Pain from cutaneous nerves can be intense. This is more commonly experienced when the saphenous nerve is left unanaesthetised for ankle and foot surgery in spite of a functioning sciatic nerve block that innervates the vast tissue of the foot. It is experienced likewise when the sural nerve is left unanaesthetised for lateral ankle surgery or just the removal of a fibular plate. Performing rescue blocks for breast lumpectomies, mastectomies and axillary lymph node dissections with nerve blocks of the lateral or anterior cutaneous branches of the intercostal nerves, despite wound infiltration by the surgeon, provides this experience also.

Regional anaesthesia of the cutaneous nerves of the superficial cervical plexus for medial clavicular fracture repair, or for shoulder surgery where the incision includes the skin more medial than covered by the interscalene nerve block, will also show the intensity of pain from cutaneous nerves. Additional anaesthesia of the intercostobrachial nerve after elbow surgery, the superior cluneal nerves, the subcostal nerve or the iliohypogastric nerve after hip surgery, and the medial femoral cutaneous nerve or saphenous nerve for anteromedial knee surgery or anterior tibial surgery respectively, are all instances where the analgesic effect of cutaneous nerve blocks can be the defining cause of having comfortable patients postoperatively, with less hours spent in the postoperative care unit.

One possible explanation for the effect of cutaneous nerve blocks is that, what have been labelled cutaneous nerves, contrary to the immediate intuition, may innervate the peristeum where no muscles cover the skeleton e.g., the saphenous nerve for the anterior tibia and the superficial cervical plexus for the clavicle. Additionally some cutaneous nerves may innervate joint capsules, e.g. the saphenous nerve at the medial ankle and the medial femoral cutaneous nerve at the medial knee. However, as mentioned above, even in cases where no deeper tissue is innervated, as with the lateral cutaneous branches of the intercostal nerves in breast surgery, the intensity of postoperative pain solely originating from cutaneous nerves can be easily observed. It is particularly recognizable when pain is resolved completely after the performance of a cutaneous rescue block postoperatively.

While cutaneous nerves have a part in mediating acute postoperative pain, they are the most frequent cause of post-surgical chronic neuropathic pain. The inescapable neurotomy of the surgical incisions, or the unintentional trauma by the surgical tools, consistently produce a procedure-dependent ratio of patients with chronic neuropathic symptoms such as disabling allodynia and hyperalgesia in the involved cutaneous areas. Historically, regional anaesthesia of the involved cutaneous nerves had only little relevance in the treatment of these chronic conditions. However, the increasing accessibility to non-neurodestructive technologies, such as percutaneous cryoneurolysis, in conjunction with improving point-of-care ultrasound equipment and the continuously ongoing scientific work into the anatomy of cutaneous nerves, changes this. Ultra-selective diagnostic nerve blocks of cutaneous nerve branches play an indispensable role in the treatment of these patients. Even in the absence of the ability to offer an interventional treatment, the diagnosis of cutaneous nerves as the cause of chronic pain has a significant potential to avoid improper secondary or tertiary surgical procedures when the pain is erroneously believed to have a different origin and a surgical solution.

The importance of cutaneous nerves in chronic postsurgical neuropathic pain should be of little dispute. Knowledge of the anatomy and ultrasonographic appearance of cutaneous nerves is essential to interventional treatments and help patients avoid unnecessary surgery. However, whether or not cutaneous nerve blocks should be of relevance to the regional anaesthetist in regard to acute postoperative pain, depends on the objective of the postoperative pain treatment. If future improvements towards opioid-free, painless, fast track procedures are an ambition, then cutaneous nerves and knowledge of cutaneous nerve blocks seem like an unavoidably part that equation.

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These symptoms are typically evident after recovery from the subarachnoid block.

Transient radicular irritation syndrome, renamed as transient neurological toxicity or transient neurological symptoms (TNS), is another neurological syndrome that can follow spinal anesthesia. It has been described as a non-permanent complication in which patients complain of pain radiating from the lower back to the dorsolateral thighs and calves. It occasionally presents with dysesthesia in the buttocks and legs without motor symptoms starting less than 24 hours after spinal anesthesia. It persists for more than 24 hours and is self-limiting, typically resolving within one week.

Numerous possible etiological factors have been proposed for both syndromes. One common hypothesis is that both CES and TNS are direct neurotoxic effects of local anesthetics (LA), a greater incidence correlating with higher LA concentrations. Initially, only lidocaine was blamed; subsequently, most LAs have been implicated in CES and TNS. It has also been suggested that the hyperosmolality of hyperbaric solutions could be related to increased axon membrane permeability, but this hypothesis has been challenged by others. The lithotomy position during surgery has also been implicated in stretching the sacral nerve roots and increasing the vulnerability of those nerves that were in contact with LA. The use of spinal microcatheters was proposed as an etiology through uneven distribution of LA within the cerebrospinal fluid (CSF), exposing some nerve roots to high concentrations of LA and enhancing the neurotoxic effect. The ‘jet effect’ of injection through these catheters has also been mentioned. Finally, direct or indirect lesions in the spinal cord, compression or ischemia of the spinal cord, contamination of LAs, and pre-existing neuropathology have also been included in the list of potential CES etiologies.

However, the true etiology of these neurological conditions after neuraxial anesthesia has never been ascertained conclusively. Despite avoiding lidocaine, high concentrations of other LAs have been implicated in CES and TNS. It has also been suggested that the hyperosmolality of hyperbaric solutions could be related to increased axon membrane permeability, but this hypothesis has been challenged by others. The lithotomy position during surgery has also been implicated in stretching the sacral nerve roots and increasing the vulnerability of those nerves that were in contact with LA. The use of spinal microcatheters was proposed as an etiology through uneven distribution of LA within the cerebrospinal fluid (CSF), exposing some nerve roots to high concentrations of LA and enhancing the neurotoxic effect. The ‘jet effect’ of injection through these catheters has also been mentioned. Finally, direct or indirect lesions in the spinal cord, compression or ischemia of the spinal cord, contamination of LAs, and pre-existing neuropathology have also been included in the list of potential CES etiologies.

In 1999 and again in 2008, our team described a tubular structure enveloping single or groups of intrathecal nerve roots. At the time, we named these tubular structures ‘arachnoid sleeves’ and proposed a new anatomical hypothesis to explain the post-spinal anesthesia complications. Recently we reported after experimental research that is possible to inject a spinal needle - in particular its distal opening - completely inside this tubular subarachnoid structure and, by extension, to inject LA preferentially into it. We extracted the full spinal dural sac and its contents from each of four fresh, unembalmed, cryopreserved human cadavers. The full samples were then immersed in a similar saline solution and different needle types were deliberately inserted into the nerve roots under direct vision to simulate lumbar punctures, penetrating the cauda equina root nerves traveling almost vertically downwards.

After using 27G and 25G Whitacre pencil-point spinal needles, simulating clinical practice for lumbar punctures and spinal anesthesia, we performed deliberately needle insertions on the nerve roots of Cauda Equina. The positions of the needle tips and their orifices relative to the arachnoid sleeves were recorded under direct vision of the cauda equina roots samples. The nerve root and surrounding tissue offered no resistance to needle penetration. During the dissection, the translucent arachnoid sleeves in the specimens could not be identified with the naked eye. However, they could be easily visualized under stereoscopic microscopy.

The translucency of the arachnoid sleeves made it possible to visualize in detail whether the needle tip had entered the compartment they enclosed and to verify the position of the needle tip opening. In cases where the tip of the needle was introduced into the parenchyma of the nerve root itself, the complete needle tip opening was not consistently observed inside the arachnoid sleeve.

The photographic images allowed us to determine whether the entire needle tip opening or only part of it was located within the arachnoid sleeves. This was observed in high number of samples after using the both needle type. In the remaining punctures, the needle orifices were not observed inside the arachnoid sleeve, either because the needle tips were positioned inside the nerve root parenchyma, or because the orifices were outside the arachnoid sleeves.

This in vitro study of human cauda equina nerve roots under stereoscopic microscopy showed that the distal needle orifice of a 27- and 25-G Whitacre pencil-point spinal needle can be placed inside an arachnoid sleeve.

The arachnoid mater is a complex structure that includes the arachnoid layer and trabeculae arachnoid. The arachnoid layer occupies the internal surface of the spinal dural sac. Inside it is the subarachnoid space filled with CSF and occupied by spinal cord and nerve roots. It is a semipermeable structure formed by cells tightly joined together. In contrast, the arachnoid sleeves have their origin in the trabecular arachnoid, which is formed by interlacing collagen fibers and is permeable. In vivo, the arachnoid sleeves could be filled with a small amount of CSF, considering their permeability, with a larger volume of CSF inside the subarachnoid space but outside the arachnoid sleeves. Even using stereoscopic microscopy, it was challenging to distinguish the arachnoid sleeves as structures independent of the nerve root because, being translucent, they were in close contact with the nerve root surfaces, though they did not adhere to them.

We now know that the entire needle tip orifice can be placed inside arachnoid sleeves and an LA thus injected into it. Although we cannot say with certainty that placing the orifice of the needle and injecting LA is the cause of CES and TNS, we are reasonably certain that if the entire needle tip orifice is positioned inside an arachnoid sleeve, LA can be injected preferentially here. Anatomical variation must also be considered; injection pressure could possibly rupture the arachnoid sleeves in patients in whom they are very fragile structures.

It then becomes plausible to speculate that, alone or in combination with intraparenchymal injection of LA, this could well be the cause of neurological complications following subarachnoid block. This could be a toxic effect due to under- or non-dilution of the LA. If the entire needle orifice is positioned inside the arachnoid sleeve, the bulk of the LA will be injected into the sleeve with only minimal back leakage into the greater pool of CSF via the needle perforation. We thus propose that LA injected into this small pool of CSF inside the arachnoid sleeve would be less diluted than if it were injected into the larger pool of CSF, therefore most likely exposing the naked and unprotected nerve fibers to a relatively high and potentially toxic concentration of LA.

More distally, the arachnoid sleeves are simple tubular structures, but closer to the conus medullaris they form complex branching structures. At this level, the arachnoid sleeves have multiple interconnections where LA spread could affect
more nerve roots, thus increasing the extent of neurological injury.

REFERENCES

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NEEDLE ARCHITECTURE: VULNERABILITY OF DIFFERENT NERVES TO INTRAFASCICULAR INJECTION WITH DIFFERENT NEEDLE BEVELS

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Deliberate or inadvertent intraneural injection may be associated with a variety of neurologic complications. Intraneural injection has traditionally been subdivided into intrafascicular or extrafascicular injection. In the latter instance, the injected solution spreads between the fat cells without breaking or disturbing them, causing the nerve to swell, which can be seen during ultrasound-guided injection.

We have recently demonstrated that after deliberate intraneural injection in fresh cadavers, the possibility of intrafascicular spread is low.1,2 In this study, spread occurred mainly in the extrafascicular adipose tissue and between the cells of the perineuriums of some fascicles. At low magnification, intrafascicular spread could be demonstrated as an ‘island’ inside the fascicle, but this spread on high magnification clearly proved to be between the perineurial cellular layers that formed the perineurial septae inside the fascicles. This gave rise to the concept of ‘pseudo-intrafascicular’ spread, which appeared intrafascicular at low magnification but in fact was not within the endoneurial tissue when the tissues were examined under high magnification light microscopy. 1,2 Our original assumption was that because of the pressure gradient between the intra- and extrafascicular spaces, the entire orifice of the needle had to be inside a fascicle to result in pure intrafascicular spread. When the distal needle orifice was partially outside the fascicle and partially inside the fascicle, the spread would preferentially follow the route of least resistance toward extrafascicular tissue, which contains low-resistance adipose tissue. A mathematical model allowed us to calculate the portion of a needle orifice inside fascicular tissue to evaluate the influence of the type of needle, the angular approaches used for nerve blocks, and the kind of nerve being blocked.

We used cross-sectional images from subgluteal- and popliteal-level sciatic nerves approximately at their bifurcation, a median nerve at the wrist level, and nerve roots of the 5th, 6th, and 7th brachial plexus.3 On these sections, we superimposed images of the distal orifices of a 22-gauge, 15° Stimuplex® D’ needle and a 22-gauge, 30° Stimuplex® Ultra 360° needle at exactly the same magnification. These samples served only to evaluate the proposed method and were not intended to be clinically relevant.

Three virtual angular approaches of the needles to the nerves were studied: 1) perpendicular (90°) to the cross-section of the nerve (90CS), 2) at a 45° angle to the cross-section of the nerve (45CS), and 3) at a 45° angle to the longitudinal axis of the nerve (45LA).

Pre-processing of needle and nerve images: Digital image processing techniques were used to measure the portion of the distal needle orifice section that was inside the fascicles.4,5 First, we performed a pre-processing of the nerve and needle images to reduce the complexity and the computation burden of the proposed method. The three-dimensional (3D) colored cross-section images (RGB images) were converted to two-dimensional (2D) images, assigning the white color (value equal to 1) to the fascicles and the background of the image, and the black color (value equal to 0) to the extrafascicular tissue. The same procedure was followed for the needle images; the white color was assigned to the distal needle orifice, with the background in black. This procedure was done manually using Adobe Photoshop. Although both nerve and needle images were captured with the same magnification.

Because the background of the image and the fascicles were white to simplify the manual pre-processing of converting 3D images to 2D images, the fascicle tissue was automatically identified by searching white pixels (fascicular tissue) on a black background (extrafascicular tissue). A new gray-scaled image was created by assigning the value 1 to the extrafascicular tissue, the value 2 to the fascicular tissue, and the value 0 to the background of the image.

The needle image was superimposed onto the cross-section image, simulating the insertion of the needle into the nerve. To evaluate all the possible scenarios where the needle could be inside the nerve fascicle, the needle orifice image was progressively positioned from the bottom to the top of the nerve image in steps of 61 pixels (or equivalent millimeters) and from left to right in steps of 82 pixels (or equivalent millimeters).3 A new gray-scaled image was obtained for each move. This image represented the sum of the nerve image and the distal needle orifice image, where extrafascicular pixels had the value of 2, the fascicular pixels had the value of 3, and the background pixels had the value of 1 in the regions where the needle orifice was positioned. From this gray-scaled image, the percentage of the orifice needle inside of fascicular tissue was computed as the number of pixels with value 3 divided by the number of pixels with value lower than 3 inside the orifice needle. As a result, a new image of these percentages, Pr×T, was obtained, where R is the number of displacements in the vertical axis and T is the number of displacements in the horizontal axis. This procedure is performed using Matlab R2017b (Math Works, Inc., Natick, MA).

Bidimensional and tridimensional graphical representations of the percentage of the needle orifice inside the fascicle were obtained with different views.4 A 0% to 100% scale with its