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Transient Neurological Symptoms and Spinal Anesthesia

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Abstract

The spinal anesthesia induced by injection of local anesthetics in the subarachnoid space, which blocked the conduction function of some spinal nerves. However, there are various kinds of anesthetics used in spinal anesthesia; many of them can cause transient or permeant neurological symptoms. One often-overlooked complication of intraspinal anesthesia is Transient Nervous Syndrome (TNS). TNS can affect patients' mood and postoperative recovery. This paper mainly describes the related factors of TNS after spinal anesthesia, such as local anesthetics, surgical position and operation time and discusses the mechanism, prevention and treatment of TNS.

Keywords: Transient neurological symptoms • Local anesthetics • Spinal anesthesia • Prevention

Introduction

Transient Neurological Symptoms (TNS)

The clinical manifestations of TNS are unilateral or bilateral hip pain after the disappearance of lumbar anesthesia, most of which are accompanied by back pain and a few of which are insensitive radiated to the thigh. These symptoms last from a few hours to about a week [1-3]. There are a lot of clinical reports about TNS. Some patients complained of lumbosacral pain (radiated to lower limbs, aggravated by sitting), some patients have severe pain, mainly in the thigh, most patients have pain levels of 6 to 7. In some patients with cesarean section, a small number of people have thigh pain, hip pain, these symptoms last for 12 hours-24 hours and some lasts for 24 hours-48 hours. The pain was usually relieved or disappeared about a week after surgery. Physical and imaging examinations often show no neurologically positive changes. Although TNS is a complication of anesthesia, it does not damage the nerve and has no serious consequences for the nervous system. Factors associated with TNS include the patient's surgical position, duration of surgery, needle type, the type of anesthetics in spinal anesthesia [4-7].

Spinal anesthesia with lidocaine, the incidence of TNS varied between 10 percent and 40 percent. In addition, TNS has been reported in 39.6% of the total cases of spinal anesthesia with lidocaine or bupivacaine [8,9]. Although TNS does not cause nerve damage, the pain after surgical anesthesia will affect postoperative recovery, affect patient's mood, reduce patient's time to get out of bed and walk and cause certain psychological burden. This review will describe TNS related anesthesia and possible mechanisms in detail [10].

Literature Review

Etiology and pathogenesis

Bupivacaine is highly toxic and its specific local anesthetic pharmacological properties may cause TNS. Numerous studies have reported that significantly higher incidence of the TNS when using lidocaine [11-13]. Spinal needles used in spinal anesthesia may also cause nerve, endorhachis, or nerve root damage, however, the intensity of dural injury does not seem to be an important factor in the development of TNS. The patient's surgical position often leads to the concentration of local anesthetics in one area, for example, the left and right lying positions are the local anesthetic concentration on the unilateral limb. However, few studies have conducted on this type of muscle, because the abnormal muscle itself could cause TNS. The duration of surgery is also a factor affecting the occurrence of TNS. We know that the longer the surgery, the longer the postural fixation, the longer the stimulation time to the spinal nerve and therefore the incidence of TNS will increase correspondingly [14]. The type of surgery and the patient's own factors may also be associated with TNS, such as arthroscopic surgery and obesity. Some studies of TNS after muscle disorders such as muscle cramps and myofascial trigger points are associated with a patient's position and anesthesia needle. Although there are many factors related to TNS, most of them focus on spinal anesthesia operation and anesthetic drugs, the puncture position and injection position of lumbar anesthesia needle and the position requiring block in operation are not the etiology of TNS [15,16]. For the reasons explained above, personal preference for local

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anesthetics is more important [17-20]. As we all know, the mechanism of local anesthetics is the reversible blocking of the neural Voltage-Gated Sodium Channel (VGSC), the binding site of VGSC is the sodium ion channel in the cell, which affects the transmission of nerve impulses and leads to anesthesia [21,22]. TNS is a kind of complications associated with pain, neurological symptoms caused by certain stimulation, the pharmacological toxicity of local anesthetics, namely the central nervous system and cardiovascular toxicity, is the most worth TNS research explore factors [23]. Nerve stimulation by local anesthetics can lead to a range of symptoms, most commonly pain and numbness. Therefore, the occurrence of TNS largely related to the toxicity of local anesthetics [24-28].

The local anesthetics related to TNS

Pain in the lower back and radiated to the lower extremities after spinal anesthesia is affected by a variety of factors. One particularly important factor is the type of anesthetic used [29,30].

Lidocaine: Lidocaine is a local anesthetic of amides derivatives. Lidocaine is the most commonly used drug in clinical anesthesia, its pharmacological low toxicity, can penetrate mucosa block of peripheral nerve conduction, clinical applications, can be used in gastroscope intubation, can also be used for emergency treatment of ventricular arrhythmia [31,32]. Due to the high risk of TNS, lidocaine is no longer recommended. In 125 patients given lidocaine anesthesia, 85 patients developed back pain, neuropathic pain in the buttocks or thighs [33-35]. Just as mentioned earlier in this article scholars found the relative risk for developing TNS after spinal anesthesia with lidocaine was 4.35, as compared to other local anesthetics (bupivacaine, prilocaine, procaine, levobupivacaine and ropivacaine). In a lidocaine and chloroprocaine control study, the authors found that 4 patients accepted the lidocaine spinal anesthesia occur TNS postoperative, the pain symptoms relieved in 2 days, while patients accepted chloroprocaine did not appear the TNS, thus lidocaine is not good for spinal anesthesia. No matter what concentration of lidocaine is used, the occurrence of TNS is inevitable. However, There were no reports of TNSs as the patients received a single-shot spinal injection, with 2% isobaric lidocaine along with titrated propofol sedation [36,37].

Prilocaine: Prilocaine is a short-acting lipid local anesthetic, with low lipophilic property and weak penetration to mucosa. It is not suitable for surface anesthesia, but has low toxicity. Since it was used for spinal anesthesia in 1960, studies have found that its incidence of TNS is lower than that of lidocaine, so it is often used to replace lidocaine for spinal anesthesia [38-40]. Procaine and lidocaine for comparison study found that the use of procaine did not occur TNS after spinal anesthesia, by contrast, patients with spinal anesthesia with lidocaine occurred within 24 hours of TNS probability is 20%, the symptoms disappeared in 4 days. Similarly, an analysis of more than 5,000 cases of spinal anesthesia using prilocaine found that none had TNS. The low doses of hyperbaric prilocaine used in spinal anesthesia do not produce a sufficient amount of σ-toluidine thereby avoiding additional risk for the patient. Hampl compares the relative risk of TNS in prilocaine, lidocaine and bupivacaine and finds that there is a 30% risk of TNS in lidocaine, 3% in procaine and no TNS in bupivacaine.

The above study found that procaine appears to be an alternative to lidocaine for spinal anesthesia because of its low toxicity and low risk of TNS. However, one of the biggest disadvantages of procaine is that it is easy to cause allergies and has a low anesthetic effect, which limits its clinical application [41].

Mepivacaine: The pharmacokinetic of mepivacaine was special similar to lidocaine, accordingly, but because of its side effects are similar to those of lidocaine, the risk of TNS is similar, so don't recommend using it for clinical spinal anesthesia [42,43].

Articaine: Compared with bupivacaine or lidocaine, the anaesthesia effect is fast and the action time is short. Although the anesthetic effect of atecaine is good and fast, the adverse reactions are more and the toxicity is greater. Therefore, the use of atecaine in spinal anesthesia generally not recommended. Although it is not recommended for spinal anesthesia, but has been reported, compared with lidocaine, it is high risk of intraoperative hypotension, lower risk for postoperative TNS. The overall incidence of Transient Neurologic Symptoms (TNSs) in these studies was low (Articaine 1.2%, Prilocaine 1.6%) [44].

Chloroprocaine: Chloroprocaine has similar pharmacological effects as procaine, with relatively fast and strong anesthetic effect and low toxicity. It's used for infiltration anesthesia, nerve block anesthesia, sacral canal and epidural anesthesia [45] .

Chloroprocaine outpatient, there are 503 cases used for anesthesia and no one appear the symptom of TNS postoperative, the authors conducted a more in-depth study of this study, more than 4000 cases of spinal anesthesia in patients using the chloroprocaine none appeared serious sequelae of nervous system, only four TNS. Through epidemiological and statistical analysis found that chlorine pp because the incidence of TNS is 0%-1.9%, compared to the downside than lidocaine much lower, its basis is in a prospectie, randomized, double-blind study, chlorine pp because incidence of TNS is 0% and the incidence of lidocaine was 33%. Thus for spinal anesthesia of outpatient surgery is a good choice. There are two sides to everything. Chloroprocaine drug toxicity and allergic reaction limit its clinical application [46,47].

Levobupivacaine: Levobupivacaine is a local anesthetic of amides, which blocks the generation and conduction of nerve stimulation by increasing the threshold value of nerve electrical stimulation, slowing down the transmission of nerve stimulation and reducing the rate of increase of action potential. In spinal anesthesia, levobupivacaine had a 3.33% risk of developing TNS compared to lidocaine TNS and lidocaine had a 26.6% risk.

This study shows that levobupivacaine is more suitable for spinal anesthesia. But it doesn't mean it's completely safe, a literature search revealed only one study that mentioned the incidence of TNS after spinal anaesthesia with levobupivacaine. Thus levobupivacaine is usually used for epidural anesthesia but not for subarachnoid anesthesia [48].Commercial bupivacaine is a racemic mixture of(R)-and(S)-stereoisomers. In addition to this problem of cardiovascular toxicity as a result of accidental intravenous injection of bupivacaine drug, single enantiomers were developed in the hope that they would be gradually safer. Ropivacaine (Naropin) and levo-(S)-bupivacaine (Chirocaine) 123 were formulated to exploit this stereo selectivity of the drug.

Ropivacaine is a single(S)-stereoisomer that differs from levobupivacaine in the replacement of a propyl for the butyl group on the piperidine ring. With these modified changes in molecular structure, it was hoped that ropivacaine and levobupivacaine would be less intrinsically cardio toxic. Inversely, it shows that the (S)-enantiomers of mepivacaine and bupivacaine are metabolized by the liver more slowly than the corresponding(R)-enantiomers, which would may lead to somewhat greater when compared to systemic accumulation with long duration of infusions.The very slow process of reversal of Na^+ channel blockade after a cardiac action potential, which is a hallmark of bupivacaine, is considerably faster with that of ropivacaine. In addition to this kind of electrical differences, the negative inotropic potency of ropivacaine on isolated cardiac tissue appears to be considerably less than that of bupivacaine. Both electrical and mechanical differences in the toxic profiles studies may arise from the selective inhibition of Ca^{2+} currents by bupivacaine.

Ropivacaine: The Ropivacine used in spinal anesthesia has a sense of separation from movement, which means motor function recovers rapidly but the extended duration of sensory block, it has been documented to produce less motor blockade than other local anesthetics. Ropivacaine has an anesthetic strength of 8 times that of procaine and aging effect is 4 times-8 times that of procaine and its central nervous system and heart toxicity are lower than that of bupivacaine.

Ropivacaine rarely causes hypersensitivity, heterogeneous and abnormal reactions. Compared with lidocaine, the TNS risk of ropivacaine significantly reduced and the effect of anesthesia block was significant. In outpatient arthroscopic surgery patients, using ropivacaine none TNS occurs after anesthesia, but there are exceptions, in 2000, Sugantha Ganapathy reported a knee arthroscopy surgery patients, he accepted a low dose of ropivacaine intrathecal injection and occurs TNS for 3 weeks. Which means ropivacaine could also cause TNS. There are need larger studies to determine the incidence of TNS after administration of intrathecal ropivacaine.

Above all, we seem the lidocaine and mepivacaine is no longer recommended, atecaine and chloroprocaine have great toxicity, procaine cause allergies and has a low anesthetic effect, levobupivacaine is usually used for epidural anesthesia but not for subarachnoid anesthesia, it seems ropivacaine was the best anesthetic for subarachnoid anesthesia.

Discussion

The patient position related to TNS

TNS may also be caused by the patient's position and the patient's own factors during surgery, such as stone cutoff and flexion of knee, which may result in the long-term effects of drugs on certain nerves, resulting in higher incidence.

Compared with supine surgery, the incidence of lower back pain combined with sensory abnormalities was higher in patients with stone amputation, patients received lidocaine and underwent surgery in the lithotomy position occurs TNS (Figure 1).

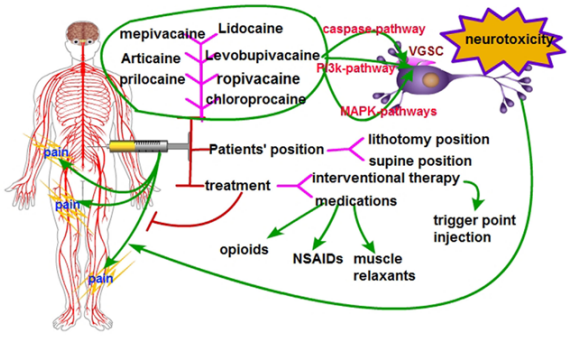

Figure 1. Mechanism diagram of TNS.

With the continuous improvement of surgical technique and the continuous improvement of the operation environment, patients' position gradually reduce the influence on the TNS and sufficient preoperative preparation make a patient's own factors influence on TNS also gradually reduced.

The possible mechanism diagram

Using Portable-Pathway-Builder-Tool-2.0 Microsoft to build the possible mechanism diagram of TN).

Needle type

The drug distribution in subarachnoid space is the same, but the injury of subarachnoid space is different, instead of cutting off the dural fibers, the nib lumbar anesthesia needle pushes the dural fibers into the subarachnoid space and the fine needle greatly reduces the incidence of pain [49].

Prevention and treatment

Until now, no patients with or without anesthesia reported permanent neurological deficits, after the occurrence of TNS clinically, there is usually no need for treatment. The risk of TNS was independent of dose and specific gravity of the drug. So the most important measure is to prevent the occurrence of TNS. The new study found that the occurrence of TNS was not associated with local anesthetics, but with the type of surgery, intraoperative posture and patient complications. Intravenous dexamethasone before combined spinal and epidural anesthesia can effectively prevent TNS, dexamethasone can also improve postoperative analgesia after spinal anesthesia. Analyze the possible reason was local anesthetics have effects on the presynaptic membrane and the posterior membrane and dexamethasone acts on the presynaptic motor nerve endings to make the release of tedylcholine easier, which plays a positive role in eliminating the blocking effect of local anesthetics. For severe TNS the medications and interventional therapy were the most common treatment strategies, those include opioids, Nonsteroidal Anti-Inflammatory Drugs (NSAIDs), muscle relaxants, symptomatic therapy as well as trigger point injection. Due to those treatments not always successfully, the emphasis of TNS is prevention. In today, lidocaine is probably not used in spinal anesthesia, avoidance of lidocaine spinal anesthesia in ambulatory patients undergoing lithotomy or knee arthroscopy is crucial. The ropivacaine has the low incidence of TNS that could use common instead of other local anesthetics [50].

Conclusion

Postoperative neurologic complications such as TNS associated with regional anesthesia are complex, including local anesthetics neurotoxicity, concentrations, species. Operation types and patient factors are also play a role on TNS. This often makes it very difficult to prevention. At the cellular level, the neurotoxicity of local anesthetics is caused by the effect on the intrinsic caspase-pathway, PI3k-pathway and MAPK-pathways. If we can develop drugs for caspase-pathway, pi3k-pathway and mapk-pathways, we may be able to effectively prevent and treat TNS.

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