Labat Lecture 2006. Regional Anesthesia: Aspects, Thoughts, and Some Honest Ethics; About Needle Bevels and Nerve Lesions, and Back Pain After Spinal Anesthesia

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Dear Mr. President, ASRA Board Members, and colleagues: it is a great honor to receive the Gaston Labat Award; I am much grateful for this. And thank you, Joseph Neal, for a fine introduction.

Regional anesthesia (RA) has been quite popular among my Swedish colleagues, especially in the 1970s and '80s. However, during the 1990s the interest and use of RA decreased, to some extent due to organizational changes in hospital care, emphasizing that time is money, and as it for many colleagues is quicker to give a general anesthesia (GA) than RA. This attitude has also influenced clinical education in anesthesiology, so unfortunately, today there are not so many RA-enthusiastic anesthesiologists in Sweden. Therefore, I am glad to be a member of the American Society of Regional Anesthesia (ASRA), because it keeps me in contact with many of ASRA's clinically and scientifically skilled and enthusiastic members. Fortunately, we also have several colleagues of that kind in the European Society of Regional Anesthesia (ESRA) (and of course in the other societies of regional anesthesia and pain medicine around the world).

My interest in local anesthetics and regional anesthesia was induced by Associate Professor Karl-Gustav Dhunér, head of the Anesthesia Department at Sahlgren's University Hospital in Gothenburg (1960-1980), and he worked hard to make his younger colleagues understand the advantages of regional anesthesia compared with general anesthesia. A further important stimulus for my interest in regional anesthesia was Professor Alon P. Winnie, for whom I had the honor to do clinical work in Chicago during the period of 1985 to 1986. Dhunér also initiated my Ph.D. thesis, "Neural Complications of Regional Block" which contained 5 studies, four on neural complications in connection with peripheral nerve blocks[1-4] (partly as a test of the safety in D. Moore's statement: No paresthesia, no anesthesia![5]), and one study on the use of catheter technique for axillary plexus block.[6]

My interest in RA and pain management, and also my work at Astra Pain Control, later AstraZeneca, have resulted in many stimulating contacts, travels, conferences, and lectures. However, there was also criticism and many comments regarding my studies, especially on the role of paresthesia and needle bevels for nerve lesions.

The topics of today's Labat lecture is my choice, and I will focus on two areas which have caused much discussion and are of special interest to me: (1) the role of the injection needle's bevel type for nerve fascicle lesions; and (2) transient neurologic symptoms (TNS) after spinal anesthesia — is it really a sign of local anesthetic (LA) neurotoxicity?

But before entering those topics, I have a short comment about who actually introduced the catheter technique for continuous peripheral nerve blockade. The first description of continuous brachial plexus block technique was probably published by the American orthopedic anesthesiologist P.F. Ansbro in 1948. However, Ansbro did not use a catheter; he described how he managed to top-up and prolong a supraclavicular plexus block via an indwelling needle passed through a cork that was fixed with tape to the patient's chest.[7] The use of a flexible catheter (inserted into the brachial plexus sheath) was actually first described by Alon P. Winnie and Vincent J. Collins in 1964.[8]

In October 1975 I woke up one morning (without knowledge about the above-mentioned references) with the idea that the neurovascular sheath invites the use of a catheter instead of a needle, at least for

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axillary block. I tested the idea the same day and found that it worked well. This initiated a clinical evaluation study, published in 1977, based on 137 cases that had an axillary block via a 47-mm long Venflon (nr 100, AB Viggo, Helsingborg, Sweden) intravenous, over-the-needle catheter inserted into the neurovascular sheath and LA doses of around 40 mL.[6]

Besides realizing the truth of the old statement that "Nothing is new under the sun," I am nowadays aware that the catheter-based continuous peripheral nerve block technique was first described by Alon P. Winnie and Vincent J. Collins, Chicago, in 1964.[8]

Now, back to the first of the main topics:

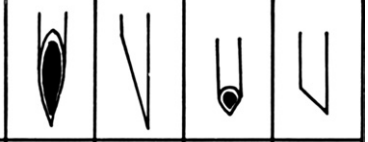
Role of the Injection Needle's Bevel Type for Nerve Fascicle Lesions

The risk of causing a nerve fascicle lesion is less with short-beveled injection needles than long-beveled, so we recommend the use short-bevel needles for peripheral nerve blocks. Selander, Dhunér, and Lundborg, 1977.[2]

I bring this up because there was a contradictory report by Rice and McMahon in 1992,[9] based on a different study design and definition of the word "frequency." The discussions regarding the conclusions of these two studies call for a description of their different designs. In short: we looked for the frequency (risk) of fascicular lesion after nerve puncture in relation to the type of needle bevel, while Rice and McMahon deliberately punctured the major fascicle in all tested nerves, and then evaluated the sum of various types of injury in relation to the needle bevel.

We studied the incidence of fascicle injury in 60 rabbits after needle penetration of the rabbit sciatic nerve in vitro (i.e., fixed in a groove on a cork-plate), or in situ, with intraneural injection of 0.05 mL fluorescent tracer. We aimed at the largest fascicle with either a long-beveled (LB; 12°-15°) or a short-beveled (SB; 45°) needle, diameters 0.4 or 0.7 mm in vitro, and 0.4 mm in situ, and with bevel parallel or transverse to the nerve fibers. We found that the fascicles were quite resistant and tended to slide or roll aside, and fascicular puncture was significantly less common with the short-bevel needle than with the long-beveled. The frequency of fascicular lesion after in vitro neural puncture, aiming at the largest fascicle, is shown in Table 1.

Table 1. Fasicular Injury after Needling of Isolated Nerve Preparations

	A	B	C	D
	Long bevel		Short bevel	
				
n	15	15	15	15
Fascicular injury	13	14	8	8

The degree of injury after penetration with the long-beveled needle was worst with the bevel transverse to the nerve axis, while the injury caused by the short-beveled needle was less dependent on the bevel orientation. But, as the risk of nerve fascicle injury was significantly lower with the short-bevel needle we recommended the use of short-beveled needles for peripheral nerve blocks.

Contrary: *Long-beveled needle is less risky than short-beveled.* Rice and McMahon 1992.[9]

About 15 years later, Rice and McMahon published an experimental study and questioned our conclusion by stating ". . . we cannot confirm the suggestion of Selander, Dhunér and Lundborg that a 45° beveled needle less frequently produces fascicular damage . . . Indeed, the results of our study suggest that there may be grounds for recommending the use of long beveled needles, especially if the bevel is aligned parallel to the nerve fibres." They based this conclusion on the results of an experimental study on rat sciatic nerves.[9]

Rice and McMahon exposed the sciatic nerves at mid-thigh level in 56 rats under GA and impaled the large, single fascicle of all nerves with one of the tested needles (LB, 12°, 23-gauge; or SB, 27°, 22-gauge) at a 45° angle, with the bevel parallel or transverse to nerve fibers, and left it undisturbed in the nerve fascicle for 10 minutes. The nerves were then examined at day 1 (n = 19), or after 7 (n = 19) or 28 (n = 18) days.

In the histological section, the authors evaluated:*(1) intraneural disruption, defined as disruption of internal elements of the nerve, scored 0 to 5 (0 no disruption, 5 complete loss of identifiable intraneu-

*Rice and McMahon also studied the degree of extravasation of Evans Blue in the sural skin of the operated (?) limb, as a sign of neurogenic edema; and withdrawal time of hind limb flexion after hot water immersion, to assess the reflex arc and its spinal processing. However, these factors mainly concerned postacute regeneration and function, which both were more disturbed after fascicular puncture with the short-beveled needle.

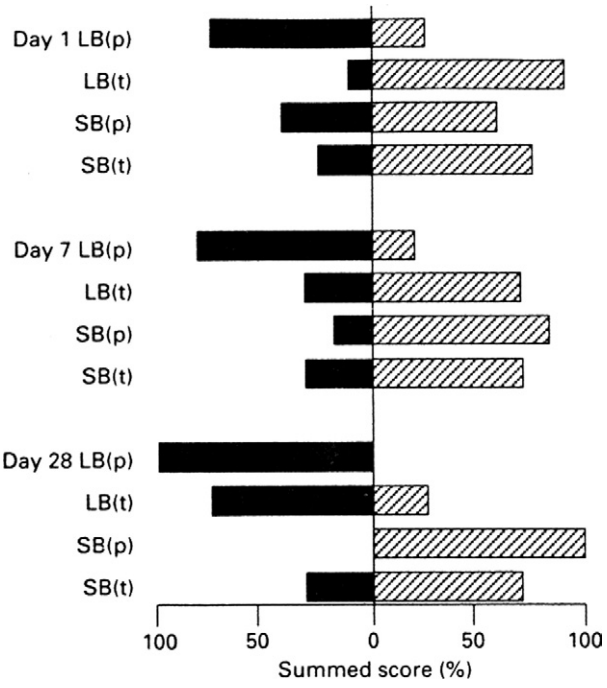


Fig 1. Summed nerve injury scores obtained from rat sciatic nerves examined immediately after (Day 1) and at 7 (Day 7) and 28 days (Day 28) after nerve impalement by long and short beveled injection needles aligned parallel (p) or transverse (t) to the direction of nerve fibers. The x-axis represents the percentage of summed nerve injury scores for each type of needle and each time point. The percentage of high scores (hatched bars) is shown to the right of the midline and the percentage of low scores (solid bars), to the left. LB, long bevel; SB, short bevel. Reprinted from Rice and McMahon.[9]

ral elements at the site of injury), axonal degeneration, disorganized pregeneration of nerve fibres; (2) evidence of axonal degeneration distal to the insertion site at day 7 and 28, scored as "Yes" or "No" *(i.e., 1 - 100% = yes; 0 = no)*; (3) evidence of disorganized regeneration of fibres, at day 7 and 28, also on a present or absent basis; and (4) "Summed nerve injury score." The data from observations of intraneural disruption, axonal degeneration and disorganized regeneration, in addition to observations of epineural disruption (without definition) were used to calculate the summed nerve injury score (Fig 1).

Rice and McMahon opened the discussion of their article with: "The results of these experiments suggest that when nerve fascicles are impaled by commercially available injection needles (of similar diameter), lesions occur less frequently, are less severe and are repaired more rapidly if they are induced by an LB needle compared with an SB needle." Besides, their short-bevel needle had a bevel angle of 27° (it was also 13% thicker than the long-bevel needle), so of course, they could not confirm that a 45° beveled needle less frequently produces fascicular damage than a 12° to 15° beveled needle!

The central difference between these two studies was that we investigated the *frequency of fascicular injury* after test-needle puncture of the rabbit sciatic nerves, while Rice and McMahon *punctured the major fascicle of all rat sciatic nerves, and then evaluated the sum of various types of neural lesions* (Fig 1). Were these authors not aware of the difference between frequency and degree? I commented on this and their study design in a "Letter to the Editor."[10]

Rice and McMahon answered that they did not question Selander et al's conclusion, and that "we described the influence of needle bevel upon the severity and consequences of intrafascicular lesions, should accidental fascicular penetration occur." They also said, "Selander has misunderstood our use of the term frequency. Of course, all fascicles were impaled; this was the aim of the study. The term frequency was used to describe the incidence of observed lesions following fascicle injury."[10] So, obviously they did not use the term "frequency" as we did; apparently Rice and McMahon focused on the frequency of different degrees of nerve fascicle lesions. That was good to know.

Many colleagues accepted Rice and McMahon's conclusion that Selander et al.'s recommendation for short-bevel needles was wrong. However, I soon realized that many of them had not read the Rice and McMahon article properly, and therefore missed that these authors had punctured a fascicle in every sciatic nerve, causing a 100% incidence of fascicular lesion with both needles. Nor had these colleagues noted the fact that Rice and McMahon used the word "frequency" for describing the appearance of different degrees of nerve fascicle lesions, which was highest after puncture with a short bevel needle.

In fact, we had also noticed that the degree of nerve lesion varied with the needle bevel, and our findings regarding degree of fascicle lesion actually basically agreed with Rice and McMahon's results on day one.

To look at the positive outcome of this controversy, these two experimental studies showed: (1) significantly less risk of nerve fascicle puncture with short-bevel needles;[2] and (2) if fascicular puncture, short-bevel needles may cause more lesions than long-bevel needles.[9]

And, I think both teams agree on this overall conclusion: nerves should be handled with care!

Transient Neurologic Symptoms After Spinal Anesthesia—Is It Really a Sign of LA Neurotoxicity?

This topic has been much discussed and many clinical and experimental studies have been performed. No definite etiology for transient neurologic symptoms (TNS) has yet been presented, although many believe in a neurotoxic origin. However, I suggest an alternative explanation, which will be presented after reviewing the background of the current label.

In 1993, Schneider and coworkers described 4 patients with a transient lumbosacral pain syndrome that appeared 1 to 20 hours after recovery from subarachnoid anesthesia with 5% hyperbaric lidocaine.[11] The pain was described as a dull ache, occasionally decreasing when the patient was up walking around, it responded well to nonsteroidal anti-inflammatory drugs (NSAIDs) and resolved spontaneously within 2 to 5 days. There were no neurological dysfunctions.

However, the authors labeled the pain as "radicular" and suggested that the syndrome was due to neurotoxic effects of the 5% hyperbaric lidocaine solution; they called it "transient neurologic toxicity" (TNT). It was hypothesized that the flattening of the lumbar lordosis in the lithotomy position resulted in stretching of the L5 to S1 roots which caused partial ischemia and abnormal sensitivity to neurotoxic effects of 5% lidocaine.

Besides the name "transient neurologic toxicity (TNT)," other labels such as "transient radicular irritation (TRI)" and "transient neurologic symptoms/syndrome (TNS)" were introduced. Today, TNS is the name mostly used.

The neurotoxic hypothesis was obviously based on cases of cauda equina syndrome (CES) reported after continuous spinal anesthesia with microcatheters and 5% hyperbaric lidocaine, but also on experimental studies confirming a relatively high neurotoxic potency of lidocaine. However, that CES and TNS both may appear after spinal anesthesia with lidocaine does not imply that both are of a neurotoxic origin.

Characteristics of the TNS Syndrome

Typically, the TNS pain appears 1 to 20 hours after recovery from the spinal anesthesia. The lidocaine dose was around 75 mg, and the spread of anesthesia was normal, on an average to segment T10 or higher. The majority of patients were women who underwent short gynecologic procedures in the lithotomy position, but also after other treatments in the supine position.[12] The dull aching pain is symmetrically localized in the lumbar area and buttocks, sometimes radiating to the thighs or even forelegs. Many patients described less pain when moving around than when resting in bed, and analgesics such as NSAIDs and common opiates provided good pain relief.[11,13] And, most important there were no signs or symptoms of neurological dysfunction: reflexes were normal, there was no sensory or motor deficit, and no disturbance in bladder or bowel functions.

Which Local Anesthetics May Cause TNS?

A number of prospective clinical studies have been performed showing a TNT-TRI-TNS incidence varying between 0% and 37% after lidocaine spinal anesthesia.[14-21] These studies also showed that the syndrome occurred independently of the osmolarity of the lidocaine solution,[15] and, surprisingly, it was equally frequent after lidocaine 0.5%, 1%, 2%, and 5%.[16-19]

However, TNS is not limited to lidocaine. Hiller and Rosenberg reported a 30% incidence of TNS after spinal anesthesia with 4% mepivacaine,[13] while Liguori et al. found no case of TNS after spinal anesthesia with 1.5% mepivacaine for knee arthroscopy, but a 22% incidence with 2% lidocaine.[22] Other clinical studies indicated that the risk of TNS was considerably lower with prilocaine than lidocaine,[23-26] while Östgaard et al. found no difference between prilocaine and lidocaine.[26]

Several studies show that spinal bupivacaine 0.5% only rarely, if at all, causes this syndrome.[14,15,17,19,23,27] A prospective epidemiologic study by Freedman et al. showed that the incidence of TNS was significantly higher with lidocaine than with bupivacaine, and that the lithotomy position was a considerable TNS risk factor.[28]

As the 1.5% and 5% lidocaine produce equally effective spinal anesthesia, Drasner expected that the risk of neurotoxic injury should be minimized by using a low concentration and dose, although this did not seem to affect the incidence of TNS.[29]

What do the Terms Neurologic, Radicular and Neurotoxic Stand For?

Neurologic–Radicular. Although the distribution of the TNS pain may be consistent with rhizopathy involving nerve roots L5 to S1, the symmetric appearance without neurological symptoms does not fit the term "radicular." A radicular pain, e.g., due to a herniated disk is usually described as sharp, tender, and shooting, frequently with some degree of dysesthesia and paresthesia. It is exacerbated by activities and positions that cause addi-

tional stretching of the nerve, and sensory, motor, and reflex deficits are frequently present.[30,31]

The nonneurogenic character of the TNS pain, and the lack of sensory and motor deficits and abnormal reflexes, indicate that the terms "neurologic, radicular, and deficit" are not correct. The short duration of TNS and good pain relief by ordinary analgesics also contradict a radicular/neurogenic nature. Carpenter noted this in an editorial: "although repeatedly defined as neurologic symptoms, no evidence is provided to support a neurologic origin."[32]

Neurotoxicity

Cauda Equina Syndrome

The tragic cases of cauda equina syndrome (CES) after continuous spinal anesthesia clinically demonstrated that 5% lidocaine has a neurotoxic potential,[33] and the neurotoxic potency of this preparation has been experimentally confirmed.[34-36] The CES obviously depended on insufficient dilution of the 5% lidocaine in the CSF due to the very slow injection allowed by the microcatheter. Restricted spread of anesthesia indicated that the catheter had deviated into the caudal part of the dural sac; to compensate for that, very high doses of lidocaine 5% (up to 300 mg) were given. Consequently, the cauda equina was exposed to an unduly high dose of lidocaine 5%, and this caused neurotoxic injury.[37]

TNS clearly differs from what was described for the neurotoxic CES: in all CES cases, symptoms were recognized directly after the spinal anesthesia had worn off as a patchy, asymmetric numbness or analgesia in the saddle area, asymmetric leg weakness, loss of reflexes, and bladder and/or bowel incontinence, while pain is not a typical symptom. With time the condition may regress, but this is usually a matter of several months, and not infrequently some dysfunction becomes chronic.[30,33,38]

Neurotoxic Potency

Generally, the neurotoxic effects of a drug, e.g., a local anesthetic, depend on its neurotoxic potency, its concentration, the duration of exposure, and the susceptibility of the exposed tissue.[34,35,39,40]

That lidocaine has a relatively high neurotoxic potential has been shown in several experimental studies.[35,36,40-43] Malinovsky et al. studied neurotoxic effects in rabbits 7 days after intrathecal injection of 0.2 mL lidocaine 5% or ropivacaine 0.2%, 0.75%, 1%, and 2%. Lidocaine caused paralysis and considerable histopathologic changes, while none of the ropivacaine groups showed clinical or microscopic signs of neurotoxicity.[41] Kasaba et al. compared the neurotoxic effects of 7 different LAs on cultured neurons from the freshwater snail *Lymnaea stagnalis*. Of the amide local anesthetics, mepivacaine showed the lowest neurotoxicity in this model.[43] Kishimoto et al. studied the spinal neurotoxic effects 3 days after subarachnoid injection of 2.5% lidocaine or 2.5% prilocaine in rats. They found that the two drugs were equally neurotoxic, and their conclusion was that these results "may indicate that neurologic injury (e.g., cauda equina syndrome) and TNS are not mediated by the same mechanism."[42]

Neurotoxic Effects are Concentration-Related

A strong argument against the neurotoxic explanation for the TNS is its lack of concentration/dose dependency.[16,17] In a typical concentration/dose-response curve, there are two ranges where a toxic response would show very little variation with concentration: the low, ineffective, atoxic range and the highly effective and possibly toxic range, corresponding to the two relatively horizontal parts of the curve, and, between these areas, there is the concentration dependent range.

Experimental studies indicate the neurotoxic threshold concentration for lidocaine appears between 2% and 3% (20-30 mg/mL), when injected around a nerve[44]; i.e., a lidocaine concentration higher than 2% may, if maintained for long enough, induce toxic lesions to nerve tissue.[45] Normally, the concentration of the LA will decrease rather rapidly after injection due to dilution with the cerebrospinal fluid (CSF) or interstitial fluid and absorption into the blood. This means that local toxic reactions only can be expected when the LA concentration remains sufficiently high during a sufficiently long period, or if the neural tissue is abnormally sensitive, e.g., due to ischemia or a neurologic disease.

Lidocaine Concentrations in CSF

The CSF volume in the lower thoracic-lumbosacral section of the spinal canal is estimated to be 30 mL to 80 mL in adults, and dermatomal spread of anesthesia has been shown to correlate to the dose and CSF volume.[46,47] Patients presenting with TNS usually received around 75 mg lidocaine intrathecally, *i.e.,* 1.5 mL of the 5% solution, and all described TNS cases presented a normal and expected spread of anesthesia (typically T8-S5). If we approximate the actual CSF volume to be 50 mL and assume an even distribution in this CSF volume, the expected initial lidocaine concentration would be 1.5 mg/mL (0.15%), which is well below the estimated neurotoxic level.

That this calculation is relevant was confirmed by studies in humans[48,49] in which the lidocaine con-

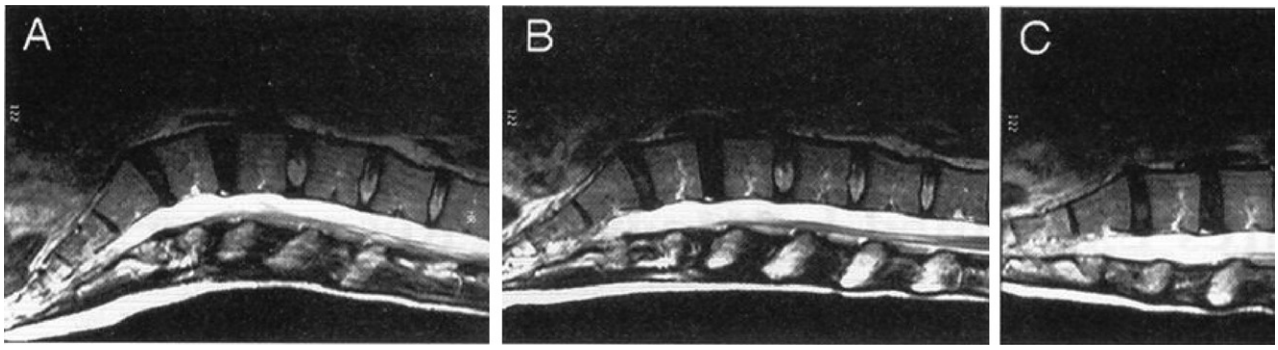


Fig 2. T2-weighted magnetic resonance images showing midsagittal slices of the lumbar spine from a 34-year-old woman. Images were obtained in the supine position with (A) knees straight, (B) slightly flexed, and (C) fully flexed. The maximum downward angle of the lumbar spinal canal in the cephalad direction against the plane of the magnetic resonance platform was 14° in (A), 7° in (B), and 0° in (C), the fully-flexed leg position. From Hirabayashi et al.[60]

centration in CSF was assessed in samples taken via an intrathecal catheter, which also was used for the lidocaine injections. After injection of 150 mg of the 5% hyperbaric lidocaine solution via a catheter inserted at the L4-5 interspace (and advanced cranially 17 cm to 20 cm, aiming for the T11-12 level), Mörch et al.[48] found lidocaine concentrations in the CSF two minutes after injection varying between 0.2% and 1.45%, followed by a rapid decrease to 0.03% to 0.25% in 30 minutes. They also noted an average analgesic threshold concentration of lidocaine at 250 μg/mL, or 0.025%.

Van Zundert et al.[49] injected 70 mg lidocaine intrathecally in patients via a catheter inserted 3 cm to 4 cm at the L3-4 level. They used 5 different concentrations (0.5%, 1.0%, 2.0%, 5.0%, or 10%) and achieved a median upper spread to T4-5. The highest lidocaine concentration at 5 minutes after injection of the 4 weaker solutions varied between 0.2% to 0.37%, while the 10% solution resulted in a top CSF lidocaine concentration of 0.57% at 5 minutes, which was statistically different from the other 4. All concentrations then decreased rapidly, and were similar at 20 minutes, and around 1 mg/mL (0.1%) at 30 minutes.

There were no reports on TNS-type pain or neural complications in these 2 studies. Thus, the CSF lidocaine concentration in the TNS cases has been too low to induce neurotoxic complications.

In summary, the lack of neurologic symptoms and signs in the TNS cases and the independency of the LA concentration contradict the possibility of a neurotoxic cause.[27,42,50-53]

Alternative Explanation: Myofascial Stress-Related Pain

The TNS syndrome, as it is described, seems more likely to be a myofascial pain syndrome caused by the extreme flattening of the lordotic arch due to the effective muscle relaxation of some spinal anesthetics. The key is the stretching of ligaments, fasciae, and muscles, but *not* of the cauda equina and lumbosacral nerve roots! In fact, a myofascial etiology of the TNS syndrome was suggested by Hartrick in 1997, who also objected to the use of the terms "neurologic" and "radicular."[54] Similar thoughts were presented in 1998 by Naveira et al.,[55] Corbey and Bach,[56] Denny and Selander,[57] Selander,[58] and in the Cochrane databased report by Zaric et al.[27] Interestingly, Holmdahl described a discussion at the Karolinska Hospital in the late 1940s where Gordh suggested that the back pain, sometimes seen after spinal anesthesia, was caused by the flat supine position on the operating table, due to the concomitant muscle relaxation.[59]

Effect of Position

The supine position results in a flattening of the lumbar lordosis, and this will be more pronounced in the lithotomy position, as described by Schneider et al. in the first TNT report.[11] The flattening of the lumbosacral arch leads to extension of dorsal longitudinal ligaments, tendons, muscles, and fascia. So, rather than extending the longer spinal roots of the cauda equina, it is the dorsal myofascial structures that may be subject to extraordinary stress.

This hypothesis is strongly supported by a recent MRI study involving female volunteers, which showed that the lithotomy position causes a significant flattening of the lordotic arch.[60] Due to the prolongation of the dorsal curvature, the flattening will cause stress on the corresponding spinal muscles, ligaments, and joint capsules (Fig 2). The ventral movement of the cauda equina during the flattening of the lumbar lordosis, as seen on their MRI

Table 2. Spinal Anesthesia and Motor Block

Study	% Complete Motor Block (Bromage 4)			Statistical Significance
	Lidocaine 5%	Mepivacaine 4%	Bupivacaine 0.5%	
Hiller and Rosenberg, 1997[13]	—	96	81	$P < .001$
Pitkänen et al., 1984[62] (Gr 1 ND)	90	100	40	$P < .01$*
Pitkänen et al., 1984[62] (Gr 2)	90	70	60	—
Salmela and Aromaa, 1998[63]	97	87	57	$P < .01$† $P < .05$*

NOTE. Percent complete motor block (Bromage 4) after spinal anesthesia with hyperbaric lidocaine 5%, mepivacaine 4%, and bupivacaine 0.5%.

Abbreviations: Gr 1 ND, group 1 nondependent leg, slow injection (2 minutes), lateral position for the first 15 minutes; Gr 2, group 2, fast injection (≤ 10 seconds), then immediate supine horizontal position.

Data from Hiller and Rosenberg,[13] Pitkänen et al.,[62] and Salmela and Aromaa.[63]

*Mepivacaine vs. bupivacaine.

†Lidocaine vs. bupivacaine.

pictures, may rather represent a relaxation of the cauda equina, not an increased tension.

Effect of Spinal Anesthesia

Spinal anesthesia with lidocaine results in a profound motor block in anesthetized segments,[61] and the loss of the protective muscular tension can lead to a supramaximal flattening of the lumbar lordosis, especially the lithotomy position. This may cause an abnormal tension in dorsal ligaments, joint capsules, and muscles, which later may induce inflammatory reactions in the lumbosacral area. The corresponding pain would be of a nociceptive nature and respond well to ordinary analgesics and NSAIDS. After a few days, these inflammatory reactions heal, and the pain disappears.

However, all patients will not suffer from TNS, because its appearance is probably related to individual variations in factors such as the patient's physical constitution, lumbosacral configuration, spread of anesthesia, duration in supine or lithotomy position (and possibly the quality of the operating table mattress).[13]

Resemblance to Posttraining Stiffness

The resemblance to the aches felt after heavy physical exercise, especially in an untrained person, is striking. Like the TNS pain, it has a free interval, the pain decreases after "warming up," it is relieved by NSAIDs and other analgesics, and it wears off within a few days.

Why Is TNS Not Seen After Spinal Anesthesia With Bupivacaine?

Most likely because bupivacaine provides a less profound motor block. It has been found that the motor block in spinal anesthesia with bupivacaine 0.5% is significantly less than with lidocaine 5% and mepivacaine 4% (Table 2).[13,62,63] These studies also show that the onset of motor block with lidocaine and mepivacaine is considerably faster than with bupivacaine. Together, these findings explain why the incidence of TNS after spinal anesthesia with 4% mepivacaine is similar to that with lidocaine. However, regarding a lower risk of TNS prilocaine: I have not found any reports stating that prilocaine provides less motor blockade than lidocaine and mepivacaine.

Conclusion: Rename to TLP!

As I see it, the TNS syndrome is not of a neurologic or neurotoxic nature. Instead, the clinical picture indicates that TNS is a myofascial pain condition, related to a supramaximal flattening of the lordotic arch. Therefore a more suitable name would be "transient lumbar pain," or TLP, if another abbreviation can be accepted.

How Could We Avoid TLP . . .

To reduce the risk of postoperative lumbar pain after spinal, and also general anesthesia, especially with the patient in supine or lithotomy position, the positional stress on the lumbosacral myofascial structures should be minimized. A simple way to do that is to place a suitable support (e.g., a pillow) under the patient's lower back to reduce flattening of the lumbosacral lordosis,[59,64] especially when using local anesthetics providing profound motor block. It would be interesting to see a clinical study on this!

Some Aspects of Future Regional Anesthesia

The regional anesthesia concept has many advantages: it provides selective anesthesia or analgesia with limited motor block and reduced need for

opioids, and this will minimize disturbing side effects. A local anesthetic neural blockade used in combination with general anesthesia (GA) for surgery, will reduce the central alarm degree; therefore the patient may sleep well at relatively low doses of GA drugs. And the use of RA for surgery has a lower rate of serious complications than general anesthesia.[65] It also provides a quicker rehabilitation and shorter hospital stay with effective postoperative pain relief, and again, less dependency on opioids and other analgesics.

Even if today's local anesthetics are sufficiently effective and safe, there are several factors that could be improved, e.g., toxicity, afferent pain, fiber selectivity, degree of motor blockade, and duration. And the way of administration: do we always need to inject LAs in the future? In fact there is need for further research regarding alternative mechanisms for more selective blockades, better control of duration, reduction in both systemic and neurotoxicity, and methods for localizing the nerves to be blocked. Better pain-selective LAs would improve treatment of both postoperative and other types of pain. And what about a reversal agent that could eliminate the block? There are also other interesting qualities of the LAs, such as antihypercoagulation and anti-inflammatory effects, which may be further analyzed for new developments in these areas.

Unfortunately, today there seems to be only a vague interest for such projects in the pharmaceutical industry. Hopefully it will change with time, as this would offer regional anesthesia and pain management a more stimulating, effective, and valuable future.

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