

Regional Anaesthesia Update - Simple Steps to a Safer Block

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This talk is aimed at both regional enthusiasts and occasional-ists alike. The main aim is to outline the evidence on enhancing safety in regional anaesthesia, primarily in the avoidance of neurologic complications, as well as local anaesthetic systemic toxicity. I will discuss a number of techniques that may help reduce these risks, according to our current knowledge.

It was established in the 1970's by Dag Selander that intraneural injection caused both functional and histological adverse outcomes¹. It was generally assumed that intraneural injection was the cause of post-operative complications, and that assumption carried through the landmark-guided and nerve stimulator phases.

In 2006 Paul Bigeleisen published a paper in which he described intentional intraneural injection for his axillary blocks in 26 patients². The majority described paraesthesia or dyesthesia, and in about 50%, the symptoms increased during injection. He followed them up at 6 months and none of the patients had any permanent neurological deficit. This study (despite some methodological limitations), suggests that intraneural injection does not invariably lead to neurologic injury.

This may be explained by the structure of a nerve. An intraneural injection may be intra-fascicular or extra-fascicular. It is suggested that the high pressure required to inject within the poorly compliant perineurium (i.e. an intrafascicular injection) may herald the increased likelihood of nerve damage.

This was explored by Hadzic et al in 2004³, where they used a microscope to position a needle either intra- or extrafascicularly in surgically exposed canine sciatic nerves. They found the extrafascicular injections routinely had a low injection pressure (<4 psi), a normal functional recovery and normal histology. The intrafascicular injections fell into two groups: those that had a low injection pressure and essentially behaved like extrafascicular injections; a those that had a high injection pressure (>25 psi), which showed severe neurological injury clinically and histological evidence of injury. This suggested that the high injection pressure did indeed predict poor outcome.

The same group repeated their experiments in 2007⁴ and showed the same outcome, although with a lower incidence of high pressure injection in the intrafascicular group (40% vs 60% in 2004).

Another study from Duke University in 2010 used ultrasound to locate femoral and brachial plexus nerves in pigs, impaled and injected the nerves using a short-beveled needle⁵. Dye tests later revealed that none of the injections were intrafascicular despite all being intraneural. Two injections recorded an injection pressure of greater than 25psi, showing that there is a false positive rate for high pressure injections indicating intrafascicular injection. The fact that there were no intrafascicular injections probably relates to the short beveled needle, a theory backed up by experiments showing that a short beveled needle is likely to push the fascicles away as it traverses a nerve, whereas a long beveled needle is more likely to penetrate the fascicle⁶.

However, if a short beveled needle does enter a fascicle it is more likely to cause more severe damage⁷.

A study by Chan et al⁸ showed that low pressure intraneural injection in pigs did not cause any functional adverse outcome, however histological signs of inflammation were detected.

This animal evidence suggests that the monitoring of injection pressure, or the limitation of injection pressure below a safe threshold may reduce the incidence of poor neurological outcome after peripheral nerve blockade.

One cheap, easily taught and reproduced method to limit injection pressure is the Compressed Air Injection Technique⁹. 10mL of air is introduced above the fluid in the syringe (I use a 30mL syringe using 20mL of fluid and 10mL of air), the syringe is inverted to have the air at the top, the air is compressed to 5mL to initiate the flow of fluid. This follows Boyle's Law, in which pressure x volume is constant. The volume is halved so the pressure is doubled, to 2 x ATM. The net pressure felt at the needle is 1xATM (as the pressure in the needle is already at atmospheric pressure) which is 760mmHg or 14.7 psi, much less than the 25 psi thought to cause damage as part of an intrafascicular injection. This will also slow the injectate rate down, which will reduce the maximum concentration of the local anaesthetic¹⁰. A recent letter suggested the initial injection of the test solution be carried out using this technique¹¹.

The initial solution I inject is a non-local anaesthetic solution. This is for several reasons: it ensures the correct location of the needle tip prior to using the local anaesthetic, thus avoiding any wastage of local anaesthetic. The use of a test solution also allows hydrodissection to be performed, again without incorrectly placed local anaesthetic. Saline has been shown not to cause any functional or histologic damage when injected intrafascicularly¹². It also gives a margin of safety in case of intravascular injection. Having said that, a relatively large dose, ie 60mg of ropivacaine consistently produces symptoms of mild CNS toxicity in young healthy volunteers, in unpremeditated patients as well as those who have received midazolam¹³. This means a large volume ie 12mL of 0.5% ropivacaine can be injected with only mild CNS symptoms, although that dose may be a lot lower in elderly or frail patients¹⁴.

The initial test solution that I prefer is 5% dextrose. This allows nerve stimulation to be performed after the solution has been injected. The reason for this is that 5% dextrose is a non-conducting solution, so the current density is maintained at the tip of the insulated needle¹⁵. Saline and local anaesthetics conduct the current so the current density is greatly reduced, and is not enough to stimulate the nerve, even though the needle might be right next to the nerve.

Nerve stimulation is still a potentially useful tool for detecting intraneural injection. Chan et al showed that a low minimum stimulating current (MSC) was not a great predictor of intraneural placement in pigs¹⁶. A twitch with an intraneural injection was obtained with an MSC of 0.2mA or less in less than a third of cases, and greater than 0.5mA in 50% of cases.

A study by Tsai et al¹⁷ showed that as the needle approached a nerve the lowest current that could elicit a twitch was 0.24mA, however inside the nerve the twitch could be elicited by a current as low as 0.08mA, but in one out of eight cases, the MSC was greater than 0.8mA.

A study by Bigeleisen¹⁸ in humans demonstrated that an MSC of 0.2mA is only achieved with an intraneural injection in the brachial plexus. This study, however, has been the topic of debate given that he is describing an intra-plexus injection, rather than an intraneural injection^{19,20}.

Thus, as another tool in the prevention of neurologic injury, a nerve stimulator can be used with a current set for 0.2mA. A lack of twitch has no value (you could be intraneural with no twitch), but a twitch at that current can only be coming from an intraneural needle tip position.

Another potential use for the nerve stimulator is the measurement of impedance that is produced by some devices, such as the Stimuplex HNS 12 from B-Braun. Tsui et al²¹ was able to show that the interior of a nerve has a higher impedance than tissue outside of the nerve due to a difference in water and fat components. No absolute numbers have been shown and there was a high variability in the numbers produced, so a relative increase may indicate intraneural needle placement, but further investigation is required.

Ideal placement of local anaesthetic in the correct plane under ultrasound is key to both a safe and effective block. In a single nerve, subepineural injection can be identified by nerve swelling, and as little as 1ml has been able to be detected in animal studies²². In humans, as well as being able to visualise the needle within the substance of the nerve, an increase in the nerve diameter, separation of the fascicles and a 'halo' appearance are all suggestive of a subepineural needle position. The issue becomes less clear with a double nerve i.e. the sciatic nerve and multiple nerves as contained within the sheath of the brachial plexus. In the case of the sciatic nerve, a paraneural sheath, which surrounds the epineurium of both the common peroneal and tibial nerves has been identified²³. Injection into this space, instead of the subepimysial space appears to have improved spread and block dynamics. With regards to the brachial plexus, the prevertebral fascia or the axillary sheath can be breached to enter the area containing the extraneural connective tissue. Injection here will cause an expansion of the whole complex under the fascia, whereas the individual nerves will remain the same size²⁰. This does not necessarily imply an intraneural injection, although several authors interpret this as one^{18,24}.

Of course simply using the ultrasound does not exclude an intraneural injury. A recent study comparing different methods of injecting around the sciatic nerve showed an unintended subepineural injection in 8% of patients²⁵.

However, if you combine these techniques, using 5% dextrose as the initial test solution to allow low current nerve stimulation prior to a pressure limited injection, in the correct plane under ultrasound, ensuring that no individual nerve swelling is visualised, you have taken several steps to make the block as safe as possible, according to our current understanding.

Please note there are other safety issues that have not been discussed including asleep vs awake patients, intravascular markers such as adrenaline and safe doses of local anaesthetics.

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