Transient Neurologic Symptoms: Lidocaine Hurting for Attention

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Citation

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Abstract

The aging population is expected to impact ambulatory surgery upward by 53% by the year 2020. In the past, lidocaines popularity in neuraxial anesthetics for short procedures allowed for a quick recovery and discharge. However, the growing number of outpatient procedures combined with rapid discharge criteria has brought attention to lidocaine intrathecal (IT) regional anesthesia and concern over developing Transient Neurologic Symptoms (TNS). A literature search was performed for available research on intrathecal administration of lidocaine with a focal measurement on TNS as a complication. Additionally, comparison of lidocaine to other local anesthetics, etiological factors for developing TNS, and pain association are gathered and reported. The role of TNS on future IT lidocaine use for outpatient procedures is contemplated and special patient populations that may be protected from developing TNS are discussed. Anesthesia providers should base their continued usage of intrathecal lidocaine on evidence based practice.

INTRODUCTION

According to the American Association of Ambulatory Surgery Centers (AAASC), more than 80% of surgeries were performed on an ambulatory basis in 2006. [1] The demand on outpatient and ambulatory centers is expected to increase as the baby boomer era ages. A 2003 study performed by Etzioni et al. [2] forecasts a 14-47% growth in the surgical work market by 2020 due to a 53% increase in the 65+ age group. Additionally, the 2006 proposal by the Center for Medicare and Medicaid Services (CMS) calls for an additional 793 surgical procedures to be supplemented as outpatient or ambulatory status. [3] Patient preferences, advances in technology and medical care, evolving patient demographics, Medicare changes and economics are driving forces for ambulatory surgery centers to accommodate the growing surgical needs of society. The transition from inpatient to outpatient surgery coupled with age related co-morbidities in the older patient population places considerable challenges on the anesthesia provider to deliver safe, effective anesthesia within the time constraint design of same day surgical facilities.

Ambulatory care and out patient surgical facilities define their success by surgical turnover, cost effective care and patient satisfaction. Ambulatory anesthesia, therefore, is directed toward early discharge and successful outcomes in anesthesia and analgesia, resulting in overall patient satisfaction with their anesthetic experience. Pain, postoperative nausea and vomiting (PONV) are major contributors in delayed discharge from ambulatory and same day surgical facilities, resulting in additional cost and negatively impacting patient satisfaction with their perioperative experience. [4,5] Compared to general anesthesia, regional anesthesia has been shown to significantly decrease pain, PONV, and the overall total costs of anesthesia and recovery; representing a savings of possibly up to 30%. [6,7,8] Subarachnoid block (SAB) can offer excellent pain control and decrease PONV. However, it may prolong recovery and delay discharge in ambulatory surgery due to a prolonged motor blockade and sympathetic residual effects of the local anesthetic (LA). In order to effectively use regional anesthesia for the same day surgery patient, a short acting LA needs to be utilized. The LA choice is determined by patient co-morbidities, surgical or procedural length and the intensity of blockade required.

Since the 1940's, lidocaine (xylocaine) has been utilized and famed for its fast-onset and short-duration characteristics in subarachnoid approaches. [9] These characteristics have contributed to a world–wide popular use of intrathecal lidocaine in short surgical procedures accommodating for a quick recovery. Interestingly, toxicological reports on intrathecal local administration did not appear in literature

for more than 15 years after lidocaine and other LA were routinely utilized for subarachnoid blocks. Dripps and Vandam [10] in a prospective study of more than 10,000 patients that received spinal anesthesia report less than 0.01% incidence of neurologic involvement after SAB. Supporting those findings, another prospective study of an almost equal subject number, by Phillips et al. [11] report an even fewer incidence of neurologic sequelae; most of which were transient in nature. These studies suggested lidocaine was safely administered to patients for intrathecal regional anesthesia. A safe track record and rapid regression of a local anesthetic are economically appealing to ambulatory centers because it promotes early discharge in ambulatory surgery and promotes patient satisfaction. For decades, lidocaine subsequently took top rank as the local anesthetic of choice in short surgical procedures requiring spinal anesthesia owing to its exclusive pharmacologic characteristics. Lidocaine use expanded as outpatient procedures grew in number. The increased utilization was mainly secondary to its pharmacological effects that resulted in improved pain control and a decreased incidence of PONV. The benefits of rapid motor and sensory regression after spinal anesthesia expedited patient discharge, improved patient satisfaction, and met cost reduction goals.

Complications associated with intrathecal lidocaine emerged in the early 1990's from case reports and early clinical trials implicating 5.0% lidocaine as being potentially neurotoxic. [12] In 1992 spinal microcatheters were withdrawn from Food and Drug Administration (FDA) approval after several episodes of cauda equina syndrome were reported, mostly involving continuous infusions of 5.0% lidocaine. [13,14] Retrospective evaluation of the catheter withdrawal has suggested a possible neurotoxicity or overdose of lidocaine on injection since the catheters were administered multiple times in those cases which led to permanent neurologic damage. Nonetheless, several case reports of transient neuropathy continued to emerge after single-shot intrathecal injections of 5.0% lidocaine. [12] The cases were limited in number and no correlation could be made between spinal needle type or size, technique, patient positioning or cord ischemia, leading to the assumption that a direct neurotoxic effect occurred from pooling of highly concentrated 5.0% lidocaine at the sacral one level. The neuropathic symptoms, labeled Transient Neurologic Symptoms (TNS), involve a characteristic burning pain and dysesthesia in the buttocks radiating to the dorsal thighs and calves after full recovery from spinal anesthesia. [15] These temporary symptoms usually present within 24 hours following full recovery from the local anesthetic and can possibly last up to 7-10 days; resolution may be as soon as 72 hours. [12] Subarachnoid block with lidocaine and the associated risk of TNS raises concern for the safety of future lidocaine use. Growth in the number of outpatient surgeries requires a local anesthetic that has the capacity for a rapid recovery. Undoubtedly, intrathecal lidocaine use will continue to expand to meet outpatient needs. A responsibility exists to consider whether lidocaine use is the safest choice for intrathecal anesthesia if the incidence of TNS will also increase. Recent studies have focused on the prevalence of TNS following intrathecal lidocaine as compared to other local anesthetics, prevailing risk factors, the etiology of TNS, and the impact of TNS on the surgical patient.

This paper will review lidocaine use in ambulatory and outpatient surgery from a historical perspective, define TNS and review the incidence of TNS associated with intrathecal lidocaine administration. A literature review will discern incidence of developing TNS while relaying comparative data of risk factors for developing TNS with intrathecal lidocaine. Surgical trends and outpatient needs will be considered while evaluating the impact of TNS on post spinal recovery. Additionally, etiological inference of TNS and possible protective physiology will be reviewed so anesthesia providers can consequently determine their patient's anesthetic choice based on current research.

TRANSIENT NEUROLOGIC SYMPTOM INCIDENCE

Following the diagnosis of cauda equina syndrome and the case reports of TNS, the study perspective of new onset neuropathies after intrathecal local anesthesia became more focused on risk, etiological and incidental factors. Treatment modality studies are absent or usually inclusive of other related studies likely due to transient neurologic symptoms brief nature and minimal need for aggressive therapy.

Local anesthetics have been associated with a low incidence of neurological sequelae when administered intrathecally (IT). Historical data initially reported radiculopathy from intrathecal local anesthesia administration as 1 per 10,000. [10,11] After several reports of cauda equina syndrome with 5.0% lidocaine through continuous spinal microcatheters, the concern over neurotoxicity resulted in Food and Drug Administration withdrawal of catheters from the market. [13] Rigler and colleagues associated the catheters as a potential cause of pooling and resultant maldistribution of hyperbaric 5.0% lidocaine after catheter injection. [13] In 1993, Schneider

et al [12] reported four patients that developed radicular pain after total recovery from spinal anesthesia with 5.0% hyperbaric lidocaine during gynecological procedures. [12] The symptoms, later labeled as TNS, involve buttock pain or dysesthesia that radiates to the unilateral or bilateral dorsal thighs that has increased intensity at night. The incidence of transient neurologic symptom is variable. Occurrence may be as high as 37% and appears to be greatest associated with lidocaine, though it occurs after other intrathecal local anesthetics. [15,6,17] Freedman et al. [16] performed a fourteen-month large scale epidemiological study at fifteen medical centers. Findings suggested a profound risk for developing TNS in patients having received intrathecal lidocaine compared to those receiving bupivicaine or tetracaine. Without any attempt to modify practice, anesthesia providers were asked to complete a data sheet on all patients receiving intrathecal analgesia. A random selection of 8,400 patients resulted in 1,883 interviewed. After performing adjustments for age, sex, race, body mass index, preexisting neurologic conditions, surgical type, and inpatient or outpatient status, the authors found an incidence of TNS after intrathecal administration of 1.5%-5% lidocaine. Comparitively, lidocaine had a relative risk (RR) of 5.1 and 3.2 respectively to that of bupivicaine and tetracaine. These findings indicate a relative high risk of developing TNS with lidocaine compared to an unlikely risk with the other two local anesthetics [16] A meta analysis of randomized controlled trials and quasi- randomized controlled trials by Zaric and colleagues reported the overall risk of TNS with lidocaine versus bupivacaine, mepivacaine, prilocaine and procaine was higher. [17] Major databases were searched and fifty full text were extracted for review once quality was verified. Heterogeneity was examined between the studies and corrected for by a random effects model. A resultant fourteen studies were analyzed including 1361 patients. The development of TNS after lidocaine was greater than three percent than that of the other locals in the study, (p-value of less than 0.001). Interestingly, by excluding mepivacaine from the data, lidocaines' RR increased to 7.6 to bupivacaine, 6.4 to prilocaine, and 7.8 to procaine with a 95% CI. [17] Based on their results, approximately one in seven patients will develop TNS if intrathecal lidocaine is used. Large-scale epidemiological reviews warrant controlled trial studies. The Cochran review by Zaric et al [17] allowed for comparative findings involving many collective studies. Study findings between the groups vary, even after homogeneity was ensured. For example, Hampl et al [18] found a 32% incidence of TNS in their study after intrathecal lidocaine compared to Phillip et al [19] finding of 3%. Multiple uncontrolled variables can exist between studies. The review findings can be bias since variables between each study cannot be manipulated, such as the case with Freedman et al. [16] Technique was not controlled for, nor was there blinding to the local administration. However, the person performing the interview was blinded to the anesthetic choices and logistic regression was performed to control for the confounders. The analysis was also diverse in clinical practice approach. An earlier review of multiple prospective, randomized controlled studies by Pollock report the overall incidence of TNS as variable from 4.0%-37% after intrathecal lidocaine. [20] The relative findings in each study that were reviewed again varied in TNS outcomes. The authors support that patient positioning during the surgical procedure influenced the variable outcomes for TNS.

Positional orientation during surgery has been associated in the development of TNS. Lithotomy and knee arthroscopy positions following subarachnoid block with lidocaine has been associated with increased incidence of TNS. [16,21,22,23] However, intrathecal bupivacaine in the mentioned surgical positions has not shown an increased incidence of TNS. [16,21,22,23] The initial reports of TNS after IT lidocaine resulted after a lithotomy surgical procedure. [12] Freedman et al [16] identified a 24% risk of TNS using intrathecal lidocaine while patient is in lithotomy compared to 5% in the supine position. Pollock and colleagues developed a randomized, double-blinded, prospective study to determine the incidence of TNS and identify factors possibly contributing to its development. [21] Random assigned subjects numbering 159 undergoing knee arthroscopy or inguinal hernia repair received 5.0% hyperbaric lidocaine with epinephrine, 2.0% isobaric lidocaine without epinephrine, or 0.75% hyperbaric bupivacaine without epinephrine in a double-blinded fashion. Subjects undergoing knee arthroscopy had a 13% incidence of TNS after intrathecal lidocaine compared to 5% of the hernia supine position subjects. Unfortunately, Zaric et al [17] was incapable of measuring risks of TNS related to positioning secondary to inconsistency in available data. Canady et al [23] conveniently sampled 243 adults receiving spinal anesthesia of lidocaine 5.0% and 0.75% bupivacaine in the supine, prone, and lithotomy surgical positions. The incident of TNS in the lidocaine lithotomy group were significantly higher, p-value less than 0.05, and equal in the prone and supine positions. Pollock suggests that lithotomy positioning influenced her multiple study review. [20] The study subjects in Hampl et al [18] were mostly placed in lithotomy position and a 37% incidence of TNS was found.

Patient positioning after intrathecal administration of local anesthetics has been linked to an increased incidence of TNS, and noted to be higher after 5% lidocaine.

Freedman et al [16] identified a 3.6 RR of the outpatient lidocaine group compared to the inpatient group and no TNS in the outpatient bupivacaine group. Their outpatient lithotomy group developed TNS at a greater incidence than the inpatient lithotomy subjects; 24% and 7.7% respectively. Factoring out a possible lithotomy positional influence they compared the non-lithotomy subjects and found that 9.5% of the outpatients compared to 3% of the inpatients developed TNS. The incidence of TNS in the outpatient population was attributed to early ambulation. Unfortunately, Zaric and colleagues [17] did not include ambulation or outpatient statistics in their study, however other data suggest early ambulation may increase the incidence of TNS after IT lidocaine.

POSITIONAL RISKS

Lithotomy and knee arthroscopy position has been identified as altering the incidence of TNS after intrathecal lidocaine which is not present with bupivacaine. [16,21,22,23]. In Pollocks' prior mentioned review of randomized clinical trials, there were varied results of TNS dependent on positional status and local anesthetic utilized. [20] Patients placed in lithotomy position showed an TNS incidence of 30%-36% and knee arthroscopy position showed a lesser incidence of TNS of 18%-22%. [20,21,18,24,25] Pollock also presented studies suggesting a 4%-8% incidence of TNS after SAB in the supine surgical position. [20,21] These outcomes are fairly low compared to the prior discussed surgical positions. In the study by Pollock et al, [21] isobaric 2% lidocaine, hyperbaric 5.0% lidocaine, and 0.75% hyperbaric bupivacaine were compared between patients placed in the arthroscopy position. The authors surmised patient position might have placed the sciatic nerves at an extreme stretch, making them vulnerable to injury once exposed to the from the local anesthetic. Keld and colleagues [26] compared hyperbaric solutions of 5.0% lidocaine and 0.5% bupivacaine in seventy patients. Twenty-six percent of the patients developed TNS after lidocaine compared to three percent of the bupivacaine group. The associated risk with the supine position is minimal. Bruce Ben David et al [27] performed a comparative study in 2000 examining lidocaine alone and a lidocaine fentanyl mix in patients positioned in the arthroscopy position. Study outcomes suggested the knee arthroscopy position as a relative risk of developing TNS. This risk markedly increased (32.7%) when intrathecal lidocaine alone was administered compared to that of the mixed lidocaine and fentanyl group. Whether intrathecal fentanyl played a role in decreasing the development of TNS is not known. The lidocaine group had significant alterations in blood pressure that may have altered the study outcome. Some of the patients labeled as having TNS had persistent numbness in the buttocks and legs. Transient neurologic symptoms are not associated with numbness. Mislabeling these patient symptoms as TNS may have confused the study outcome.

Knee arthroscopy surgical position has been associated with the development of TNS. In a dilutional study among 109 patients, Pollock et al [28] associated the incidence of TNS with varying dilutions of lidocaine following SAB as insignificant. However, the study suggested that the positional orientation of the lower extremity during the knee arthroscopy could directly influence the incidence of TNS by 18-22%. Jack-knife prone position came into question as a possible link to TNS when Alley and Pollock [29] reported a case of TNS following hypobaric lidocaine in 2002. Again, the authors related the outcome to a sciatic stretch and possible maldistribution leading to some form of neural insult and related this stretch to outcomes seen in lithotomy and knee arthroscopy position. That same year, another study by Buckenmaier and colleagues found no incidence in seventy-two patients undergoing anorectal surgery after receiving low-dose intrathecal hyperbaric lidocaine and hyperbaric ropivacaine. [30] The local anesthetics were combined with 20 micrograms of fentanyl. No report of TNS occurred in either group. In a prospective study, Morisaki et al [31] reported a low incidence (0.4%) of TNS in 1045 patients undergoing anorectal surgery in the jack-knife prone position after subarachnoid 3.0% hyperbaric lidocaine. The studies are suggestive of increased risk factors related to lithotomy and knee arthroscopy positioning for developing TNS. The frequency of TNS after intrathecal injection of LA possibly is increased after knee arthroscopy or lithotomy surgical approaches. Placing the patient in the jack-knife prone position likely did not alter the risk of TNS in the above-mentioned case report. The anesthesia provider should not alter the anesthesia approach based on a single report of less than optimal outcome. However, reporting of findings such as these allows for future reference and basis for further study. Awareness of patient positions during surgery and after spinal anesthesia should be considered while planning the anesthesia approach.

EARLY AMBULATION

Freedman and co-workers, [16] after removing lithotomy positional influence from their study, found that 9.5% of the outpatients developed TNS, relating the increased incidence to early ambulation. A recent study revealed contradicting findings in which only 7.5% of early ambulation patients developed TNS among 120 patients who were randomized into groups of three hours, six hours and 21 hours after receiving 50mg intrathecal lidocaine with maximal TNS noted in the six hour group at 28%. [32] Silvanto et al [32] did not support their findings in the current literature. Consideration should be made for the small subgroup sizes of forty. A larger study group is needed to evaluate to outpatient ambulatory population. Ten of the patients had incomplete blockade requiring additional analgesia. Its not clear in the study which group was affected by incomplete block or if it was spread over all the groups. Another study by Cramer et al lacked conclusive findings to support current literature. Sixty patients were given intrathecal anesthesia with lidocaine in which half were allowed to ambulate immediately after spinal anesthesia had regressed. The second group lay supine for six hours. No correlation could be made between ambulation time and TNS. Early ambulation group developed a 23% incidence of TNS compared to 27% incidence in the supine group. The effects of early ambulation on developing TNS after local anesthetic injection with lidocaine are consistent with most research in the literature. Transient neurologic symptoms have an increased incidence in the ambulatory population. [16] Variable studies refute or lack supportive evidence to acknowledge the relationship between TNS and early ambulation. Small study numbers cannot be applied to the population at large, but can be directly utilized for future studies purposes and to evaluate outcomes of larger numbers. Those authors may want to repeat and enlarge their study group.

BARICITY AND DOSE

Baricity (hypertonic) and concentration of 5.0% lidocaine was implicated in neurotoxicity to the spinal neurons with the early cauda equina and TNS reports. However the risk of TNS is unaffected by baricity or dilution of lidocaine as dilute as 0.5%. [28] Freedman and colleagues studied lidocaine baricity ranges of 2.0%-5.0% and found no difference in TNS outcomes between the groups. [16] The authors examined variable doses of less than 50 milligrams (mg), 51-74mg, and greater than 75mg without significant TNS outcomes. [16] Zaric et al [17] discussed undifferentiated outcomes between hyperbaric and isobaric solutions, however failed to identify baricities, doses and concentrations in their meta analysis. The first case report of TNS following intrathecal hypobaric lidocaine was reported by Alley et al [29] in 2002 after 40mg of 2.0% lidocaine was diluted to 1.0% with two milliliters of sterile water after jack-knife surgical position. Pollock et al [28] failed to identify differences in TNS in patients receiving 2.0%, 1.0% and 0.5% lidocaine. Development of TNS in the study group was 15.8%, 22.2%, and 17.1% respectively. Consistent findings in literature offer little controversy to this specific risk.

VARIABLE RISK FACTORS IN TNS LITERATURE

Freedman et al [16] reported alterations in anesthesia approach not affecting or increasing the risk of developing TNS. These approaches include the addition of epinephrine to lidocaine, the type and size of spinal needle, needle aperture direction, position of the patient upon insertion of the spinal needle, age and gender, presentation of a bloody tap, and hypotension. Variables not shown to increase risk factors in the meta analysis performed by Zaric and colleagues were age, tourniquet time, surgery length, spinal needle bevel position upon insertion, midline or paramedian approach, and difficulty placing the spinal. [17] A relationship between certain needle types and developing TNS may exist. Pencil point needles are being evaluated in association with TNS. A pencil point needle during subarachnoid injection may possibly displace arachnoid tissue from dura. Use of small gauge needles could slow LA injection flow rates and decrease mixing of the local anesthetic with the CSF, both of which could lead to increased TNS risk. Although the above studies supported no relationship with needle type, new studies suggest an increased TNS risk when pencil point needles are utilized. Beardsley et al [34] found in their spinal model that 25 gauge and 27 gauge Whitacre spinal needles when directed sacrally will allow for a 2% concentration of local anesthetic at the sacral level. They suggest this is a toxic level which results from maldistribution of the local with the cerebral spinal fluid. The initial case reports of TNS by Schneider et al [12] utilized Whitacre spinal needles in two of the cases and a Sprott spinal needle in the other two cases, both of which are pencil point. Hampl et al [18] did not directly compare the needle type used in the study and relate them to TNS outcomes. However, the patients that had pencil point injections had a high incidence (43%) of TNS compared to Quinke needle incidence of TNS (29%). [18] In comparing the two pencil point needles, Whitacre and Sprott, incidence of TNS was 57% and 29% respectively. [18] The literature

suggests that there may be increased risk of TNS when using the pencil point needle during a subarachnoid injection.

PAIN ASSOCIATION WITH TRANSIENT NEUROLOGIC SYMPTOM

The pain associated with TNS is mild to moderate to severe. These symptoms are usually self-limited and therapy usually consists of non-steroidal anti-inflammatory (NSAID) agents and if severe, pressure point injections. [28] Hiller et al [35] had a 28% trial outcome of TNS in their study of which six of those people requested analgesia. They failed to list the analgesic utilized or specific pain score. Pollock et al [28] and Cramer et al [33] report a median pain score of six and five respectively but did not discuss analgesics or therapies for pain. Schneider and colleagues used diclofenac sodium and ambulation with the initial TNS patients. [12] Based on a zero to ten pain score, Freedman et al [16] reported pain scores of eight to ten in 30.1% of the TNS subjects and a four to seven in 48.2% of the TNS subjects. The impact of pain on the patient is difficult to assess on a psychological level without significant tests. However, as outpatient procedures climb in number, so will the episodes of TNS, associated pain, and possible negative outcomes in the less than healthy patient.

ETIOLOGY: TNS EVOLVING FROM UNCERTAINTY

The studies have suggested multiple risk factors for developing TNS following SAB. However, the underlying pathophysiology of TNS is not as well understood as the associative risk factors that may lead to its development. Neither baricity nor dilution of local anesthetics has been associated with the incidence or the etiology of TNS. [36] Proposed causes of TNS are the local anesthetics direct or indirect neurotoxic effects, myofascial injury, and direct nerve injury from trauma.

Schneider suggested a likely misdistribution and subsequent pooling of 5% hyperbaric lidocaine leading to a direct neurotoxic effect as a potential cause for the transient radicular symptoms seen in their case reports. [12] Later studies evaluated the possible neurotoxic effects of local anesthetics. Supporting the claims of Schneider and his colleagues, Beardsley et al [34] showed potentially neurotoxic effects from 2% concentrations of intrathecal lidocaine when slowly injected in a sacral direction during a single shot approach. Slower injectates may predispose to poor mixing of the LA with cerebral spinal fluid (CSF), thus potentiating pooling in the sacral region and leading to neurotoxicity. Hodgson et al [37] states that direct neurotoxicity to the nerve root may manifest as neural cellular damage, alterations in electrophysiology of the nerve conductive pathway, or clinically seen behavioral changes such as pain, sensory or motor deficits, or bowel/bladder dysfunction. Based on prior findings, the study proposed that less than 1% lidocaine concentration administered intrathecally would not result in TNS. However, 18% of their subjects developed TNS without significant differences between 0.5%, 1.0%, and 2.0% lidocaine. [37] The symptoms of TNS are clearly manifested as pain or dysesthesia, which is clinically behavioral. However, the relationship between TNS and pain remain to be linked specifically to neurotoxicity.

In a spinal nerve function study by Pollock et al, [38] electromyography (EMG), somatosensory electrophysiology (SSEP), and nerve conduction tests were performed and then compared between non-TNS and TNS subjects following intrathecal administration of 50 milligrams(mg) of a 5% lidocaine. [38] Although all volunteers in this study developed differences in the pre and postspinal testing of the peroneal and tibial nerves, there was no significant difference between the two groups of volunteers for changes in the nine nerve conduction test ($p = 0.4$), somatosensory, or electromyography exams. [38] Suggestion that no nerve damage exists in TNS patients may be considered a bold statement from Pollock and her colleagues. The study group was small, not randomized nor were they blinded. Additionally, these tests are neither specific to etiology nor conclusive of specific nerve damage.

Histological exams on neuronal cells are difficult to perform. Access to the spinal nerve cell in the living person is challenging and not without risk of injury. Johnson [36] discusses a possible mechanism of cellular injury after intrathecal LA administration as an increased intracellular calcium level. Johnson also speculates that sodium blockade is not a direct cause of the increased cytoplasmic calcium, but rather a biphasic response of release of internal calcium stores from the endoplasmic reticulum combined with an influx of calcium across the plasma membrane as the concentration of LA increases. [36] All LA are shown to elevate levels of calcium, with a sustained level increase associated with lidocaine. Local anesthetics have also been shown to exhibit effects on the neuronal growth cone of neurons, causing collapse and possible cytoarchitecture disturbance. [39] Radwan et al [39] studied chick neurons after exposure to four LA. Although dose dependent collapse occurred with each type of LA, the percentage of collapse was highest with lidocaine and mepivacaine showing a

respective 94% and 60 % collapse after 20 hours of exposure. The study results suggest a direct neurotoxic action of local anesthetic that is dose dependent on the in vitro chick neuron. Comparitively, the growth cone collapse induced by the ropivacaine and bupivacaine group were nearly equivalent at equipotent doses and exhibited a higher degree of reversibility than lidocaine. Rabbit spinal nerve root was found to have histological changes after exposure to 1.0%, 2.0%, and 4.0% tetracaine, with neurological dysfunction exhibited in the 2.0% and 4.0%. [37] The study authors found that the myelin sheaths at the nerve root entry zone are vulnerable to tetracaine induced elevated glutamate levels in the CSF microdialysate causing degeneration of the nerve root itself. The study used higher than standard normal concentrations seen clinically, however showed a correlation between tetracaine and glutamate concentrations and neuronal damage. The authors relate the glutamate neurotoxic effects seen with intrathecal tetracaine to other LA concentrations, suggesting that TNS likely has lower spectrum neurotoxicity. Elevated glutamate levels in the CSF can be neurotoxic. Yamashita et al [41] report that there is an insignificant difference in glutamate levels between the four LA utilized in intrathecal administration in their rabbit study. The lidocaine rabbit group developed greater sensory dysfunction compared to that of tetracaine, bupivacaine, and ropivacaine; the later two showing the lowest but nearly equal level of dysfunction. [41] The study used higher doses of LA than normally utilized clinically. However, the authors support that their goal was to evaluate the toxicity effects seen with LA and correlate them to glutamate levels. Although the lidocaine concentration used was two times greater than clinically relevant, and the other LA in the study was four times greater than clinically relevant, the glutamate levels were insignificant between the four local anesthetics This outcome suggests that all LA increase glutamate, however the neurotoxic effects may be a more direct effect of the LA and not related from the glutamate level. Lidocaine resulted in the highest toxicity between all the LA, suggesting that it has greater toxicity on the spinal nerves and likely has a narrow margin of safety.

Other studies have targeted myofascial stretching or relaxation as a probable cause of TNS, discounting the neurotoxic theories. Naviera et al [42] reported two cases that resulted in TNS after intrathecal 5.0% hyperbaric lidocaine and a full recovery from the spinal anesthesia. The treatment modality for these patients was injection of combined local anesthetics and steroid into the paraspinal musculature at specific pain trigger points. Both patients developed full recovery post injection of the combined treatment mixture. Hiller et al [35] suggests that relaxation of spinal supportive structures during spinal anesthesia is so profound from complete motor block that lordotic curve flattening and weakening occurs. They compared 5.0% lidocaine spinal anesthesia to general anesthesia that required motor blockade for intubation in the supine surgical position. [35] Motor relaxation seen with the non-depolarizing muscle relaxant may not allow for complete spinal motor block and therefore decrease chances of TNS. Incidence of TNS in this study was 27% in the spinal group and 3.0% in the general anesthesia group. Other experts support myofascial injury as a cause since the symptoms of TNS are self-limited and treatments with trigger point injections or NSAIDs are effective treatment modalities. [37] The lithotomy and knee surgery position have an increased incidence of TNS. If myofascial injury is causative in TNS, then increased stretching of the lumbosacral region during these positions may also increase the incidence of developing TNS.

Local anesthetics all have the potential for neurotoxicity. Lidocaine has been shown to have a greater potential for neurotoxicity than all the other local anesthetics at equipotent, above clinical, clinical and sub clinical concentrations. [36,37,39,41] Although some studies were applied to non-human subjects, a possible relationship exists between LA related cellular neurotoxicity and the development of TNS. A cellular evaluation on human nerve root at spinal level could be injurious to the volunteer. Those studies are critical in evaluating the possible relationship of cellular effects of local anesthetics in those studies and applying results to the human neural cell. However, non-human studies cannot be specifically related to human outcomes. The literature supports neurotoxicity directly and indirectly from the local anesthetic administration, however, there is no motor or sensory deficits exhibited in the TNS patient. The EMG, SSEP and nerve conduction tests were similar in the TNS subject and the non-TNS subject. There is a possible relationship to musculoligamental strain as causing TNS, since effective treatment with NSAIDs suggest and inflammatory process. The exact etiology of TNS remains a mystery.

PREGNANGY AS A SPECIAL CO-MORBIDITY

Possible physiological changes that occur during and remain after pregnancy have been associated with altered outcomes in TNS. [19,43,44] Phillip et al [19] compared 5.0% hyperbaric lidocaine to 0.75% hyperbaric bupivacaine in twenty-eight women undergoing postpartum tubal ligation. Their findings

of 3.0% TNS in the lidocaine and 7.0% in the bupivacaine group suggest an insignificant outcome between the groups. Although the study was limited in sample size, full motor block in the lidocaine group (100%) was higher than the bupivacaine group (82%). Aouad and colleagues reported a zero incidence (95% CI 0%-4.5%) of TNS following intrathecal 5.0% hyperbaric lidocaine and 0.75% bupivacaine in 200 subjects undergoing elective caesarean section. [43] They relate their findings to elevated levels of endorphins and enkephalins found in the parturient that have analgesic properties, and elevated progesterone hormone levels that may increase the threshold of pain. Another protective factor mentioned in the study is that the hormone relaxin has reached its peak level at term and associated maximal elasticity has occurred prior to spinal placement. [40] In 2003, Beilin et al [44] designed a study of 59 women to receive intrathecal 30 mg isobaric lidocaine or hyperbaric bupivacaine 5.25 mg for cervical cerclage during first and second trimester. Each group also received fentanyl 20micrograms (mcg) in a combined spinal epidural approach. The rational for the epidural was for rescue epidural dosing in case the spinal was insufficient. None of the lidocaine subjects required further dosing and one subject in the bupivacaine group needed supplementation via the epidural catheter, at which point lidocaine was utilized. The bupivacaine group developed no TNS compared to a 7.0% incidence of TNS in the lidocaine group ($p > 0.5$). [44] The results of this study suggest an alternative to lidocaine in this specific procedure, however the relationship between the two groups of TNS was not remarkable. Additionally, the addition of lidocaine to the epidural space in the supplemented bupivacaine group speaks to the rapidity and continued need for lidocaine in short procedures. The authors failed to discuss the relationship between TNS and pregnancy, however a 7.0% incidence remains lower than prior reports of TNS after lithotomy procedures on inpatient subjects. [16] Prior studies had suggested complete motor blockade as a causal factor to developing TNS. However, complete motor block resulted in a small percentage of post-spinal TNS for the postpartum lidocaine group discussed earlier compared to the bupivacaine group who had less complete motor block. The study should be repeated with a larger population. Pregnancy may possibly decrease the risk or mask the symptomatology of the parturient developing TNS after spinal anesthesia. Further research is warranted. The decision to continue intrathecal lidocaine use in the parturient is questionable. Although there seems to be a decreased incidence of TNS in pregnancy, the etiological concern over neurotoxicity from lidocaine remains. The fact that there is an increased pain threshold in pregnancy should not be misinterpreted as protection from local anesthetic neurotoxicity.

IMPACT OF TNS ON FUTURE ANESTHESIA FUNCTIONAL DISABILITY

The impact of TNS on the outpatient can alter the patients' perspective of the type of care received overall in the surgical setting. Also, those that experience TNS are less apt to suggest a spinal anesthetic to a friend or family member. [36] Functional loss for any given moment can be devastating to the patient. Tong et al [45] identified and studied functional loss and recovery in 453 subject undergoing transurethral procedures. Intrathecal lidocaine 1.0% or 5.0% was used after patients were randomized into groups. There was a 20% incidence of TNS from each sub group of subjects. [45] The authors identified walking, sitting and sleeping as major functional problems with the TNS patients. Most affected was patients' ability to sleep within the first 24 hours. Reports were equivalent to moderate to severe dysfunction. The second effected function was that of ambulating in which patients reported a moderate to severe loss for up to 48 hours. Loss of ability to perform activity of daily living from spinal anesthesia in the same day surgical patient defeats the purpose and idea behind ambulatory surgery. Patient satisfaction with their procedure and anesthesia are high indicators for performance evaluation in most ambulatory centers. The overall impact of TNS on patient satisfaction needs to be further evaluated and studied in relation to economical impact on medicine and

CONCLUSION

Transient Neurologic Symptoms remains a current issues in spinal anesthesia. As progressive practice and treatment focuses more on outpatient care, intrathecal anesthesia techniques are being investigated to apply evidence based medicine (EBM) to practice.

Both anesthetic success and time until discharge are dependent on dose of local anesthetic. Lidocaine has been utilized for decades in short procedures and valued for its short duration. Transient neurologic symptom is clearly associated with intrathecal lidocaine, possibly as high as 30%, which raises the question of lidocaines continued use for intrathecal short duration procedures, especially procedures involving lithotomy and knee arthroscopy positions. Altering the lidocaine dose and baricity does not manipulate the outcome of TNS. Mepivacaine as well

showed some near equal risks of developing TNS to lidocaine in the meta analysis study; however, more research should be conducted comparing the two local anesthetics. The etiology of TNS is unknown and needs to be further investigated. Whether TNS is myoskeletal and encouraged more from stretching the lower limbs or twisting the pelvis, as seen in lithotomy and knee arthroscopy positions, is unknown. Knee arthroscopy and lithotomy surgical positions have been shown to increase the risk for developing TNS after intrathecal lidocaine. Comparitively, the higher incidence of patients with TNS predominates in the outpatient ambulatory subject. Again this may be related to movement at the lumbosacral area, allowing lidocaine to promote neuronal irritation during stretching. It is possible that TNS develops via varied pathways and that no one factor can be identified as the root precursor. Studies investigating a neurological cause need to be performed. Unfortunately, performing these types of studies on a living human is quite difficult, as access to the nerves themselves are not without risks of injury. Fortunately, TNS resolves shortly and there have been no long-term untoward effects seen in the subjects, possibly denouncing a true neurotoxic effect from lidocaine. Even more supportive of a musculoskeletal etiology of TNS with intrathecal lidocaine is that pain relief is often accomplished with NSAID administration and symptoms lack a sensory or motor component. Utilizing neuraxial lidocaine in outpatient procedures should be considered carefully since the incidence of TNS is near 24% in early ambulation cases. It is possible that lidocaines use may continue if early ambulation is not warranted. Whatever the etiology may be, the pain associated with TNS detracts from patients overall satisfaction of the surgical and anesthetic experience and a solution should be sought. Questions arising in the field of anesthesia include whether low dose lidocaine mixed with an opioid or an alternative therapy with another local anesthetic could be utilized for short-term procedures. However, adjunctive or alternative therapies may increase the duration of action effecting ambulatory care recovery and discharge times. These are ongoing concerns and require more clinical trials involving these alternative approaches in the ambulatory setting.

A current concern for all the studies that involve outpatient TNS evaluation is the reporting of pain from the patient and lack of evaluation from a neurospecialist. Telephone interviews are not the optimal source of data collection and weaken a study significantly. Reported symptoms of pain may not be well understood by the patient nor correctly identified. Neurologic sensory or motor dysfunction may be present in these patients and can only be determined by a physical evaluation. Some symptoms may be so slight, that the patient is unaware of the dysfunction. Finally, a patient may be more apt to report pain if directly asked about that specific pain.

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