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The Effectiveness of Dexmedetomidine in Patients Receiving Spinal Anesthesia

Eric Heiden

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by

Eric Heiden

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Title: The Effectiveness of Dexmedetomidine in Patients Receiving Spinal Anesthesia.

Background: A failed subarachnoid block due to prolonged surgical duration can be difficult and frustrating issue for anesthesia providers. The overall failure rate for SABs has been reported to be 0.6%, with about one-fourth of all failed SABs attributed to prolonged surgical times. Dexmedetomidine has been shown to extend the duration of subarachnoid block.

Purpose: To conduct an extensive literature review of dexmedetomidine efficacy as an adjunct to prolonging duration subarachnoid anesthesia.

Process: A systematic literature review was conducted using PubMed, CINAHL, Cochrane and Access Medicine for articles that related to the use of intravenous dexmedetomidine during subarachnoid anesthesia. The literature was then integrated to develop evidence-based recommendations for use of intravenous dexmedetomidine in patients receiving subarachnoid anesthesia.

Results: Administration of intravenous dexmedetomidine with or without bolus dose has been shown to be safe and effective at prolonging the duration of subarachnoid anesthesia.

Implications: Anesthesia providers must weigh the risk and benefits when using intravenous dexmedetomidine as an adjunct to subarachnoid anesthesia. Use and dosage of bolus dose should be made on a case-by-case basis due to bradycardia concerns. Dexmedetomidine has the added benefit of providing titratable intraoperative sedation without causing significant respiratory depression.

Keywords: dexmedetomidine, spinal, subarachnoid block

The Effectiveness of Dexmedetomidine in Patients Receiving Spinal Anesthesia.

Spinal anesthesia can be traced back to the late 1800s with the work of James Leonard Corning followed by August Karl Gustav Bier. The basis of these experiments is given to William Halstead and Richard Hall, who had published work demonstrating the utilization of cocaine for local and regional anesthesia**. (**Looseley, 2009, para**.** 3)

Corning first conducted experiments that involved the injection of a cocaine solution into the vertebral spaces of a young dog. He discovered that within five minutes there was a loss of coordination followed by weakness and then eventually anesthesia of the dog's hindquarters that resolved in four hours. Following this discovery, Corning next experimented on a human subject in which he injected a cocaine solution into the T11/T12 interspinous space. With no initial changes he repeated the injection and found that after 10 minutes the patient could feel changes in his legs and there was a significant disruption to the sensitivity of a pinprick and electrical current to the lumbar region down with no motor deficit. Corning did not indicate any finding of cerebrospinal fluid (CSF), so he most likely was injecting into the epidural space. Fourteen years later in 1898, the pioneer of spinal anesthesia Dr. August Karl Bier published the first reports of a subarachnoid block (SAB), which was used successfully and the primary anesthetic for a surgical procedure. Dr. Bier's remarkable breakthrough is similar to the process we use today. (Looseley, 2009, para**.** 3-4)

Spinal anesthesia is a very safe, reliable, and effective anesthesia technique with reliable results that can be used in alone, in conjunction with general anesthesia, epidural and IV anesthetic (Nagelhout & Plaus, 2013; Butterworth, Mackey, Morgan & Wasnick, 2013). The advantages of using neuraxial (spinal and epidural) anesthesia include

decreased nausea, vomiting, urinary retention, reduced opioid need, and greater mental alertness compared to patients who had general anesthesia alone (Nagelhout & Plaus, 2013). There is evidence that neuraxial anesthesia blunts the body's stress response to surgery; provides preemptive anesthesia; and decreases intraoperative blood loss, postoperative thromboembolic events and ileus. Additionally, spinal anesthesia increases patency of vascular grafts, and improves respiratory function and cardiac stability, and improves outcomes in high-risk patients (Nagelhout & Plaus, 2013; Butterworth et al., 2013). The subarachnoid block (SAB) is commonly used to provide anesthesia for operations below the umbilicus such as orthopedic procedures involving the hips and below, caesarean delivery, endovascular aortic aneurysm repair, urological procedures, hernia repair, and some gynecological procedures (Schug, Saunders, Kurowski, & Paech, 2006).

One disadvantage of a SAB is the inability to extend the duration of the anesthetic intraoperatively due to the needs of a prolonged surgical procedure (Johnson & Pugh, 2016). The overall failure rate for SABs has been reported to be 0.6%, with approximately one-fourth of all failed SABs attributed to prolonged surgical times (Guglielmo, Pignataro, Difiore, Lanza, & Mercadante, 2010). Research has shown that dexmedetomidine can extend the duration of the SAB (Johnson & Pugh, 2016). Patients who receive a SAB most often desire sedation, which dexmedetomidine can provide in conjunction with extending the duration of the block (Johnson & Pugh, 2016).

Purpose

A major disadvantage of spinal anesthesia is unexpected prolongation of the surgical procedure. The purpose of the literature review was to determine if

dexmedetomidine was viable adjunct to spinal anesthesia and will it prolong the spinal block. Intravenous dexmedetomidine is a possible pharmacological adjunct that could be added to allow coverage for the extended procedure. The risk and benefits of adding intravenous dexmedetomidine to the anesthesia plan must be addressed to each individual patient. Use and dosage of bolus dose should be made on a case-by-case basis due to bradycardia concerns. Dexmedetomidine has the added benefit of providing titratable intraoperative sedation without causing significant respiratory depression over propofol.

Case Report

A 64-year-old, 90 kilogram (kg), 175 centimeter (cm) male presented for a right total hip arthroplasty (RTH) due to a previous injury. Surgical history includes a tonsillectomy as a young child. No prior issues or concerns with anesthesia were reported. The patient's medication regime included omeprazole for gastroesophageal reflux disease, acetaminophen as for right hip pain, and trazadone for sleep daily. No known drug allergies.

A pre-operative airway evaluation revealed a Mallampati classification II, thyromental distance of greater than three fingerbreadths, intact dentition, and full neck range of motion. Preoperative vital signs were heart rate (HR) 84/min, blood pressure (BP) 136/84 mmHg, respiratory rate (RR) 16/min, pulse oximetry (SpO2) 98% on room air, and temperature (T) 97.9 Fahrenheit. Physical exam found clear lung sounds, S1 and S2 heart sounds without murmur, and bilateral extremity pulses present and equal. The patient was considered an American Society of Anesthesiologists (ASA) physical status level two. In the preoperative area, an 18 gauge peripheral intravenous (IV) catheter was

inserted into the patient's left forearm. No medications were given prior to being transferred to operating room (OR).

Upon entering the OR suite, the patient transferred himself onto operating room table, standard monitors were simultaneously applied which included: a finger pulse oximeter, non-invasive blood pressure cuff on his right upper arm, and a 5-lead electrocardiogram (EKG). Oxygen was administered at 2 liters per minute (LPM) via an end-tidal carbon dioxide (ETCO2) sampling nasal cannula. Initial vital signs were HR 91/min, BP 141/90 mmHg, RR 18/min, SpO2 97% on 2 LPM nasal cannula. He was given midazolam 2 milligrams (mg) IV and fentanyl 100 micrograms (mcg) IV due to pain in his right hip and anxiety regarding the spinal placement. He was assisted to sitting position on the side of the OR table with a chair at the side of the bed for him to rest his feet. The circulating was nurse standing in front of the patient to assist with positioning for spinal placement. The lumbar area was prepped in a sterile fashion, a 2 mL 1% Lidocaine skin wheel was performed at the L3-4 region. A 25 gauge 3 ¾ inch pencil tip spinal needle was inserted midline in the lumbar 3-4 interspace. Correct placement of the SAB was confirmed with CSF presence; 1.7 milliliters (mL) of 0.75% bupivacaine (in 8.25% dextrose) with 0.2 mg preservative-free morphine was injected. Once the SAB was completed, the patient was placed in the supine position and sensory loss was assessed to dermatome level T6-T7 via sharp pinprick. The patients systolic BP decreased from 140s to 110s and remained constant throughout the procedure.

Due to the patient exhibiting airway obstruction after his premedication of midazolam 2 mg IV and fentanyl 100 mcg IV, dexmedetomidine was selected for IV sedation. Dexmedetomidine was chosen due to its ability to provide sedation without

respiratory depression. Oxygen was titrated up to 4 LPM due to the SpO2 decreasing from 97% to 93%. A dexmedetomidine bolus was initiated at 0.5 mcg/kg over 10 min followed by maintenance infusion of 0.5 mcg/kg/hr. A propofol bolus of 20 mg IV was given before incision as the dexmedetomidine bolus had not yet infused; the patient was exhibiting signs of anxiety. A single dose of glycopyrrolate 0.2 mg IV was administered to counteract bradycardia of 50-55/min, thought to be caused by the dexmedetomidine bolus and infusion. HR returned to 60-65/min within 5 minutes and remained stable for the duration of the case. During the procedure, the patient required one additional dose of propofol 20 mg IV for sedation. The remainder of the case was uneventful. The patient remained hemodynamically stable and was able to maintain his airway throughout the three hour and twenty minute surgical procedure with oxygen saturations greater than 94% on 4 LPM nasal cannula.

Near the conclusion of surgery and beginning of incision closure, the dexmedetomidine infusion was discontinued, and ondansetron 4 mg IV was given for post-operative nausea and vomiting (PONV) prophylaxis. Prior to the patient being transferred to the post-anesthesia care unit (PACU) he was able to open his eyes and follow commands appropriately.

Upon arrival to PACU, vitals signs were within normal limits, the patient was following commands, answering questions, and denied any presence of pain or nausea. Oxygen was titrated to 2 LPM with oxygen saturation at 99%.

Literature Search

Search Strategy

The comprehensive review of literature was conducted through the University of North Dakota (UND) Harley E. French Library of Health Sciences online library website. Articles were obtained by the Cumulative Index to Nursing and Allied Health (CINAHL), PubMed, Access Medicine, Cochrane databases and the American Association of Nurse Anesthetists (AANA) journal. Key words applied to conduct this search included: dexmedetomidine, spinal, subarachnoid block.

Research began with a search in CINAHL. It is an easy to use database that utilizes journals from the nursing field and the 17 allied health disciplines with around 2700 journals. The first search term used was "dexmedetomidine" which returned 93 articles. The search was limited the search to English language and newer than 2012, which returned 22. Of the 22 articles, 2 were relevant to the PICO question. My second search was "spinal", returning 4 articles. Between the 2 searches a total of 3 articles were found to be relevant to the PICO question.

The next database used was PubMed. PubMed is a service of the United States National Library of Medicine. It provides journal articles from a broader array of specialties such as: medical, nursing, dental, health care and preclinical sciences. This broader reach of specialties offers a greater likelihood of finding relevant articles. The initial search of "dexmedetomidine " returned 372 and with a 3-year limitation it was cut down to 143. The search term "spinal" was added, which weaned the total to 28. There were 3 articles that directly addressed the specific topic of interest. From the references I searched the DOI numbers and found the articles on PubMed. The snowball continued from there by continuing to look up the resources from the original paper and then the subsequent papers from there. This search also led to the AANA journal article with a

applicable topic. The reference section was again used to identify relevant articles. In all, 19 articles were printed for possible use in the paper.

Review of Literature

Administration of Spinal Anesthesia

The majority of subarachnoid blocks are placed in either the sitting or lateral position on the OR table (Schwartz & Brunicardi, 2015). Positioning is dependent on the patient's ability to follow commands, maintain the position, pain level, desired dermatome coverage, as well as type of OR table being used (Schwartz & Brunicardi, 2015). Before the SAB the patient is connected to the BP, EKG, and SpO2 monitors and O2 is administered via nasal cannula. Premedication for the spinal procedure is at the discretion of the anesthetist and is most often dependent on comorbidities and anxiety level of the patient (Schwartz & Brunicardi, 2015). The patient's back is cleansed with an antiseptic solution, skin is infiltrated with local anesthetic, a 25-27 gauge spinal needle is introduced in the lower lumbar region, and the subdural space is identified by the presence of CSF (Sanford, 2014). The local anesthetic is injected directly into the dural sac that surrounds the spinal cord (Sanford, 2014). The level of injection is usually below L1 to L2, where the spinal cord ends in most adults (Nagelhout & Plaus, 2013). Local anesthetic and addition of opioid are chosen based on the length of surgery and surgical procedure. Once the local anesthetic is injected, the patient is placed in the supine position for 5-10 minutes to allow for the proper spread of the local anesthetic (Sanford, 2014). During this period, hypotension and bradycardia can be induced by a sympathectomy due to the cephalad spread of the local anesthetic (Sanford, 2014).

Patient positioning should be maintained to allow the anesthetic block to set up properly. Once the block has stabilized, the surgical preparation and positioning can proceed (Sanford, 2014).

Mechanism of Action

The exact mechanism of action for subarachnoid anesthesia continues to contain much speculation. The primary site of action for the blockade is the nerve roots within the spinal cord (Nagelhout & Plaus, 2013). A local anesthetic is injected into the CSF, distributing through the subarachnoid space. The spread of local anesthetic is based on the positioning of the patient, physical/chemical properties of the drug, and characteristics of the space in which it is to spread (Nagelhout & Plaus, 2013). When drug concentrations reach a minimally effective concentration at the nerve root, neuronal transmission is altered in a clinically significant way, which provides anesthesia (Nagelhout & Plaus, 2013). "Blockade of neural transmission in the posterior nerve root fibers interrupts somatic and visceral sensation, whereas blockade of anterior nerve root fibers interrupts efferent motor and autonomic outflow" (Nagelhout & Plaus, 2013, p. 1073). The nerve size, length, myelination and distance from injection are all important factors in the susceptibility of the nerve to the local anesthetic (Nagelhout & Plaus, 2013; Butterworth et al., 2013).

Local anesthetics work by reversibly binding to the voltage-gated sodium channels, preventing channel activation and inhibiting the sodium influx associated with membrane depolarization (Butterworth et al., 2013). This process alters nerve transmission with increasing drug concentrations until action potential is unable to be generated and impulses transmitted (Butterworth et al., 2013).

Dexmedetomidine

Mechanism of action Dexmedetomidine is a selective alpha-2 adrenergic agonist that functions by stimulating the receptors, resulting in a decrease of catecholamine release and sympathetic nervous system (SNS) response (Eilers & Yost, 2015). Dexmedetomidine's primary effects are sedation with minimal respiration depression, anxiolysis, reduced postoperative shivering and agitation, and cardiovascular sympatholytic actions (Nagelhout & Plaus, 2013). This drug profile makes it a useful adjunct for sedation with spinal anesthesia.

The highly selective alpha-2 agonist with spinal anti-nociceptive (visceral and somatic) properties produces a synergistic effect with intraspinal local anesthetics (Kalso, Pöyhiä, & Rosenburg, 1991). The reduced noradrenergic outflow is thought to strengthen the inhibitory nociceptive effect on the spinal cord (Samuels & Szabadi 2008). The higher alpha-2 receptor selectivity for IV dexmedetomidine reduces the severity of hemodynamic instability (bradycardia and hypotension) and is capable of extending the duration of a SAB longer than clonidine (Abdallah, Abrishami, & Brull, 2013).

Dosage Standard adult dosing is 0.5-1 mcg/kg IV bolus infused over 10 minutes followed by a maintenance infusion of 0.2-0.7 mcg/kg/hr (Nagelhout & Plaus, 2013).

Side effects Dexmedetomidine has been shown to exhibit moderate decreases in heart rate and systemic vascular resistance, which leads to a decrease in systemic blood pressure (Eilers & Yost, 2015). A bolus injection may produce a transient increase in systemic blood pressure and pronounced decrease in heart rate (Eilers & Yost, 2015). Bradycardia associated with dexmedetomidine infusion may require treatment (Eilers &

Yost, 2015). Heart block, severe bradycardia, and asystole have been observed and may result from unopposed vagal stimulation (Eilers & Yost, 2015).

Pharmacokinetics Dexmedetomidine has a rapid onset with a loading infusion of approximately 10-20 minutes and duration of action post infusion of 10-30 minutes (Nagelhout & Plaus, 2013). The elimination half-life is 2 hours (Nagelhout & Plaus, 2013). Dexmedetomidine shows linear kinetics in the dosage range of 0.2 – 0.7 mcg/kg/hr (Nagelhout & Plaus, 2013). It is highly protein bound at 94%, rapidly metabolized by the liver, and excreted in urine and bile. Patients with severe liver dysfunction or impaired kidney function may need a dose adjustment (Nagelhout & Plaus, 2013).

Dexmedetomidine as Adjunct in Surgery with Spinal Anesthesia

A supraspinal mechanism of action of IV dexmedetomidine has not been clearly defined. It is thought that when administered IV alpha-2 agonists inhibit the activity of the locus coeruleus in the brain, leading to noradrenergic nuclei (Johnson & Pugh, 2016). The decreased noradrenergic outflow is believed to strengthen the inhibitory nociceptive effect on the spinal cord (Samuels & Szabadi 2008).

The two alpha-2 agonists commonly used in practice are dexmedetomidine and clonidine. Dexmedetomidine has a higher affinity for the alpha-2 receptor versus alpha-1 when compared to clonidine, reducing the bradycardia and hypotension (Schug et al., 2006).

According to a systematic review with meta-analysis by Abdullah, Abrishami, and Brull (2013), IV dexmedetomidine may be an effective spinal adjunct for prolonging

the duration of regional blockade, sedation, and postoperative analgesia in patients receiving SAB. This systematic review contained seven randomized controlled trials (RCTs) examining 364 subjects distributed evenly between placebo and IV dexmedetomidine treatment groups. The outcome of primary focus was the duration of motor and sensory block, postoperative analgesia, and adverse-related effects such as hypotension, bradycardia, respiratory depression, and postoperative sedation (Abdallah, et al., 2013).

The results of the Abdallah et al. (2013) review suggests that IV dexmedetomidine in conjunction with spinal anesthesia can prolong the duration of sensory block and to a lesser extent motor block. It was also found that IV dexmedetomidine might delay the time to first analgesic request after spinal anesthesia. With these effects there is a heightened risk of transient reversible bradycardia. The study researched another alpha-2 agonist clonidine, and their findings suggest IV dexmedetomidine produces a greater differential blockade by preferentially targeting sensory myelinated alpha fibers over unmyelinated C fibers involved in motor conduction. Variations among spinal and dexmedetomidine dosing make it difficult to translate into clinical practice guidelines (Abdallah, et al., 2013). Dexmedetomidine bolus doses varied from 0.5-1.0 mcg/kg with and without maintenance rates of 0.2-0.5 mcg/kg over varying durations (Johnson & Pugh, 2016). It was noted that isolated boluses of 0.5 mcg/kg administered as isolated boluses significantly prolonged the anesthetic effect of the SAB (Johnson & Pugh, 2016). Dosing of dexmedetomidine throughout the studies was highly variable and there was a lack of standardization of assessment means for sensory and motor block duration (Johnson & Pugh, 2016). Suggestions were made to

study optimal dosing of dexmedetomidine and formulate standardization of evaluation techniques (Abdallah, et al., 2013).

In 2016, the American Association of Nurse Anesthetists (AANA) Journal conducted and published an evidence-based review, which updates the Abdallah et al. 2013 systematic review. Initially, 79 sources were narrowed to eight RCTs based on inclusion and exclusion criteria. Within the RCTs there were 480 subjects. The RCTs looked at the outcomes of IV dexmedetomidine administered in conjunction with a SAB. Outcome measures included motor and sensory blockade, postoperative analgesia, and the side effects of bradycardia and hypotension. Several limitations were noted within the RCT, such as three studies failed to describe blinding methods, twos studies lacked power analysis to determine sample size for outcomes measured and one study had an underpowered base due to subject withdrawal (Johnson & Pugh, 2016).

Dexmedetomidine was dosed as either a bolus only or a bolus with maintenance infusion (Johnson & Pugh, 2016). Among the studies, there was a variation in the start time of the dexmedetomidine infusion (Johnson & Pugh, 2016). Two of the RCTs neglected to administer a preload IV fluid bolus before the SAB (Johnson & Pugh, 2016). Bupivacaine was the local anesthetic of choice, but the dose and concentration were not disclosed in all the studies (Johnson & Pugh, 2016). The surgical procedures varied from pelvic, lower extremity, to urological. A 2-dermatome regression (TDR) of at least 20 minutes was utilized in all eight studies to evaluate sensory block duration (Johnson & Pugh, 2016). The Modified Bromage Scale (MBS) 0-3 scale was used to measure motor block duration in six studies with a MBS of 0-6 in another (Johnson & Pugh, 2016). Another study only used knee flexion to measure motor recovery (Johnson & Pugh,

2016). The MBS scores recorded endpoints for motor recovery ranging from 0-1 and 4 (Johnson & Pugh, 2016). The observer variability, subjective measurements, patient participation, and IV dexmedetomidine sedation may have an impact on the validity and reliability of these findings (Johnson & Pugh, 2016). Four studies evaluated dexmedetomidine IV with SAB's effect on postoperative analgesia, but there were a wide variety of assessment tools used hindering the ability to find correlations (Johnson & Pugh, 2016). Observers noted differences with highest initial maximum sensory level achieved by SAB (in those that reported this finding) but found that dexmedetomidine administration before the SAB had no influence on the initial spread of spinal local anesthetic (Johnson & Pugh, 2016). The differences in highest initial maximum sensory level could be due to many different factors including height, weight, local anesthetic dose and baricity, and positioning (Johnson & Pugh, 2016). The impact was determined to be that IV dexmedetomidine prolonged the maximal duration with little effect on the initial level of sensory blockade (Johnson & Pugh, 2016).

Pain Pathway

The primary purpose of subarachnoid anesthesia is to block transmission of sensory, motor, and pain during surgical intervention (Nagelhout & Plaus, 2013). Disrupting the afferent and efferent pathways is accomplished by administration of subarachnoid local anesthetics (Nagelhout & Plaus, 2013). Depending on the area of surgical interest, a certain level dermatome level of sensory blockade is desired (Nagelhout & Plaus, 2013). Spinal nerves provide a particular area of cutaneous innervation that is anatomically mapped out in dermatome levels (Nagelhout & Plaus,

2013). A dermatome is the defined area that is innervated by the specific spinal nerves (Nagelhout & Plaus, 2013).

According to Nagelhout and Plaus (2013), "Pain and temperature receptors in the skin of the trunk and extremities send signals to the spinal cord via the dorsal roots of the spinal nerves" (p. 696). The dorsal root sensory fibers terminate in the dorsal horn before crossing over to lateral fibers (Nagelhout & Plaus, 2013). The sensory information then ascends via the anterior spinothalamic and lateral spinothalamic tracts (Nagelhout & Plaus, 2013). The dorsal column-medial lemniscal tract also carries afferent sensory information with a greater degree of localization (Nagelhout & Plaus, 2013).

Motor pathways follow a descending ventral pathway that originates in the corticospinal tract (Nagelhout & Plaus, 2013). A majority of neurons cross over to form the lateral corticospinal tract, where they continue their descent (Nagelhout & Plaus, 2013). At each spinal cord level, fibers leave the lateral corticospinal tract to the ventral horn gray matter where they synapse with lower motor neurons (Nagelhout & Plaus, 2013). The neurons that do not cross descend via the ventral corticospinal tract (Nagelhout & Plaus, 2013). These neurons cross over before synapsing with lower motor neurons within the gray matter(Nagelhout & Plaus, 2013). The axons from the lower motor neurons travel in the spinal nerves to innervate voluntary muscle (Nagelhout & Plaus, 2013). Local anesthetic from a SAB interrupts the pain pathways by altering the ability of spinal nerves to generate and transmit impulses.

Conclusion

A significant disadvantage of subarachnoid anesthesia is the inability to extend the duration of the anesthetic intraoperatively to address the needs of prolonged surgical procedures (Johnson & Pugh, 2016). There are several methods available to solve this problem that include using intrathecal adjuncts, combined spinal-epidural techniques, and higher subarachnoid local anesthetic doses. Intravenous dexmedetomidine offers a simple, effective method for prolonging the duration of motor and sensory blockade and postoperative analgesia with a minimal side effect profile (Johnson & Pugh, 2016). Utilization of IV dexmedetomidine as primary subarachnoid block adjunct must be evaluated on a case by case basis (Johnson & Pugh, 2016).

Intravenous dexmedetomidine has the additional benefit of providing sedation during the procedure, which is often desired by most individuals receiving subarachnoid blocks (Johnson & Pugh, 2016). Anesthesia providers should be cognizant of the undesirable side effects associated with IV dexmedetomidine, as well as the management of adverse reactions. Bradycardia is a common side effect related to IV dexmedetomidine, which may be detrimental to patient depending on comorbidities. Attachment of standard monitors and frequent monitoring should be utilized to evaluate the tolerance of bradycardia in each patient. Bradycardia that is not tolerated should be treated immediately. Intravenous dexmedetomidine does not cause major respiratory depression while providing a safe, titratable level of intraoperative sedation (Venn, Hell, & Grounds, 2000). Intravenous dexmedetomidine has shown to safely and effectively prolong a subarachnoid block, therefore anesthesia providers should consider the use of IV dexmedetomidine in surgical procedures that may be unexpectedly extended.

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The Effectiveness of Dexmedetomidine in Patients Receiving Spinal Anesthesia

Eric Heiden, SRNA

UND Nurse Anesthesia
University of North Dakota

Introduction

- Spinal anesthesia is a common anesthesia technique used with reliable results within the operating room.
  - A failed subarachnoid block due to prolonged surgical duration can be difficult and frustrating issue for anesthesia providers.
  - The overall failure rate for Subarachnoid blocks has been reported to be 0.6%, with about one-fourth of all failed SABs attributed to prolonged surgical times.
  - One disadvantage of a SAB is the inability to extend the duration of the anesthetic intraoperatively due to the needs of a prolonged surgical procedure

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Case Information

- Right Total Hip Arthroplasty
- 64 year old
- Weight – 90 kg
- Gender - Male
- ASA 2
- No known allergies

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Pre-operative Evaluation

- Medical History
  - Gastroesophageal reflux, insomnia
- Surgical History
  - Tonsillectomy as a young child
- Pre-op VS
  - BP 136/84, HR 84, RR 16 , SpO2 98%
- Airway evaluation
  - Mallampati II, TMD > 3 fingerbreadths, full neck range of motion, intact dentition

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Anesthetic Course

- Preoperative sedation
  - 100 mcg Fentanyl
  - 2 mg Versed
- Technique
  - Spinal anesthesia with 22g Pencan Needle
  - 1.7 ml Bupivacaine 0.75%
  - 0.2 mg Morphine
- EtCO2 NC with O2 at @ 2 LPM
- Maintenance
  - Dexmedetomidine bolus 0.5 mg/kg/hr followed by maintenance at 0.5 mg/kg/hr
  - Tranexamic Acid
  - Ondansetron 4 mg

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Intraoperative Issues

- Total Anesthesia Time
  - 3 hrs 20 min
- Estimated Blood Loss
  - 500 ml
- Complications
  - Airway obstruction (improved with positioning and chose dexmedetomidine over propofol for sedation)
  - Bradycardia (glycopyrrolate 0.2 mg)

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Postoperative

- Dexmedetomidine discontinued at incision closure.
- Patient following commands and denied pain in PACU.
- Vital signs within normal limits.

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Spinal Anesthesia

- Premedication for the spinal procedure is at the discretion of the anesthetist and is most often dependent on comorbidities and anxiety level of the patient.
- Local anesthetic and addition of opioid are chosen based on the length of surgery and surgical procedure.
- The exact mechanism of action for subarachnoid anesthesia continues to contain much speculation.

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Spinal Anesthesia

- The primary site of action for the blockade is the nerve roots within the spinal cord.
- The spread of local anesthetic is based on the positioning of the patient, physical/chemical properties of the drug, and characteristics of the space in which it is to spread.
- When drug concentrations reach a minimally effective concentration at the nerve root, neuronal transmission is altered in a clinically significant way, which provides anesthesia

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Spinal Anesthesia

- Blockade of neural transmission in the posterior nerve root fibers interrupts somatic and visceral sensation, whereas blockade of anterior nerve root fibers interrupts efferent motor and autonomic outflow.

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Dexmedetomidine

- A selective alpha-2 adrenergic agonist that functions by stimulating the receptors, resulting in a decrease of catecholamine release and sympathetic nervous system (SNS) response.
- Dexmedetomidine's primary effects are sedation with minimal respiration depression, anxiolysis, reduced postoperative shivering and agitation, and cardiovascular sympatholytic actions.

(Eilers & Yost, 2015; Nagelhout & Plaus,)

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Dexmedetomidine and Spinal Anesthesia

- The highly selective alpha-2 agonist with spinal anti-nociceptive (visceral and somatic) properties produces a synergistic effect with intraspinal local anesthetics. (Kalso, Pöyhiä, & Rosenburg, 1991).
- The reduced noradrenergic outflow is thought to strengthen the inhibitory nociceptive effect on the spinal cord. (Samuels & Szabadi 2008).
- The higher alpha-2 receptor selectivity for IV dexmedetomidine reduces the severity of hemodynamic instability (bradycardia and hypotension) and is capable of extending the duration of a SAB longer than clonidine. (Abdallah, Abrishami, & Brull, 2013)

(Kalso, Pöyhiä, & Rosenburg, 1991; Samuels & Szabadi, 2008; Abdallah, Abrishami & Brull, 2013)

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Dexmedetomidine

- Dosage
  - Standard adult dosing is 0.5-1 mcg/kg IV bolus infused over 10 minutes followed by a maintenance infusion of 0.2-0.7 mcg/kg/hr.
- Side Effects
  - Dexmedetomidine has been shown to exhibit moderate decreases in heart rate and systemic vascular resistance, which leads to a decrease in systemic blood pressure.
  - A bolus injection may produce a transient increase in systemic blood pressure and pronounced decrease in heart rate.
  - Bradycardia associated with dexmedetomidine infusion may require treatment.
  - Heart block, severe bradycardia, and asystole have been observed and may result from unopposed vagal stimulation

(Nageihout & Pleus, 2013)

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Dexmedetomidine and Spinals

- A supraspinal mechanism of action of IV dexmedetomidine has not been clearly defined.
- It is thought that when administered IV alpha-2 agonists inhibit the activity of the locus coeruleus in the brain, leading to noradrenergic nuclei (Johnson & Pugh, 2016).
- The decreased noradrenergic outflow is believed to strengthen the inhibitory nociceptive effect on the spinal cord (Samuels & Szabadi 2008).

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Studies

- A significant disadvantage of subarachnoid anesthesia is the inability to extend the duration of the anesthetic intraoperatively to address the needs of prolonged surgical procedures.
- The results of the Abdallah et al. (2013) review suggests that IV dexmedetomidine in conjunction with spinal anesthesia can prolong the duration of sensory block and to a lesser extent motor block. It was also found that IV dexmedetomidine might delay the time to first analgesic request after spinal anesthesia.

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Studies

- In 2016, the American Association of Nurse Anesthetists (AANA) Journal conducted and published an evidence-based review, which updates the Abdallah et al. 2013 systematic review.
- The impact was determined to be that IV dexmedetomidine prolonged the maximal duration with little effect on the initial level of sensory blockade.

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Discussion

- There are several methods available as adjuncts to standard one shot spinal alone that include using intrathecal adjuncts, combined spinal-epidural techniques, and higher subarachnoid local anesthetic doses. Intravenous dexmedetomidine offers a simple, effective method for prolonging the duration of motor and sensory blockade and postoperative analgesia with a minimal side effect profile

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Recommendations

- If procedure is thought to be long, consider using Dexmedetomidine IV infusion.
- Bolus dose isn't necessary, but 0.5 mcg/kg/min is recommended over 1 mcg/kg/min.
- Maintenance infusion at 0.5/mcg/kg/min.
- Study of cost analysis of propofol vs Dexmedetomidine.
- Better comparison of local anesthetics.

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Conclusion

- Prolonged surgical duration is difficult to address with spinal anesthesia.
- Intravenous dexmedetomidine offers a simple, effective method for sedation, prolonging the duration of motor and sensory blockade and postoperative analgesia with a minimal side effect profile.

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Thank You
Are There Any Questions?

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