A universal goal of anesthesia providers is to provide the safest, most effective anesthesia and analgesia for their patients. When reports emerge showing problems or complications with an agent or technique that previously was thought safe, recommendations often are adopted in anesthesia departments to avoid or abandon the agent or technique, or alternatives are sought. Hyperbaric 5% lidocaine has been an effective and safe spinal anesthetic agent for short procedures for years. During the past decade, controversy arose over its use because it was implicated as the cause of transient neurologic symptoms and cauda equina syndrome. Use of bupivacaine or tetracaine results in a much lower incidence, but these agents are not as well suited to brief or outpatient procedures as is the shorter acting lidocaine. 

Substantial research has been conducted detailing the search for reasons these complications occur and how to prevent them. A sample of the findings is summarized in an attempt to present current knowledge about the apparent causes and prevention of transient neurologic symptoms. There is promising research showing that safe and effective short-acting intrathecal anesthesia can be accomplished with procaine, prilocaine, meperidine, and sufentanil.

Key words: Spinal anesthesia, spinal narcotics, transient neurologic syndrome.

Objectives

Upon completion of the article, the reader will be able to:

1. Differentiate between the signs and symptoms of cauda equina syndrome and transient neurologic syndrome.

2. Identify factors that have or have not been found to contribute to the occurrence of transient neurologic syndrome.
3. Describe the occurrence of transient neurologic syndrome associated with various local anesthetics.

4. Describe the characteristics of a narcotic intrathecal anesthetic.

5. Identify alternatives to lidocaine for short spinal anesthetics.

Introduction
Since 1991, controversy has arisen regarding the use of hyperbaric 5% lidocaine for spinal anesthesia. Reports of cauda equina syndrome after continuous spinal anesthesia and transient neurologic symptoms (TNS) after single-dose spinal anesthesia have prompted investigation into the cause and avoidance of further occurrences. Cauda equina syndrome is defined as a permanent neurologic deficit, resulting in dysfunction of the bowel, bladder, or both; perineal sensory loss; and variable lower extremity paresis.\(^1\) The onset is usually immediately after the causative injury. Transient neurologic syndrome and transient neurologic symptoms are terms used to describe temporary symptoms that appear 1 to 24 hours after the complete resolution of the spinal anesthetic and usually disappear within a few days or a week.\(^2\) The symptoms typically include back pain, weakness and numbness radiating to one or both buttocks and down the legs, or both. An earlier term for this syndrome was transient radicular irritation, but since the incidence of back and leg pain do not consistently manifest with sensory-motor deficits or reflex abnormalities, radicular irritation cannot be pinpointed as the reason for symptoms. Transient neurologic syndrome seems to be the more appropriate term.\(^3\)

Many surgery- and anesthesia-related variables have been studied as possible causative agents and alternatives proposed. The purpose of this article is to review current research that seeks to quantify and compare incidences of TNS caused by various agents used for spinal anesthesia and other perioperative factors. Although a few case studies and anecdotal reports are presented, most of the articles reviewed involved prospective study groups of more than 50 patients.

Background
When reports of cauda equina syndrome after continuous spinal anesthesia with microcatheters were made in 1991,\(^4,5\) the cause was thought to be pooling of large amounts of concentrated local anesthetic at the lumbosacral roots, leading to excessive exposure and toxic effects. Injection of local anesthetic through 28- to 32-gauge microcatheters in models showed uneven mixing and maldistribution of the anesthetic in cerebrospinal fluid.\(^6,7\) Subsequently, microcatheters used for continuous spinal anesthesia were removed from the market by the US Food and Drug Administration,\(^8,9\) and the manufacturer of lidocaine issued recommendations to dilute the drug before using it in spinal anesthesia.\(^10\) Larger gauge (20-24 gauge) macrocatheters continued to be used for continuous spinal anesthesia.

However, case reports of TNS after single-dose spinal anesthetics began to surface, prompting several randomized studies.\(^10-21\) These studies confirmed that patients experience TNS significantly more after hyperbaric lidocaine spinal anesthesia than after anesthesia with bupivacaine, tetracaine or prilocaine,\(^11,12,14,16-19\) but the question was “Why?”

Comparing local anesthetics
Once it was noted that the incidence of TNS occurring after 5% lidocaine spinalwas as high as 40% compared with bupivacaine (0.8% to 3%) or tetracaine (1.6%) (Table), studies were done to determine what, if any, compounding factors existed.

In animal studies, it was determined that higher than clinically used concentrations of lidocaine and tetracaine are toxic to unsheathed nerves.\(^21-23\) Plain tetracaine was associated with an incidence up to 1.6% of TNS, but tetracaine with phenylephrine caused a 12.5% incidence.\(^7\) The occurrence of TNS with lidocaine with or without a vasoconstrictor was not different.\(^14\) Dilution of the injected drug with cerebrospinal fluid did not seem to make a difference, nor did using lower concentrations of lidocaine.\(^15\) Comparisons of 5% lidocaine with 2% lidocaine yielded similar incidences of TNS.\(^25\) Although Chan et al\(^25\) found that equivalent continuous spinal anesthesia could be accomplished with 0.5% lidocaine as with 5% lidocaine, concentrations as low as 2% lidocaine caused nerve damage in animals.\(^22\) Although the exact mechanism has not been pinpointed, lidocaine seems to have more neurotoxic effects than bupivacaine or tetracaine. A study of 1,045 patients receiving low doses of 3% lidocaine (30-45 mg) demonstrated an occurrence of TNS of 0.4%.\(^15\) All patients in that study underwent...
surgery in the prone jackknife position and stayed in the hospital overnight.

**Contributing factors**

The 2 most highly correlating nondrug variables in the incidence of TNS are lithotomy position during surgery and outpatient status. A much higher incidence of TNS has been found in patients undergoing surgical procedures involving the lithotomy position or positions in which the knee or hip is bent.12,14,25 This positioning causes stretching of the lumbosacral nerve roots, possibly causing them to be poorly perfused and more vulnerable to the toxic effects of the local anesthetic.2,15 Although the majority of cases of TNS are associated with the use of lidocaine, the cases in which other local anesthetics have been involved were associated with the lithotomy position12,14 or knee arthroscopy.12,25

There also could be confusion of true TNS symptoms with pain or discomfort caused by other factors related to the surgery. A known possible complication associated with the lithotomy position is compression of the peroneal nerve when the patient’s leg is pressed against the leg holder or stirrup.26 In the study comparing lidocaine with bupivacaine by Hampel et al,3 the 1 patient identified in the bupivacaine group as having TNS actually had numbness and dysesthesia of 1 foot. She had been in the lithotomy position for more than 200 minutes, and a consulting neurologist thought the symptoms were due to positioning. No other defined symptoms of TNS, such as back pain or buttock pain radiating down the legs, were reported for this patient.

Several studies have shown the incidence of TNS to be higher in outpatients.12,14,25 Freedman et al12 suggested that earlier ambulation and return to daily activities had a role in perceived TNS symptoms.

**Noncontributing factors**

Intrathecal 10% glucose and epinephrine27,28 in animal studies seem, by themselves, to be nonneurotoxic, although it was postulated that addition of epinephrine to spinal anesthetics could increase toxic effects by vasoconstriction and delaying dispersion of the local solution.12,14,28 In studies on osmolality, hyperbaric lidocaine, and isoobaric lidocaine produced the same incidence of TNS.21

Freedman et al12 found intrathecal morphine or fentanyl to be noncontributory to the incidence of TNS. The extensive study by Hodgson et al29 on the neurotoxic effects of spinal agents concludes that most spinal agents have not been studied adequately for safety. However, little evidence exists to show neurotoxic effects resulting from the intrathecal use of opioid analgesics, clonidine, neostigmine, or most preservatives. Despite the lack of controlled animal and human histologic studies, these agents have a long history of clinical safety.29

Since the use of microcatheters for administration of continuous spinal anesthesia has a definite role in causing TNS and cauda equina syndrome, the size and type of needles used for administration of single-dose anesthetics also was studied. In 1,863 patients studied by Freedman et al,12 no significant difference in the incidence of TNS was associated with spinal needle size or type (Quincke versus pencil point). The needle data were applied only to patients receiving lidocaine.

### Table: Incidence of transient neurologic symptoms with local anesthetics*

<table>
<thead>
<tr>
<th>Authors</th>
<th>Lidocaine 5%</th>
<th>Lidocaine 2%</th>
<th>Tetracaine 0.5%</th>
<th>Bupivacaine 0.5-0.75%</th>
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<tbody>
<tr>
<td>Hampel et al3</td>
<td>37</td>
<td>...</td>
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<tr>
<td>Hampel et al10</td>
<td>32</td>
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<td>Hampel et al16</td>
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<td>Hampel et al21</td>
<td>33</td>
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<tr>
<td>Freedman et al12</td>
<td>11.9</td>
<td>...</td>
<td>1.6%</td>
<td>1.3</td>
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<td>Pollock et al14</td>
<td>16</td>
<td>16</td>
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*Data are given as percentages. Ellipses indicates not studied.
although bupivacaine and tetracaine also were used in the study. König and Ruzicic also found that varying the needle types and sizes did not affect the incidence of TNS associated with lidocaine spinal anesthetics.

Problems assessing the true incidence of TNS

No particular reason has been studied as to why outpatients have a higher incidence of TNS than inpatients, although inaccurate reporting by patients is highly suspected. A lower reported incidence of TNS occurs when patients are assessed in person by healthcare professionals rather than by telephone. It is suggested that patients undergoing procedures extensive enough to require inpatient stays may have surgical pain that masks true incidences of TNS or the corollary, and better pain relief by stronger analgesics that they would not be receiving at home.

Musculoskeletal causes

Moore and Thompson describe the incidence of back pain following spinal anesthesia that was thought to be muscular in origin. Reference is made to rough handling of the paralyzed patient, leading to musculoskeletal pain postoperatively. Relaxation of back muscles caused by spinal anesthesia may result in postoperative muscular pain and spasms. Myofascial trigger points that cause pain in the back and buttocks as described by Naveira et al exhibit symptoms similar to those of TNS. In the absence of reflex, sensory, or motor deficits, muscle spasm should be studied as a possible causative agent. Damage to the peroneal nerve, which is a known complication of the lithotomy position, can show symptoms similar to those of TNS.

Preexisting back and leg pain, neuropathies, nerve injuries, and disease states should be identified and documented preoperatively. In the postoperative period, a careful and complete assessment of the patient must be done to differentiate between new and preexisting conditions and to determine whether pain is of musculoskeletal or neural origin.

Alternative local anesthetics

In an attempt to identify an acceptable alternative to hyperbaric 5% lidocaine for short case and outpatient spinal anesthesia, some prospective studies using prilocaine, procaine, and mepivacaine have been conducted since these agents are similar in duration and potency to lidocaine.

In a report by König and Ruzicic, 2% prilocaine caused no cases of TNS in 5,000 patients, but this study was neither prospective nor blinded. In a prospective, double-blinded study by Hampl et al, 2% lidocaine, 2% prilocaine, and 0.5% bupivacaine were compared. The characteristics of the spinal blocks and recovery times were similar for lidocaine and prilocaine. The incidence of TNS in this study was 13% for lidocaine and 3% for prilocaine, but the study groups comprised only 30 patients each. Martinez-Bourie et al found a 1% incidence of TNS occurring in a study group of 100 patients receiving 5% prilocaine spinal anaesthetics vs a 4% incidence in 100 patients receiving 5% lidocaine.

Axelrod et al used 5% procaine for spinal anesthesia in 106 patients with an incidence of TNS of 0.9% (1 patient). This short-acting local anesthetic provided adequate surgical anesthesia for procedures lasting 45 minutes. With the addition of fentanyl to the procaine spinal anesthetic, the duration of the block was prolonged to almost 100 minutes. Stewart’s description of success with intrathecal 10% procaine was not in the form of a prospective study, but during several years, no incidence of TNS was noted in approximately 500 patients who received it.

Mepivacaine demonstrated a 30% to 37% incidence of TNS in 2 studies comparing it with bupivacaine; otherwise block characteristics were similar to those of lidocaine.

In summary, although research on alternatives to lidocaine for spinal anesthesia is scant, prilocaine and procaine seem to be safe alternatives to lidocaine for short procedures. Mepivacaine also deserves more research to determine its safety as a spinal anesthetic, but the current data point to an incidence of TNS similar to that of lidocaine.

Intrathecal narcotics

As an alternative to local anesthetics, intrathecal meperidine as a sole anesthetic agent has been studied for short surgical procedures. Commonly used in the parturient, spinal narcotics have provided adequate analgesia for short surgical procedures, such as knee arthroscopy, urological procedures, hernia repair, and perianal surgery. Meperidine provides sensory and motor block with a low incidence of hypotension, while providing excellent postoperative pain control. Intrathecal sufentanil has been used successfully for extracorporeal shock wave lithotripsy as the sole anesthetic agent, providing excellent analgesia and improved recovery time compared with intrathecal lidocaine.
intrathecal narcotics have been reported as drowsiness, fall in blood pressure, pruritus, and nausea, but they were considered by patients and providers to be minor and low in incidence.\textsuperscript{40,43,44}

Addition of prilocaine to meperidine for spinal anesthesia in a study by Tausin-Fin et al\textsuperscript{45} produced acceptable surgical anesthesia without adverse effects. Sangarlangkarn et al\textsuperscript{46} reported a high frequency of undesirable adverse effects with meperidine spinal anesthesia, but the dose used in this study (reported in 1987) was 100 mg in each patient. Most later studies used 0.5 mg/kg to a maximum dose of 60 mg of meperidine intrathecally.\textsuperscript{40,41,43}

**Conclusion**

Despite various research studies that seek to isolate a distinct cause of TNS, the answer remains elusive. Undoubtedly, lidocaine possesses some property that causes a greater incidence of TNS than found with other local anesthetics, but whether this is due to true neurotoxic effects or some other mechanism has not been proven. Patient positioning has a definite effect on the incidence of TNS, although whether it promotes muscle spasms, pressure injuries, or stretching of the sacral nerve roots to cause the symptoms has yet to be determined.

Current research, although somewhat lacking in animal and human studies for safety, indicates that most common spinal adjuvants, such as epinephrine, glucose, and opioids, are safe for intrathecal use and do not contribute to TNS.

In surgical situations in which an intrathecal local anesthetic is preferred over narcotics, procaine and prilocaine seem to provide safe, short-acting spinal anesthesia. Although more research is needed with these agents to confirm safety, it seems that they are highly suitable for short cases. Outpatient recovery and discharge times need not be prolonged by use of bupivacaine or tetracaine or by avoidance of regional anesthesia.

Many studies from Europe describe using intrathecal meperidine or sufentanil for short surgical procedures, such as knee arthroscopy, urological procedures, hernia repair, and perianal surgery. In comparison studies, the intrathecal narcotics provided good intraoperative analgesia and shorter recovery times than intrathecal local anesthetics.

With several intrathecal agents to choose from to provide anesthesia and analgesia and so many variables involved, many more research studies are needed to clarify the factors to avoid so we as anesthesia providers can spare our patients possible TNS.

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