Corona: Jurnal Ilmu Kesehatan Umum, Psikolog, Keperawatan dan Kebidanan
Vol.2, No.3 September 2024
e-ISSN: 3031-0172 ; p-ISSN: 3031-0180, Hal 159-185
DOI : https://doi.org/10.61132/corona.v2i3.597
Available Online at : https://journal.arikesi.or.id/index.php/Corona

Effect of Atropine as Premedication to Prevent Vasovagal Attack in Male Patients Undergoing Spinal Anaesthesia

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Abstract. *Background: Spinal anaesthesia (SA) is associated with many complications among which the most common side effects are hypotension and bradycardia, The aim of the current study was to assess the effects of prophylactic atropine in prevention of spinal anaesthesia induced hypotension and bradycardia in male. Aim of the Study: The aim of the current study was to assess the effects of prophylactic atropine in prevention of spinal anaesthesia induced hypotension and bradycardia in male. Method: Seventy (70) patients aged 25-60 years old, they had more than one type of surgery. the patients were monitored by the anaesthesiologist, and we monitored the patient's vital sign related to the operation period, such as blood pressure, heart rate, body temperature, and oxygen saturation, for patients who received atropine before the operation and patients who received atropine during the operation. Results: There were statistically significant differences in the results of the men who received atropine at the beginning of the operation, while for the people who did not receive atropine at the beginning of the operation, there were values that were not statistically significant in the variables of systolic and diastolic blood pressure and heart rate within 5 minutes of receiving spinal anaesthesia. Conclusion: Prophylactic atropine within 1 min of induction of spinal anaesthesia in male patients undergoing surgery reduce the incidence of hypotension and bradycardia.*

Keyword: *Anaesthesia, Hypotension, Spinal, Bradycardia.*

1. INTRODUCTION

Spinal anaesthesia (SA) is associated with many complications among which the most common side effects are hypotension and bradycardia. Spinal anaesthesia induced hypotension is believed to occur due to two possible mechanisms. The first and widely accepted mechanism is systemic vasodilation induced by sympathetic blockade, resulting in venous pooling of blood and reduction in systemic vascular resistance. This could be treated by administering peripheral vasoconstrictors thereby increasing the systemic vascular resistance and facilitating the venous return.

The second cause is believed to be the blunted reflex tachycardia. This may result from the blockade of cardio accelerator sympathetic fibres at T1 to T4, and due to the "reverse" of the Bainbridge reflex. To prevent this mechanism, prophylactic use of intravenous (IV) atropine might have importance.

Currently, different techniques are being used for the prevention of hypotension and bradycardia which include pre or co-loading of IV fluid, vasopressors, and physical methods such as table tilt, leg binders, and compression devices. However, none of these techniques alone are effective and there is a search for a technique or combinations of

Received: Juni 29, 2024; Revised: Juli 13, 2024; Accepted: Juli 27, 2024; Online Available: Juli 29, 2024

techniques for the proper prevention of spinal anaesthesia induced hypotension and bradycardia.

Elderly patients are prone to spinal anaesthesia induced hypotension and bradycardia than young adults. This is because they may have coexisting degenerative cardiovascular diseases with deranged reflex compensatory mechanisms for hypotension and bradycardia.

Atropine is more easily available and cost effective than vasopressors and IV fluids. Therefore, the results of the current study could be easily applicable in any resource limited hospital setups. Research showing the effect of atropine to prevent spinal anaesthesia induced hypotension are limited.

2. LITERATURE REVIEW

Spinal Cord

The spinal cord is a long, thin, tubular structure made up of nervous tissue that extends from the medulla oblongata in the brainstem to the lumbar region of the vertebral column (backbone) of vertebrate animals. The centre of the spinal cord is hollow and contains a structure called central canal, which contains cerebrospinal fluid. The spinal cord is also covered by meninges and enclosed by the neural arches. Together, the brain and spinal cord make up the central nervous system.

In humans, the spinal cord is a continuation of the brainstem and anatomically begins at the occipital bone, passing out of the foramen magnum and then enters the spinal canal at the beginning of the cervical vertebrae. The spinal cord extends down to between the first and second lumbar vertebrae, where it tapers to become the caudal equina. The enclosing bony vertebral column protects the relatively shorter spinal cord. It is around 45 cm (18 in) long in adult men and around 43 cm (17 in) long in adult women. The diameter of the spinal cord ranges from 13 mm (1⁄2 in) in the cervical and lumbar regions to 6.4 mm (1⁄4 in) in the thoracic area.

The spinal cord functions primarily in the transmission of nerve signals from the motor cortex to the body, and from the afferent fibres of the sensory neurons to the sensory cortex. It is also a centre for coordinating many reflexes and contains reflex arcs that can independently control reflexes. It is also the location of groups of spinal interneurons that make up the neural circuits known as central pattern generators. These circuits are responsible for controlling motor instructions for rhythmic movements such as walking.

Structure

The spinal cord is the main pathway for information connecting the brain and peripheral nervous system. [9] Much shorter than its protecting spinal column, the human spinal cord originates in the brainstem, passes through the foramen magnum, and continues through to the conus medullaris near the second lumbar vertebra before terminating in a fibrous extension known as the filum terminal.

It is about 45 centimetres (18 inches) long in males and about 43 cm (17 in) in females, ovoid-shaped, and is enlarged in the cervical and lumbar regions. The cervical enlargement, stretching from the C5 to T1 vertebrae, is where sensory input comes from, and motor output goes to the arms and trunk. The lumbar enlargement, located between L1 and S3, handles sensory input and motor output coming from and going to the legs.

The spinal cord is continuous with the caudal portion of the medulla, running from the base of the skull to the body of the first lumbar vertebra. It does not run the full length of the vertebral column in adults. It is made of 31 segments from which branch one pair of sensory nerve roots and one pair of motor nerve roots. The nerve roots then merge into bilaterally symmetrical pairs of spinal nerves. The peripheral nervous system is made up of these spinal roots, nerves, and ganglia.

The dorsal roots are afferent fascicles, receiving sensory information from the skin, muscles, and visceral organs to be relayed to the brain. The roots terminate in dorsal root ganglia, which are composed of the cell bodies of the corresponding neurons. Ventral roots consist of efferent fibres that arise from motor neurons whose cell bodies are found in the ventral (or anterior) Gray horns of the spinal cord. [10]

The spinal cord (and brain) are protected by three layers of tissue or membranes called meninges, that surround the canal. The dura mater is the outermost layer, and it forms a tough protective coating. Between the dura mater and the surrounding bone of the vertebrae is a space called the epidural space. The epidural space is filled with adipose tissue, and it contains a network of blood vessels. The arachnoid mater, the middle protective layer, is named for its open, spiderweb-like appearance. The space between the arachnoid and the underlying pia mater is called the subarachnoid space. The subarachnoid space contains cerebrospinal fluid, which can be sampled with a lumbar puncture, or "spinal tap" procedure. The delicate pia mater, the innermost protective layer, is tightly associated with the surface of the spinal cord. The cord is stabilized within the dura mater by the connecting denticulate ligaments, which extend from the enveloping pia mater

laterally between the dorsal and ventral roots. The Dural sac ends at the vertebral level of the second sacral vertebra.

In cross-section, the peripheral region of the cord contains neuronal white matter tracts containing sensory and motor axons. Internal to this peripheral region is the grey matter, which contains the nerve cell bodies arranged in the three grey columns that give the region its butterfly-shape. This central region surrounds the central canal, which is an extension of the fourth ventricle and contains cerebrospinal fluid. The spinal cord is elliptical in cross section, being compressed dorsolateral. Two prominent grooves, or sulci, run along its length. The posterior median sulcus is the groove in the dorsal side, and the anterior median fissure is the groove in the ventral side.

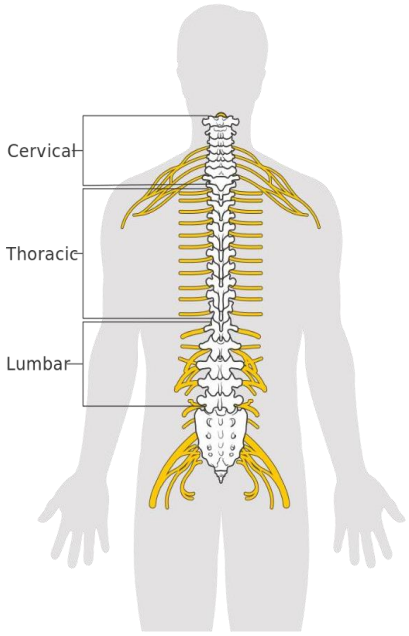

Figure (1): Diagram of the spinal cord showing segments

Segments

The human spinal cord is divided into segments where pairs of spinal nerves (mixed; sensory and motor) form. Six to eight motor nerve rootlets branch out of right and left ventrolateral sulci in a very orderly manner. Nerve rootlets combine to form nerve roots. Likewise, sensory nerve rootlets form off right and left dorsal lateral sulci and form sensory nerve roots. The ventral (motor) and dorsal (sensory) roots combine to form spinal nerves (mixed; motor and sensory), one on each side of the spinal cord. Spinal nerves, except for C1 and C2, form inside the intervertebral foramen. These rootlets form the demarcation between the central and peripheral nervous systems.

Generally, the spinal cord segments do not correspond to bony vertebra levels. As the spinal cord terminates at the L1-L2 level, other segments of the spinal cord would be positioned superior to their corresponding bony vertebral body. For example, the T11 spinal segment is located higher than the T11 bony vertebra, and the sacral spinal cord segment is higher than the L1 vertebral body.

The grey column, (as three regions of grey columns) in the centre of the cord, is shaped like a butterfly and consists of cell bodies of interneurons, motor neurons, neuroglia cells and unmyelinated axons. The anterior and posterior grey column present as projections of the grey matter and are also known as the horns of the spinal cord. Together, the grey columns and the grey commissure form the "grey H."

The white matter is located outside of the grey matter and consists almost totally of myelinated motor and sensory axons. "Columns" of white matter carry information either up or down the spinal cord.

The spinal cord proper terminates in a region called the conus medullaris, while the pia mater continues as an extension called the filum terminal, which anchors the spinal cord to the coccyx. The cauda equina ("horse's tail") is a collection of nerves inferior to the conus medullaris that continue to travel through the vertebral column to the coccyx. The cauda equina forms because the spinal cord stops growing in length at about age four, even though the vertebral column continues to lengthen until adulthood.

This results in sacral spinal nerves originating in the upper lumbar region. For that reason, the spinal cord occupies only two-thirds of the vertebral canal. The inferior part of the vertebral canal is filled with cerebrospinal fluid and the space is called the lumbar cistern.

Within the central nervous system (CNS), nerve cell bodies are generally organized into functional clusters, called nuclei. Axons within the CNS are grouped into tracts.

There are 31 spinal cord nerve segments in a human spinal cord:

8 cervical segments forming 8 pairs of cervical nerves (C1 spinal nerves exit the spinal column between the foramen magnum and the C1 vertebra; C2 nerves exit between the posterior arch of the C1 vertebra and the lamina of C2; C3–C8 spinal nerves pass through the intervertebral foramen above their corresponding cervical vertebrae, with the exception of the C8 pair which exit between the C7 and T1 vertebrae)

12 thoracic segments forming 12 pairs of thoracic nerves

5 lumbar segments forming 5 pairs of lumbar nerves

5 sacral segments forming 5 pairs of sacral nerves

1 coccygeal segment

In the fetus, vertebral segments correspond with spinal cord segments. However, because the vertebral column grows longer than the spinal cord, spinal cord segments do not correspond to vertebral segments in the adult, particularly in the lower spinal cord. For example, lumbar and sacral spinal cord segments are found between vertebral levels T9 and L2, and the spinal cord ends around the L1/L2 vertebral level, forming a structure known as the conus medullaris.

Although the spinal cord cell bodies end around the L1/L2 vertebral level, the spinal nerves for each segment exit at the level of the corresponding vertebra. For the nerves of the lower spinal cord, this means that they exit the vertebral column much lower (more caudally) than their roots. As these nerves travel from their respective roots to their point of exit from the vertebral column, the nerves of the lower spinal segments form a bundle called the cauda equina.

There are two regions where the spinal cord enlarges:

Cervical enlargement – corresponds roughly to the brachial plexus nerves, which innervate the upper limb. It includes spinal cord segments from about C4 to T1. The vertebral levels of the enlargement are roughly the same (C4 to T1).

Lumbar enlargement – corresponds to the lumbosacral plexus nerves, which innervate the lower limb. It comprises the spinal cord segments from L2 to S3 and is found about the vertebral levels of T9 to T12.

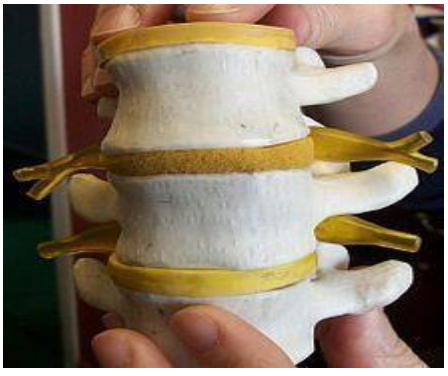

Figure (2): A model of segments of the human spine and spinal cord. Nerve roots can be seen extending laterally from the (not visible) spinal cord.

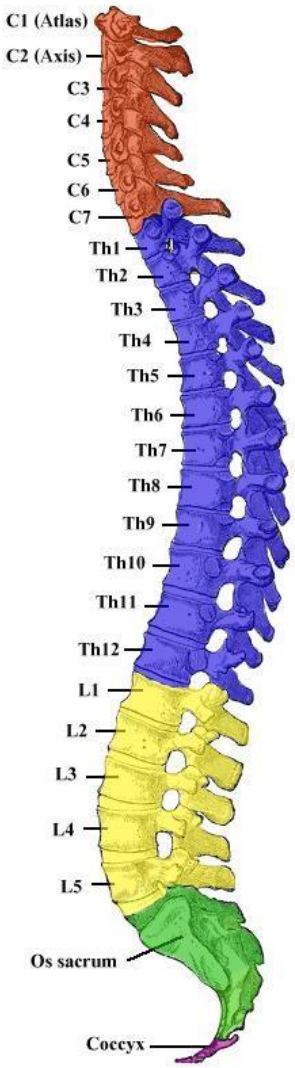

Figure (3): Segments of spinal

Development

The spinal cord is made from part of the neural tube during development. There are four stages of the spinal cord that arises from the neural tube: The neural plate, neural fold, neural tube, and the spinal cord. Neural differentiation occurs within the spinal cord portion of the tube. [13] As the neural tube begins to develop, the notochord begins to secrete a factor known as Sonic hedgehog (SHH). As a result, the floor plate then also begins to secrete SHH, and this will induce the basal plate to develop motor neurons. During the maturation of the neural tube, its lateral walls thicken and form a longitudinal groove called the sulcus limitans. This extends the length of the spinal cord into dorsal and ventral portions as well. Meanwhile, the overlying ectoderm secretes bone morphogenetic protein (BMP). This induces the roof plate to begin to secrete BMP, which will induce the alar plate to develop sensory neurons. Opposing gradients of such morphogens as BMP

and SHH form different domains of dividing cells along the dorsal ventral axis. Dorsal root ganglion neurons differentiate from neural crest progenitors. As the dorsal and ventral column cells proliferate, the lumen of the neural tube narrows to form the small central canal of the spinal cord. The alar plate and the basal plate are separated by the sulcus limitans. Additionally, the floor plate also secretes netrins. The netrins act as chemoattractant to decussation of pain and temperature sensory neurons in the alar plate across the anterior white commissure, where they then ascend towards the thalamus. Following the closure of the caudal neuropore and formation of the brain's ventricles that contain the choroid plexus tissue, the central canal of the caudal spinal cord is filled with cerebrospinal fluid.

Earlier findings by Viktor Hamburger and Rita Levi-Montalcini in the chick embryo have been confirmed by more recent studies which have demonstrated that the elimination of neuronal cells by programmed cell death is necessary for the correct assembly of the nervous system.

Overall, spontaneous embryonic activity has been shown to play a role in neuron and muscle development but is probably not involved in the initial formation of connections between spinal neurons.

Blood supply

The spinal cord is supplied with blood by three arteries that run along its length starting in the brain, and many arteries that approach it through the sides of the spinal column. The three longitudinal arteries are the anterior spinal artery, and the right and left posterior spinal arteries. These travel in the subarachnoid space and send branches into the spinal cord. They form anastomoses (connections) via the anterior and posterior segmental medullary arteries, which enter the spinal cord at various points along its length. The actual blood flow caudally through these arteries, derived from the posterior cerebral circulation, is inadequate to maintain the spinal cord beyond the cervical segments.

The major contribution to the arterial blood supply of the spinal cord below the cervical region comes from the radially arranged posterior and anterior radicular arteries, which run into the spinal cord alongside the dorsal and ventral nerve roots, but with one exception do not connect directly with any of the three longitudinal arteries. These intercostal and lumbar radicular arteries arise from the aorta, provide major anastomoses, and supplement the blood flow to the spinal cord. In humans the largest of the anterior

radicular arteries is known as the artery of Adamkiewicz, or anterior radicular is magna (ARM) artery, which usually arises between L1 and L2, but can arise anywhere from T9 to L5. Impaired blood flow through these critical radicular arteries, especially during surgical procedures that involve abrupt disruption of blood flow through the aorta for example during aortic aneurysm repair, can result in spinal cord infarction and paraplegia.

Spinal Anaesthesia

The development of regional anaesthesia started with the isolation of local anaesthetics, the first being cocaine (the only naturally occurring local anaesthetic). The first regional anaesthetic technique performed was spinal anaesthesia, and the first operation under spinal anaesthesia was in 1898 in Germany by August Bier. Before this, the only local anaesthetic techniques were topical anaesthesia of the eye and infiltration anaesthesia.

The central nervous system (CNS) comprises the brain and spinal cord. Neuraxial anaesthesia refers to the placement of local anaesthetic in or around the CNS. Spinal anaesthesia is a neuraxial technique where local anaesthetic is placed directly in the intrathecal (subarachnoid) space. The subarachnoid space houses sterile cerebrospinal fluid (CSF), the clear fluid that bathes the brain and spinal cord. An adult human has roughly 130 to 140 mL of CSF, which continually cycles throughout the day. Approximately 500 mL of CSF is produced daily.

Other neuraxial techniques include epidural and caudal anaesthesia, each having its particular indications. Spinal anaesthesia is only performed in the lumbar spine and is used for surgical procedures involving the lower abdomen, pelvis, and lower extremities.

Anatomy and physiology

The administration of spinal anaesthesia requires appropriate positioning and understanding of neuraxial anatomy. The goal is to deliver appropriately dosed aesthetic into the intrathecal (subarachnoid) space.

The spine comprises 7 cervical, 12 thoracic, 5 lumbar, and 5 fused sacral vertebral bones. The different vertebral bones earn their names based on their relative positions and structural differences. The vertebrae are stacked end-to-end with articulating joints and ligaments with a hollow space called the spinal canal. This canal houses the spinal cord. The spinal nerves exit the spinal canal via lateral spaces between pedicles from adjacent vertebrae.

As mentioned earlier, spinal anaesthesia is only performed in the lumbar area, specifically the mid to low lumbar levels, to avoid damage to the spinal cord and also to prevent intrathecally injected medications from having any activity in the upper thoracic and cervical regions. The caudal end of the spinal cord is the conus medullaris and usually is at the lower border of the first or sometimes the second lumbar vertebral body. It is a little inferior in paediatric patients, generally ending around L3. In the adult population, the mean conus position is the lower third of L1 (range: the middle third of T12 down to the upper third of L3). The variation in conus positions follows a normal distribution. No significant difference in conus position is seen between male and female patients or with increasing age. The Dural sac usually extends to S2/3.

For these reasons, the insertion of the spinal needle for spinal anaesthesia is usually at the L3/4 or L4/5 interspace. Spinal cord trauma is more likely when choosing higher interspaces, especially in obese patients. On entry and starting at the skin, the needle traverses several structures. The structures traversed depend on the approach.

Understanding dermatomal anatomy is imperative for understanding the blockade level of target structures. For example, the incision is usually made below the T10 dermatome for lower abdominal caesarean sections. However, coverage of up to T4 dermatome is required to prevent discomfort or pain from peritoneal tugging, especially with uterine manipulation. Patients complain of "pulling on their inside." Some corresponding dermatomal landmarks are:

C8: 5th finger

T4. Nipple

T7: Xiphoid process

T10: Umbilicus

Indications

Neuraxial anaesthesia is used as a sole anaesthetic or in combination with general anaesthesia for most procedures below the neck. As mentioned in the introduction, spinal anaesthesia is in common use for surgical procedures involving the lower abdomen, pelvis, perineal, and lower extremities; it is beneficial for procedures below the umbilicus.

Patient counselling regarding the procedure must be provided and signed informed consent is necessary. Since the procedure is usually performed on awake or slightly sedated patients, the indication for spinal anaesthesia and what to expect during placement of

neuraxial, risks, benefits, and alternative procedures are some of the discussions that can help allay anxiety. It is crucial to let patients understand that their ability to move their lower extremities is reduced until the resolution of the block.

Spinal anaesthesia is best for short procedures. For more extended procedures or procedures that would compromise respiration, general anaesthesia is usually preferable.

Contraindications

There are major known contraindications to neuraxial anaesthesia (spinal and epidural). The absolute contraindications are lack of consent from the patient and elevated intracranial pressure (ICP), primarily due to intracranial mass and infection at the site of the procedure (risk of meningitis).

Relative contraindications are Pre-existing neurological diseases (particularly those that wax and wane, eg, multiple sclerosis). Severe dehydration (hypovolemia) due to the risk of hypotension - risk factors for hypotension include hypovolemia, age greater than 40 to 50 years, emergency surgery, obesity, chronic alcohol consumption, and chronic hypertension. Thrombocytopenia or coagulopathy (especially with epidural anaesthesia due to the risk of epidural hematoma). Other relative contraindications are severe mitral and aortic stenosis and left ventricular outflow obstruction, as seen with hypertrophic obstructive cardiomyopathy. In the setting of coagulopathy, the placement of the neuraxial block requires re-evaluation. The American Society of Regional Anaesthesia (ASRA) publishes updated guidelines that detail timing for neuraxial anaesthesia for patients on oral anticoagulants, antiplatelets, thrombolytic therapy, unfractionated, and low molecular weight heparin. Review the latest guidelines before proceeding with the procedure. Overall because these are elective procedures, it is imperative to undergo a risk/benefit analysis before proceeding.

Equipment

Since the performance of neuraxial procedures is under an aseptic technique, the clinician is expected to maintain a sterile environment. Cap, masks, hand wash, and sterile gloves are required. For a successful procedure, adequate preparation is requisite. There should be adequate equipment count and space to accommodate patients and personnel. Monitors should be set up and ready to assess the patient's circulation (blood pressure, continuous EKG), oxygenation (continuous pulse oximetry), and temperature. The clinician performing the process must be proficient in using and interpreting monitors. If

planning sedation, means to assist patient ventilation, oxygenation, and circulatory support should be in place. Intravenous access should be established before starting. A certified anaesthesiologist should be present if the patient requires general anaesthesia.

There are commercially available spinal anaesthesia kits. Contents of kits usually include chlorhexidine with alcohol, drape, and local infiltrating aesthetic (usually 1% lidocaine). Other contents include the spinal needle (Quincke, Whitacre, Sprotte, or Greene), 3 mL and 5 mL syringes, and preservative-free spinal aesthetic solution. Solutions may range from lidocaine, ropivacaine, bupivacaine, procaine, or tetracaine.

Technique or treatment

Once the patient has undergone appropriate selection, the optimal patient position for the procedure must be established. The procedure is usually carried out with the patient sitting or lateral decubitus. The patient's comfort is tantamount. Positioning aims to help establish a straight path for needle insertion between the spinal vertebrae. The most used position is the sitting position. This is because the spinal anatomy is usually not laterally symmetrical in the lateral decubitus position as in the sitting position. With the patient positioned in the sitting position and leg hanging from the side of the bed, he/she should be encouraged to maintain a flexed spine position to help open up the interspace. The sitting position is appropriate for spinal anaesthesia with a hyperbaric solution. Either left or right lateral decubitus positions are viable options as well.

The access site is identified by palpation after the patient is properly positioned. This is usually very difficult to achieve with obese patients because of the amount of subcutaneous fat between the skin and the spinous process. The space between 2 palpable spinous processes is usually the site of entry. The patient should wear a hat or cover for his/her hair to maintain asepsis.

A strict aseptic technique is always necessary, achievable with chlorhexidine antiseptics with alcohol content, adequate handwashing, mask, and cap. Cleaning always starts from the chosen site of approach in circles and then away from the site. Allow time for the cleaning solution to dry. In the spinal kit, the drape placement is on the patient's back to isolate the access area. Local anaesthetic (usually about 1 mL of 1% lidocaine) is used for skin infiltration, and a wheal is created at the site of access chosen, either midline or paramedian.

In the midline approach, the spinal approach to the intrathecal space is midline with a straight line shot. After infiltration with lidocaine, the spinal needle is introduced into the skin, angled slightly cephalad. The needle traverses the skin, followed by subcutaneous fat. As the needle courses deeper, it engages the supraspinous and interspinous ligaments; the practitioner notes this as increased tissue resistance. The practitioner approaches the ligamentum flavum, and this would present like a "pop." On popping through this ligament is the approach to the epidural space, which is the point of placement for epidurally administered medications and catheters. This also presents the point where the loss of resistance is felt to the injection of saline or air. For spinal anaesthesia, the clinician proceeds with needle insertion until penetration of the dura-subarachnoid membranes, which is signalled by free-flowing CSF. It is at this point that the administration of spinal medication takes place.

For the paramedian approach, the skin wheal from the local anaesthetic is placed about 2 cm from the midline, and the spinal needle advances at an angle toward the midline. In this approach, the supraspinous and interspinous ligaments are usually not encountered. Hence, there is little resistance encountered until reaching the ligamentum flavum.

Complications

Appropriate patient selection and care should be established to help obviate common complications associated with neuraxial anaesthesia. While many of the complications are of very low incidence, it’s worth being aware of them. Severe complications are believed to be extremely rare, but the frequency is probably underestimated. Some common complications include the following. Backache (more common with epidural anaesthesia) Postural puncture headache (as high as 25% in some studies): non-cutting needle should be utilized for patients with a high risk for postural puncture headaches, and the smallest gauge needle available is recommended for all patients.

- Nausea, vomiting
- Hypotension
- Low-frequency hearing loss
- Total spinal anaesthesia (most feared complication)
- Neurological injury
- Spinal hematoma

- Arachnoiditis [23]
- Transient neurological syndrome (especially with lidocaine)

Vasovagal *attack*

The vasovagal response is characterized by an inappropriate combination of bradycardia and paradoxical vasodilation. During a general or spinal anaesthesia induced sympathectomy, a sudden vagal activation and/or an acute reduction in sympathetic tone can cause serious vasovagal responses. spinal anaesthesia for Caesarean section may trigger vasovagal response, due to multiple risk factors; High neuraxial block, sudden haemorrhage, aortocaval compression, peritoneal manipulation, and emotional stress In general, spinal anaesthesia The fire after a vasovagal attack usually begins in less than a minute. However, if you stopped quickly after fainting — within about 15 to 30 minutes — there was no evidence of fainting again.

Hypotension *in spinal anaesthesia*

Anaesthesia-induced hypotension (SAIH) occurs frequently, particularly in the elderly and in patients undergoing caesarean section. SAIH is caused by arterial and venous vasodilatation resulting from the sympathetic block along with a paradoxical activation of cardioinhibitory receptors. Bradycardia after spinal anaesthesia (SA) must always be treated as a warning sign of an important hemodynamic compromise. Hypotension, with an incidence of 15% to 33%, is one of the most frequent side effects of spinal anaesthesia (Spa)

***Clinical* significance**

Because the physiologic changes are associated with spinal anaesthesia, bradycardia and hypotension are relatively common side effects, even if they are not always clinically significant.4 Gradual heart rate reduction Hypo that stabilizes within 10% to 15% of baseline and is not associated requires careful observance but may not require treatment. Similarly, 15% to 20% reduction of arterial blood pressure in healthy patients without pre-existing hypertension, coronary

Treatment of* hypotension *in spinal anaesthesia

Ephedrine has traditionally been considered the vasoconstrictor of choice, especially for use during SAIH associated with bradycardia. Phenylephrine, a α1 adrenergic receptor agonist, is increasingly used to treat SAIH and its prophylactic administration (ie, immediately after intrathecal injection of local anesthetics) has been shown to decrease the incidence of arterial hypertension.

Premedication

Premedication is the administration of medication before a treatment or procedure. It is most commonly used prior to anesthesia for surgery, but may also be used prior to chemotherapy. This article relates to the use of premedication to prepare the patient for anesthesia and to help provide optimal conditions for surgery. Specific needs will depend on the individual patient and procedure.

- Purposes may include:
- Reduction of anxiety and pain.
- Promotion of amnesia.
- Reduction of secretions.

Reduction of volume and pH of gastric contents (to avoid Mendelson's syndrome. Reduction of postoperative nausea and vomiting. Enhancing the hypnotic effects of general anaesthesia Reduction of vagal reflexes to intubation.

Specific indications - eg, prevention of infective endocarditis with antibiotics. Premedication is traditionally given intramuscularly but the oral route is preferred for children and those with bleeding disorders. Premedication is usually given 20 minutes to three hours preoperatively. The choice of drug(s) used for premedication depends on the procedure, patient and aesthetic technique. Some patients prefer not to have premedication and potential benefits may be outweighed by potential problems (except for specific indications), especially with day-case surgery. A Cochrane review found no evidence of a difference in time to discharge from hospital following adult day surgery in patients who received anxiolytic premedication. Anxiety, amnesia and sedation

Careful discussion of the patient's concerns is essential, including at the preoperative assessment. Benzodiazepines are the usual agents used as they provide anterograde Amnesia.

Midazolam Clonidine (unlicensed) is also increasingly used for sedation, orally or IV. Dexmedetomidine is also used as a sedative with anxiolytic and analgesic properties.

Analgesia

Analgesic drugs given pre-emptively reduce the required dose of anesthetic agent and improve patient comfort in the immediate postoperative period. Options used include opioids, paracetamol, non-steroidal anti-inflammatory drugs (NSAIDs) and gabapentin NSAIDs, Opioids, Clonidine, Anticholinergics

These may be used to dry secretions in the mouth and airways, and to help reduce vagally mediated bradycardia and hypotension. They are required less commonly than in the past.

Atropine has strong sedative, amnesic and anti-salivation properties. It is a moderately effective antiemetic and potentiates opioids. Intramuscular atropine is therefore often prescribed together with an opioid. Atropine is the most potent agent available, with the added advantage of amnesia and sedation.

However, it can cause significant perioperative confusion in elderly patients. Anti-sialagogues (eg, glycopyrrolate intramuscularly or IV) are rarely needed but may be indicated for awake fibre-optic intubation or before ketamine anaesthesia. Anti-sialagogues may cause unpleasant dry mouth.

Antiemetic and anti-acidity

Antiemetic is used either to reduce the emetic effects of an aesthetic agents (antihistamines, butyrophenones, hyoscine) or to enhance gastric emptying (metoclopramide. An H2-receptor antagonist the general use of atropine. Vagal stimulation is known to protect the heart against arrhythmogenic vulnerability. Atropine abolishes this protective effect and thereby can cause severe arrhythmias. This side-effect is especially important in diabetics who often already have vagal denervation. 'Atropine is needed in bradycardia, but it has, as far as we know, no indications for use as premedication.

Atropine

Indications

Atropine or atropine sulfate carries FDA indications for anti-sialagogue/anti-vagal effect, organophosphate/muscarinic poisoning, and bradycardia. [24] it was originally synthesized from the plant Atropa belladonna, which is where the drug derives its name.

Ant sialagogue While atropine can be used independently for anti-salivation effects, it is not formally recommended for routine use in controlled airways, though it can

be used off-label for minimizing secretions in the intubated patient. Glycopyrrolate is at least five times more potent than atropine in its ant sialagogue effect.

Anticholinergic poisoning

Acetylcholine works on three different receptors that merit attention in nerve agent poisonings. Atropine is only useful to counter muscarinic effects (pralidoxime and benzodiazepines act on the others). If there are local symptoms in the eyes or respiratory tract, atropine is not indicated. Intravenous (IV) atropine indications include patients with hypersalivation, bronchial secretions, or bradycardia. Large doses and repeat doses may be required. Ingestions especially require higher doses (up to 20 mg).

Titrate to effect by monitoring the patient's ability to clear excess secretions. Pupils and heart rate are poor indications of appropriate dosing in these patients.

Bradycardia

Atropine is the first-line therapy (Class IIa) for symptomatic bradycardia in the absence of reversible causes. Treatments for brady dysrhythmias are indicated when there is a structural disease of the infra-nodal system or if the heart rate is less than 50 beats/min with unstable vital signs. Approximately 20% of brady dysrhythmias are due to endogenous cardiac electrical systems. The structural disease may or may not require resuscitation and should be closely monitored with medication and pacing readily available. If there is no improvement in the clinical state after repeat doses of atropine, additional treatments with atropine are unlikely to be effective. However, transient improvements with repeat dosing are an indication to continue treatment with atropine (which may exceed standard cumulative dosing maximums). Paediatric bradycardia is rarely cardiac and often secondary to hypoxia and hypoventilation. If bradycardia persists despite adequate respiratory support, atropine is indicated.

Mechanism of action

Atropine is an antimuscarinic that works through competitive inhibition of postganglionic acetylcholine receptors and direct vagolytic action, which leads to parasympathetic inhibition of the acetylcholine receptors in smooth muscle. The end effect of increased parasympathetic inhibition allows for preexisting sympathetic stimulation to predominate, creating increased cardiac output and other associated antimuscarinic adverse effects, as described herein.

Administration Atropine can be administered by intravenous (IV), subcutaneous, intramuscular, or endotracheal (ET) methods; IV is preferred. For ET administration, dilute 1 mg to 2 mg in 10 mL of sterile water or normal saline before administration. For paediatric ET, double the usual IV dose and dilute in 3 to 5 mL.

- Ant sialagogue/anti-vagal: 0.5 mg to 1 mg every 1 to 2 hours
- Organophosphate or muscarinic poisoning: 2 mg to 3 mg every 20 to 30 minutes (may require doses up to 20 mg, titrate to effect for secretion control)
- Bradycardia: 1 mg every 3 to 5 minutes (3 mg max), repeat until obtaining desired heart rate, most effective for sinus and AV nodal disease.
- Paediatric: 0.01 mg/kg to 0.03 mg/kg every 3 to 5 minutes. The paediatric minimum dose is 0.1 mg, the maximum dose is 0.5 mg (child) and 1.0 mg (adolescent), and the maximum cumulative dose is 1 mg (child) and 2 mg (adolescent).
- Rapid sequence intubation pre-treatment: 0.01 mg/kg IV for adults with bradycardia secondary to repeat dosing of succinylcholine. Paediatric 0.02 mg/kg IV, minimum dose 0.1 mg. Not recommended as a routine treatment.

In general, the dosing of atropine is repeatable every 5 minutes until reaching a maximum of 0.04 mg/kg.

Dosing in adults to greater than 0.5 mg and slow IV pushes correlate with paradoxical bradycardia (though likely transient) and ventricular fibrillation (VF).

Adverse effects

The most common adverse effects are related to the drug's antimuscarinic properties, including xerostomia, blurred vision, photophobia, tachycardia, flushing, and hot skin. Constipation, difficulty with urination, and anhidrosis can occur, especially in at-risk populations (most notably, the elderly). In rare cases, delirium or coma may occur. Hypersensitivity reactions may occur and are usually limited to a skin rash that could progress to exfoliation.

Atropine decreases the rate of mexiletine absorption, which can be prevented by combined IV delivery of metoclopramide with atropine before anaesthesia.

Contraindications

Atropine does not carry an FDA Boxed Warning nor any absolute indications. Multiple conditions carry a cautionary status. However, relative contraindications are overridden by the clinical need, especially in unstable or poisoned patients.

Clinicians need to exercise caution in patients with coronary heart disease, acute myocardial ischemia, congestive heart failure, tachycardia, or hypertension, as the increased cardiac demand and possible further worsening of tachycardia and hypertension can prove detrimental to patient outcomes.

Furthermore, caution is necessary for use with elderly patients, chronic lung disease patients, acute angle glaucoma, obstructive diseases (uropathy, toxic megacolon, paralytic ileus, pyloric stenosis, prostatic hypertrophy), myasthenia gravis, or in situations with environmental heat exposure.

Clinician's understanding of the adverse reactions makes the above cautionary situations easily recognizable by compounding effects on pre-existing conditions.

3. METHODOLOGY

Patients and methods

This study was carried out at Al Hussein Teaching Hospital. From December 2023 to February 2024. Under the supervision of the department of anaesthesia technologies at the College of Health and Medical Technology – Al-Ayen university. Seventy (70) patients aged 25-60 years old, they had more than one type of surgery.

The patients were monitored by the anaesthesiologist, and we monitored the patient’s vital sign related to the operation period, such as blood pressure, heart rate, body temperature, and oxygen saturation, for patients who received atropine before the operation and patients who received atropine during the operation.

Inclusion Criteria: The study enrolled adult patients aged 25 to 60 years and American Society of Anaesthesiologists (ASA) class I and II

Exclusion criteria: Patients with contraindications to SA, with cardiac arrhythmia such as atrial fibrillation, uncontrolled hypertension (defined as systolic blood pressure more than 180 mm Hg or diastolic blood pressure more than 110 mm Hg), and severe cardiac disease such as unstable angina, were excluded. Patients who were taking βadrenergic blockers, or any drugs that may alter normal response to atropine

Study design cross-sectional.

Ethical consideration

The study was conducted after approval of the ethical and scientific committee of the department of anaesthesia at Al Hussein Teaching Hospital.

Statistical Analysis

The results of the observations were then processed using the Fisher's exact test, t-test, and regression. using SPSS version 29 application.by Pearson chi-square test. P-value < 0.05 was considered statistically significant. All statistical analyses were done using SPSS ver.29.

4. RESULTS AND DISCUSSION

Table (1): Characteristics of patients who used atropine

Characteristics of patients who used atropine N = 46		
variable	mean	Std. Deviation
Age	31.54	10.54
Weight	83.71	5.40

Table (1) This table shows the mean and standard deviation for the characteristics of patients who received atropine before the operation, with a statistical significance in both variables, a P value less than 0.05

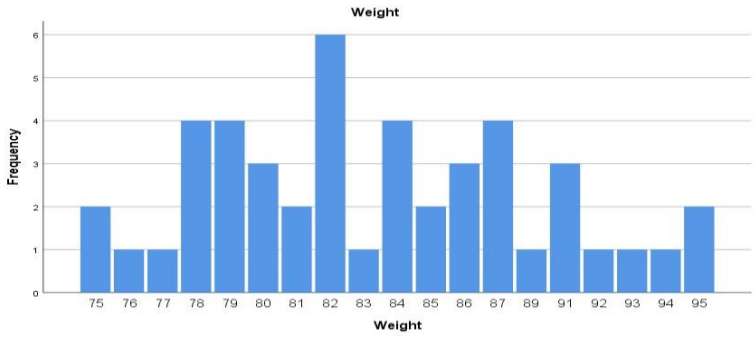

Figure (4): statistical analysis of weight

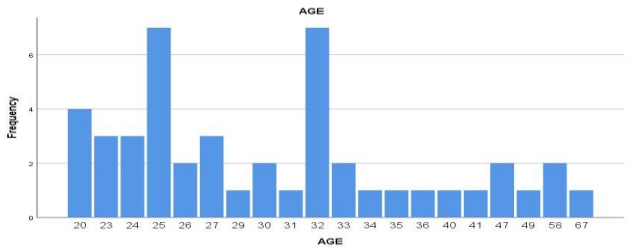

Figure (5): statistical analysis of age

Table (2): parameters of patients who used atropine

parameters of patients who used atropine N = 46			
Variable	Mean	Std. Deviation	P Value
SBP			
SBP BEFORE	129.67	16.441	<0.05
SBP DURING	129.24	24.904	<0.05
SBP AFTER 5	130.20	17.119	<0.05
SBP AFTER 30	124.02	13.010	<0.05
DBP			
DBP BEFORE	82.13	14.954	<0.05
DBP DURING	81.87	14.531	<0.05
DBP AFTER 5 m	79.00	11.679	<0.05
DBP AFTER 30 m	74.85	13.248	<0.05
HR			
HR BEFORE	80.67	14.092	<0.05
HR DURING	111.33	19.630	<0.05
HR AFTER 5	107.24	15.791	<0.05
HR AFTER 30	94.07	12.840	<0.05
SPO2			
SPO2 BEF	99.09	1.092	<0.05
SPO2 DURING	98.83	1.651	<0.05
SPO2 AFTER 5 m	98.83	1.338	<0.05
SPO2 AFTER 30 m	99.17	1.305	<0.05
RR			
RR BEFORE	12.61	1.000	<0.05
RR DURING	12.67	1.034	<0.05
RR AFTER 5 m	12.76	1.058	<0.05
RR AFTER 30 m	12.80	1.067	<0.05
TEMP			
TEMP BEFORE	37.00	.000a	<0.05
TEMP DURING	37.00	.000a	<0.05
TEMP AFTER 5 m	37.00	.000a	<0.05
TEMP AFTER 30 m	37.00	.000a	<0.05

Table (2) This table shows the mean and standard deviation for parameters of patients who used atropine before the operation, with a statistical significance in all variables, a P value less than 0.05

Table (3): Characteristics of patients who did not used atropine

Characteristics of patients who did not use atropine n = 24		
variable	mean	Std. Deviation
Age	30.21	10.409
Weight	73.88	14.795

Table (3) This table shows the mean and standard deviation for the characteristics of patients who did not received atropine before the operation, with a statistical significance in both variables, a P value less than 0.05

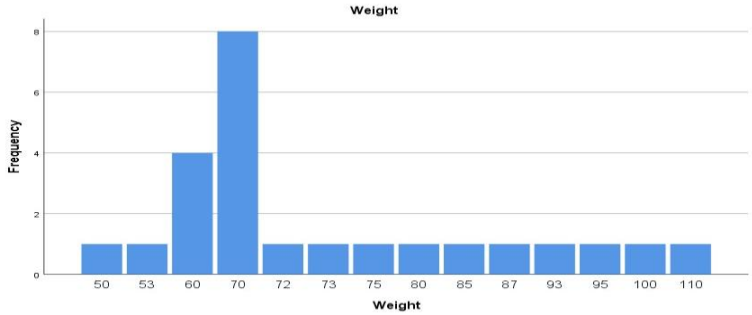

Figure (6): statistical analysis of weight

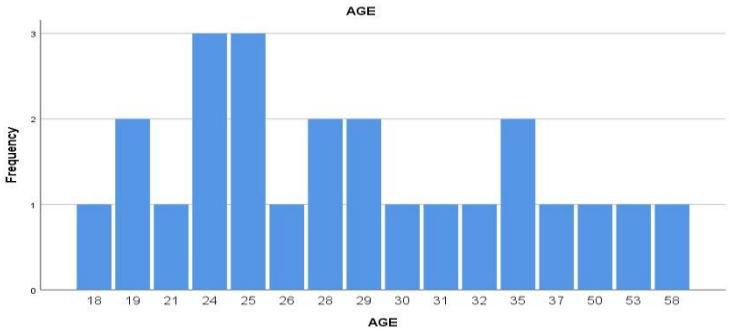

Figure (7): statistical analysis of age

Table (4): parameters of patients who did not used atropine

parameters of patients who did not used atropine N = 24			
Variable	Mean	Std. Deviation	P Value
SBP			
SBP BEFORE	134.42	17.587	<0.05
SBP DURING	131.29	18.940	<0.05
SBP AFTER 5 m	87.92	3.078	>0.05
SBP AFTER 30 m	122.63	14.215	<0.05
DBP			
DBP BEFORE	81.71	10.813	<0.05
DBP DURING	80.42	10.950	<0.05
DBP AFTER 5 m	46.04	3.014	>0.05
DBP AFTER 30 m	77.08	16.490	<0.05
HR			
HR BEFORE	79.88	11.403	<0.05
HR DURING	106.83	18.791	<0.05
HR AFTER 5	49.29	2.866	>0.05
HR AFTER 30	98.96	13.284	<0.05
SPO2			
SPO2 BEFORE	99.21	1.141	<0.05
SPO2 DURING	98.79	1.351	<0.05
SPO2 AFTER 5 m	99.17	.917	<0.05
SPO2 AFTER 30 m	99.00	1.142	<0.05
RR			
RR BEFORE	13.13	1.116	<0.05
RR DURING	12.96	1.122	<0.05
RR AFTER 5 m	12.92	1.100	<0.05
RR AFTER 30 m	12.88	1.116	<0.05
TEMP			
TEMP BEFORE	37.00	.000a	<0.05
TEMP DURING	37.00	.000a	<0.05
TEMP AFTER 5 m	37.00	.000a	<0.05
TEMP AFTER 30m	37.00	.000a	<0.05

Table (4) This table shows the mean and standard deviation for parameters of patients who did not used atropine before the operation, with non-statistical significance in, SBP AFTER 5 min, DBP AFTER 5 min, HR AFTER 5 min. P >0.05

Discussion

Hypotension and bradycardia are the two most common complications of spinal anaesthesia. There are various techniques to prevent these complications which may include fluid loading, vasopressors, leg up positioning, low dose local anaesthetic combined with opioid additives and others. However, none of these techniques are sufficient for proper prevention of hypotension and bradycardia after spinal anaesthesia.

Systemic vasodilation induced by sympathetic blockade, resulting in venous pooling of blood and reduction in systemic vascular resistance, has been regarded as the predominant mechanism for hypotension induced by SA. However, the absence of reflex tachycardia after spinal anaesthesia induced hypotension has been observed to contribute by Hwee H. Lim et al. We postulate that the absence of reflex tachycardia may be an important component in the pathogenesis of hypotension induced by SA in addition to effects of venous and arterial dilation.

The current study showed that there was a statistically significant difference in heart rate and blood pressure, spo2, respiratory rate and temperature groups throughout all the measurements with P values less than 0.001 in men who did receive atropine before the operation. (Table 4.2).

These findings were in line with other studies. A Randomized Controlled Trial done in Nepal in 2015 compared mean heart rate between a group of 20 patients given a single dose of 0.6 mg atropine and a placebo group of equal number. This study showed that the mean heart rate was significantly different between the atropine premedicated and the control groups at all the times they measure.

It was found that there were values that were not statistically significant in the systolic blood pressure variable after 15 minutes, in the diastolic blood pressure variable after 15 minutes, and in the heart rate variable after 15 minutes in men who did not receive atropine before the operation. (Table 4.4).

Another RCT that compares the prophylactic effect of atropine and ephedrine with a placebo group also showed that as compared to the placebo, mean heart rate was significantly increased in atropine and ephedrine groups at all the times. This study also showed that maximum heart rate in atropine group was 89.30 ± 14.62 bpm at 5th minute.

A study done in Chai Wan, Hong Kong to assess the effect of different doses of atropine in prevention of spinal anaesthesia induced hypotension showed that the heart rate

in both small dose (5mcg/Kg) and large dose (10mcg/Kg) groups was significantly different from that of the placebo group.

5. CONCLUSION

Prophylactic atropine within 1 min of induction of spinal anaesthesia in male patients undergoing surgery reduce the incidence of hypotension and bradycardia.

RECOMMENDATION

1.Further randomized clinical trials should be done on a multicentre basis 1.Taking a larger sample in future studies

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