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Lidocaine for Spinal Anesthesia. Can and should be used

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Introduction

In a recent e-book "Lidocaine: Current Concepts and Emerging Roles in Clinical Practice," I wrote the chapter on the use of lidocaine for spinal anesthesia [1]. Spinal anesthesia has been used now for over a century, and a number of local anesthetics are available. Increased employment of day case surgery has led to increased use of spinal anesthesia, especially with rapid onset and short duration of action, allowing patients to go home sooner.

Lidocaine, the first amino amide type local anesthetic, was first synthesized under the name xylocaine by Swedish chemist Nils Lofgren in 1943. His colleague Bengt Lundqvist performed the first injection anesthesia experiments on himself [2].

It was first marketed in 1949.The drug was a result of a series of experiments with basic anilids which were widely different in structure from the cocaine-procaine group. Many of the advantages of lidocaine over procaine are due to the substitution of the ester linkage in the long side chain by an amide group, but the presence of the two ortho-methyl groups is also vital. The use of lidocaine for spinal anesthesia was first published in 1948 with a 2 mL of 2% solution in 10% glucose for urological operations produced a rapid and satisfactory anesthesia [3]. In 1954 Berne reported his experience with the 5% solution [4]. In the late 1950s many papers were published describing the use of 5% lidocaine. In 1956, a study comparing the efficacy of 5% lidocaine in 7.5% dextrose with 2.5% lidocaine in 4% dextrose showed that the most effective spinal anesthesia was obtained with 1.5 mL of the 5% solution [5]. The reason for using 5% lidocaine for spinal anaesthesia is unclear and this concentration has been repeatedly linked to a transient radicular irritation syndrome, particularly in hyperbaric preparations. In Brazil the solutions used in spinal anesthesia are: 2% isobaric lidocaine [6], 1.5% and 2% hyperbaric lidocaine [7] and 0.6% hypobaric lidocaine [8].

Methodological Evaluation Errors

Lidocaine was synthesized in 1943 and introduced as a 2% hyperbaric preparation for use in spinal anaesthesia. It became popular for short duration procedures due to rapid regression of the sensory and motor block, which enhanced its safety. For almost 50 years, lidocaine has enjoyed an incredible popularity as a short-acting local anesthetic and was considered to represent a standard drug for short surgical procedures performed under spinal anesthesia. Its reputation was based on a remarkable safety record devoid of reports suggesting a potential for neurotoxicity and the results from a large-scale prospective review [9]. Since then, according to reasonable estimates, lidocaine has been used effectively and safely for spinal anesthesia in some fifty million patients [10]. This preparation was used safely for a long period until the onset of cauda equine syndrome with use of microcatheters for continuous spinal anesthesia [11].

Although spinal anesthesia is associated with relatively low rates of neurological complications, when complications do occur the effects may be severe and permanent. These may be a result of a combination of needle injury, unusual anatomy, and the effect of anesthetic drugs. Cauda equina syndrome (CES) is a serious neurologic condition in which there is acute loss of function of the lumbar plexus, neurologic elements (nerve roots) of the spinal canal below the termination (conus medullaris) of the spinal cord. In 1991, four cases of persistent sacral nerve root deficits were reported following continuous spinal anesthesia [11] (Table 1).

Case	Age	Needle	Catheter	Surgery	Local Anesthetic	Dose
1	68 ys	22G	28G	T.R.P.	Lidocaine 5%+Glicose 7.5%	175 mg
2	45 ys	22G	28G	Burniotomy bilateral	Lido 5%+Epi+Glicose 7.5%	300 mg
3	56 ys	22G	28G	Saphenous neuroma	Lidocaíne 5%+Glicose 7.5%	190 mg
4	67 ys	18G	20G	Bypass artery	Tetracaine 0.5%+Glicose 5%	37 mg

Table 1: Four cases described by Rigler et al [11].

In all cases, a dose of local anesthetic had been administered that by far exceeded recommended dosages for single-shot spinal anesthesia; in 3 cases, 5% lidocaine had been injected through a 28-G microcatheter, whereas in the remaining case 0.5% tetracaine was administered through a 20-G catheter (epidural catheter). Similar reports soon followed and finally led to an alert about its use in spinal microcatheters from the US market [12, 13]. As a possible mechanism, it was suggested that the slow speed of injection when using spinal microcatheters results in poor mixing of the local anesthetic with the cerebrospinal fluid and consequent maldistribution, thus exposing neural tissue to potentially toxic concentrations of local anesthetic [14,15,16]. In the four cases reported in 1991 [11], three were with 5% lidocaine injected through the microcatheter and one case with tetracaine 0.5% injected through the 20G catheter (epidural catheter). In this way, lidocaine and microcatheter could never be incriminated, because tetracaine and a large caliber catheter

were involved in these cases. What led the FDA [12] to make an alert unfortunately is not understandable. Paracelsus, physician and physicist of the sixteenth century, said "all things are poison and nothing is without poison, only the dose permits something not to be poisonous". Six cases of cauda equina syndrome with varying severity were reported to Swedish Pharmaceutical Insurance during the period 1993-1997 [17] (Table 2).

Case	Age	Needle	Punction	Surgery	Local Anesthetic	Dose
1	55 ys	No	L3-L4	T.R.P.	Lidocaine 5%+Glicose 7.5%	100 mg
2	59 ys	No	L3-L4	Toe surgery	Lidocaine 5%+Glicose 7.5%	60 mg
3	48 ys	27G	L3-L4	Hallux valgus	Lidocaine 5%+Glicose 7.5%	100 mg
4	31 ys	26G	L3-L4	Fasciotomy leg	Lidocaine 5%+Glicose 7.5%	100 mg
5	37 ys	27G	L3-L4	Vein varicose	Lidocaine 5%+Glicose 7.5%	120 mg
6	59 ys	No	L3-L4	Hallux valgus	Lidocaine 5%+Glicose 7.5%	75 mg

Table 2: Six cases described by Loo and Irested [17].

All were associated with spinal anesthesia using hyperbaric 5% lidocaine. Five cases had single-shot spinal anaesthesia and one had a repeat spinal anesthetic due to inadequate block. The dose of hyperbaric 5% lidocaine administered ranged from 60 to 120 mg. Clearly, as the authors so appropriately underscore, the diagnosis of direct local anesthetic neurotoxicity is one of exclusion. Care must be exercised to rule out trauma; spinal cord ischemia; infection; compression by hematoma, abscess, prolapsed intervertebral disc and spondylolisthesis; contamination of local anesthetics; and injurious surgical positioning. The authors considered that three of the cases were most probably the result of direct neurotoxicity of 5% hyperbaric lidocaine. Furthermore, in the other three cases, direct neurotoxicity was also probable, but a compressive etiology could not be excluded owing to the omission of appropriate radiological studies. These six cases were reported by reported to the Swedish Pharmaceutical Insurance; however it is clear that the anesthesia sheets were not adequately filled, since 50% of the cases there is no report of the caliber or type of needle. We have been using a macro spinal catheter system for continuous spinal anesthesia in obstetric analgesia [18]. It is a 24-gauge catheter mounted over a 29-gauge spinal needle (Spinocath®). The design eliminates leakage of CSF because the catheter seals the dural puncture hole. A recently published case report described the use of high doses of two local anesthetics with continuous spinal anesthesia [19] (Table 3).

Mixture Local Anesthetic	LA %	Density 37º C	Glucose
1 mL 0.5% Hyperbaric bupicaine+4 mL 0.5% Isobaric bupivacaine	0.5	1.0108 g/mL	1.6%
1 mL 2% Hyperbaric lidocaine+4 mL 2% Isobaric lidocaine	2	1.0160 g/mL	1.6%

Table 3: Evaluation of density of the mixtures [19].

Neurotoxicity of Lidocaine

In the initial study of lidocaine, the authors used the pure solution at concentrations of 0.5% and 2%, and with the addition of epinephrine at concentrations of 0.5%, 1% and 2% [3]. In an excellent Editorial [20], the potency ratio of lidocaine versus bupivacaine is approximately 4:1, and 5% lidocaine should be compared with 1.25% bupivacaine and not 0.5 or 0.75% bupivacaine. Or rather, bupivacaine 0.5% should be compared to lidocaine 2% and not 5%. Are there any reports on permanent neurologic damage after single shot uneventful spinal anesthesia with lidocaine 2%? And the author ends his sentence by saying that he is not aware of any. A study published recently in dogs on increasing doses of hyperbaric lidocaine (5%, 7.5%, and 10%, all in glucose 7.5% in water) demonstrated that the 5% lidocaine concentration is safe to be used in subarachnoid anesthesia and that concentrations higher than 7.5% lead to histological changes in the spinal cord but not in the meninges [21].

As a component of anesthetic solution, glucose was usually used to increase the density of anesthetic solution, which can be great benefit to cycle fluctuations inhibition in clinical anesthesia. Hyperbaric local anesthetics made with glucose produce effectiveness in controlling the level of anesthesia. With its different proportion in mixture, glucose of various concentrations that act as a common component in anesthetic solution is being used for spinal anesthesia. Intrathecal injection of 5% lidocaine could induce spinal nerve sensory impairment, and 10% glucose could worsen the potential neurotoxicity of rats with intrathecal administration of 5% lidocaine [22]. The study suggests that the potential neurotoxicity should be considered when the local anesthetics mixed into high concentration of glucose are used in subarachnoid block.

Articles with Lidocaine Published in the Last Decade

Lidocaine has been used for spinal anesthesia since 1948, seemingly without causing concern. Hip fractures are considered as age related diseases. Spinal anesthesia has become increasingly popular in the setting of hip and knee arthroplasty. It was confirmed that general anesthesia with sevoflurane and spinal anesthesia with low dose lidocaine 5% (75 mg) have comparable effects on hemodynamic changes in patients undergoing hip fracture surgery [23]. However, postoperative vomiting and morphine consumption in patients with spinal anesthesia was lower than general anesthesia [23]. Isobaric lidocaine spinal anesthesia appears to be a safe and effective regimen for same-day ambulation, short-stay TJA, and even outpatient hip and knee arthroplasty [24]. In this prospective small cohort of consecutive patients, all patients were discharged on the day of surgery with rapid return of motor function and time to ambulation. There were no reports of TNS. In a recent study evaluating three different doses of isobaric 2% lidocaine for ventral decubitus surgery, it showed that may have several advantages, especially for outpatient procedures, such as fast recovery, hemodynamic stability, patient satisfaction, with more sensitive than motor block in the lower limbs, fast recovery, no urinary retention, and increased risk for temporary neurological symptoms. In addition, the patient is already anesthetized in the position in which he will be operated (ventral decubitus) [25].

Foot surgeries can be performed with the patient in a position of dorsal decubitus, lateral position or ventral decubitus. Comparing 2% isobaric lidocaine (50 mg) with 0.6% hypobaric lidocaine (24 mg) in the Jack-Knife position provide surgical analgesia with complete block with isobaric lidocaine and without motor block with hypobaric lidocaine for foot surgery. Most importantly, it allowed the patient to remain in this position, providing for better surgical exposure for surgeon and patient safety. In our study, with hypobaric lidocaine all patients went from the operating table to the

stretcher without help, and better degree of satisfaction [26]. Studying the incidence of TNS with different local isobaric anesthetic, showed that the incidence of TNS after spinal anesthesia was much less after levobupivacaine, bupivacaine and articaine than after lidocaine; however it appears that TNS may occur in association with levobupivacaine and articaine [27].

Lidocaine Can and Should be Used

Lidocaine was approved by the Food and Drug Administration (FDA) in 1948 and in time became the local anesthetic typically used when quick onset and regression of spinal anesthesia were desired. A single injection is appropriate for a 1 to 2 hour surgery, whereas multiple injections through a spinal catheter allow for longer surgery time while maintaining a rapid recovery profile. Lidocaine spinal anesthesia facilitates discharge of surgical outpatients within a few hours while decreasing recovery room time and nursing costs for surgical inpatients [28]. Besides the convenience to patients, this also has considerable implications for decreasing the cost of health care. Several case reports of cauda equina syndrome were attributed to the use of the hyperbaric solution (lidocaine and tetracaine) injected through a microcatheter and epidural catheter. Following these reports with continuous spinal anesthesia, single-dose spinal anesthesia with 5% hyperbaric lidocaine was linked with TNS. Studies examining the use of lidocaine have caused many anesthetists to question whether intrathecal lidocaine should still be used.

The first review of the use of lidocaine spinal anesthesia and its association with neurological complications was published in 1969 [9]. This study involved 10,440 patients who received 5% hyperbaric lidocaine, and the dose ranged from 40 to 100 mg, with no patient receiving more than 100 mg. In this series, there were no cases of cauda equina syndrome. TNS shows no evidence for localized nerve damage. The implication is that TNS is 'no big deal' [29]. The ideal local analgesic drug should combine quick action, complete abolition of painful sensation, adequate duration of analgesia, low toxicity, ready diffusability and stability when in solution, with a low incidence of local and general side effects and it should also be an efficient surface as well as an injection agent. Lidocaine remains a popular choice for ambulatory spinal anesthesia. It provides rapid onset of surgical anesthesia for the majority of ambulatory surgical procedures along with a rapid regression of sensory and motor blockade. It appears that total dose of lidocaine, but neither concentration nor volume, is the most important factor in determining both peak level and duration of spinal anesthesia [30]. It is my opinion that a short-acting alternative to bupivacaine for spinal anesthesia is needed. To date, there have been no anesthetics with the characteristics of lidocaine for surgeries lasting 60 to 90 minutes. If a short-acting spinal is indicated, use 2% lidocaine and reduce the total dose to a maximum of 60–80 mg. We should not throw out ''an old champion'' unjustified [20]. There is still no proof that lidocaine toxicity is the explanation of these phenomena. Lidocaine for spinal anesthesia has a remarkable safety record, and in this way it can and should be used in spinal anesthesia.

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