62	69	66	62	62	68			
60	SCD	82	64	70	80	71	72	66	66	68	62	68	68	68	70	71			

SL NO.	Group	treatment_	Additional treatment_ 3min	Additional treatment_ 6min	Additional treatment_ 9min	Additional treatment_ 12min	Additional treatment_ 15min	treatment_	treatment_	Additional treatment_ 30min	Additional treatment_ 35min	Additional treatment_ 40min
1	Crystalloid					100ml bolus	3mg			3mg+200ml bolus	3mg+ 100ml bolus	3mg +100ml bolus
2	Crystalloid											
3	Crystalloid			200ml bolus								
4	Crystalloid		3mg+200ml bolus		200ml bolus							
5	Crystalloid											
6	Crystalloid			3mg+200ml bolus			200ml bolus					
7	Crystalloid		3mg+200ml bolus	3mg+200ml bolus	200ml bolus							
8	Crystalloid			200ml bolus								
9	Crystalloid											
10	Crystalloid		200ml bolus									
11	Crystalloid											
12	Crystalloid											
13	Crystalloid			3mg+200ml bolus	200ml bolus							
14	Crystalloid											
15	Crystalloid					3mg+200ml bolus		200ml bolus				
16	Crystalloid											
17	Crystalloid											

18	Crystalloid						3mg+200ml bolus	200ml bolus				
19	Crystalloid											
20	Crystalloid			3mg+200ml bolus	3mg	200ml bolus						
21	Crystalloid		200ml bolus									
22	Crystalloid											
23	Crystalloid					3mg+200ml bolus	200ml bolus					3mg+200ml bolus
24	Crystalloid											
25	Crystalloid		200ml bolus	3mg+200ml bolus								
26	Crystalloid											
27	Crystalloid		200ml bolus+3mg	200ml bolus								
28	Crystalloid		200ml bolus+3mg						200ml bolus			
29	Crystalloid											
30	Crystalloid				3mg+200ml bolus	3mg+200ml bolus						
31	SCD											
32	SCD											
33	SCD											
34	SCD				3mg+200ml bolus							
35	SCD		3mg+200ml bolus									
36	SCD											
37	SCD											
38	SCD											
3	SCD											

9												
40	SCD											
41	SCD				3mg+200ml bolus							
42	SCD											
43	SCD									200ml bolus	3mg+200ml bolus	
44	SCD											
45	SCD											
46	SCD											
47	SCD				200ml bolus							
48	SCD				3mg+200ml bolus							
49	SCD											
50	SCD											
51	SCD											
52	SCD		3mg+200ml bolus		3mg+200ml bolus							
53	SCD									200ml bolus		
54	SCD											
55	SCD											
56	SCD											
57	SCD											
58	SCD				3mg+200ml bolus				200ml bolus			
59	SCD											
60	SCD											