## CHAPTER 5: PERIPHERAL NERVE BLOCKS



### Peripheral Nerve Blocks

A successful peripheral nerve block results from injecting an adequate volume of an adequate concentration of local anesthetic solution in the proximity of the target nerve(s). Intraneural injection (especially intrafascicular) may be harmful to the nerve and can lead to permanent damage. Therefore, a balance must be achieved between the need to get close to a nerve and safety.

## Bringing the needle close to the nerve(s)

There are several methods that can be used to drive the blocking needle into the proximity of the target nerve(s). Historically purely anatomical means and paresthesias were the only methods available. Nowadays nerve stimulation and ultrasound are the preferred methods, keeping in mind that a good practical knowledge of the involved anatomy is the single most important factor contributing to the safety and efficacy of nerves blocks. Below is a summary of these different methods.

1. **Purely anatomical:** the practitioner bases his/her technique solely on anatomical facts to bring the needle in proximity to the nerve. For example, he/she can use the pulse of the femoral artery to locate the femoral nerve in the groin, or the pulse of the axillary artery to block the terminal branches of the brachial plexus in the axilla.

   This anatomical method is extremely operator-dependant with good success in the hands of the few and limited success in the hands of the majority. This method does not take into account anatomical variations, lacks depth perception and cannot gauge proximity to a nerve with any degree of certainty. Therefore, the needle might end up too far from the nerve (failed block) or too close to it (intraneural).

2. **Paresthesia:** this technique requires a combination of anatomical knowledge and patient collaboration. The needle is brought to the point of physical contact with the target nerve. The patient is instructed to acknowledge the electrical sensation elicited (paresthesia) upon nerve contact. The location of the paresthesia, as referred by the patient, provides information on needle location. At this time the needle is withdrawn a few mm, before the injection is started, to decrease the risk of intraneural injection.

For the longest time, Moore's dictum "no paresthesia no anesthesia", was the "law of the land" in regional anesthesia. Works by Selander and others, starting in the 1970s, have questioned the safety of this practice. Although, there is not enough evidence associating paresthesias to nerve damage, there seems to be enough circumstantial evidence to be cautious, especially if repeated paresthesias are elicited.

3. **Nerve stimulation:** the idea of locating mixed nerves by electrical stimulation was developed by Perthes in Germany in 1912. However, it was not until 1962 when Greenblatt and Denson introduced a portable, transistorized nerve stimulator that the technique became more popular in the clinical setting.

The nerve stimulator is connected to a needle, usually insulated, that delivers a current to its tip. The A alpha fibers (motor) are readily depolarized by the small currents used, but not the sensory fibers. As the needle approaches a mixed nerve, a painless muscle twitch is produced. The intensity of the response is inversely proportional to the needle tip-nerve distance (actually to the square root of it). A visible response at lower currents (less than 0.5 mA), suggests close proximity between the needle tip and the target nerve. There is a good amount of clinical evidence to suggest that a current of 0.5 mA or less, capable of eliciting a visible response, is a reliable indicator of enough proximity. However, evidence is lacking as to what exactly that distance is, and as to whether that distance is different for different nerves. In general it is thought that 1 mA of current will produce depolarization of a motor nerve at a distance of about 1 cm (10 mm).

Nowadays nerve stimulator techniques are widely practiced around the world. With modern nerve stimulators the practitioner can adjust the **pulse intensity** (magnitude of the current) in mA; the **pulse frequency** (amount of pulses per second) in Hz (1 or 2) and the **pulse width** (duration of the pulse) in milliseconds (ms). The pulse duration most suitable for stimulating motor fibers in a mixed nerve is 0.1 ms (100 microsec).

## Insulated versus non insulated needles

Insulated needles are the needles most commonly used in conjunction with nerve stimulation and ultrasound techniques nowadays in the United States, Europe and other parts of the world. The current applied to this needle concentrates at its tip, making the localization of nerves more accurate. Several brands of these needles exist in the market and they come ready with a connection that only fits the negative electrode. Connecting the negative electrode to the exploring needle lowers the amount of current necessary to depolarize a nerve.

Non-insulated needles transmit the current preferentially to the tip, but also along the shaft of the needle making the localization of nerves less accurate. Insulated needles are more expensive than non-insulated needles.

## Short versus long-bevel needles

Standard needles have a tip angle of around 14 degrees and are known as "sharp' needles. It is frequently recommended to perform regional block with short-bevel needles with an angle of 30 to 45 degrees. This recommendation comes from studies by Selander et al who demonstrated more neural damage in isolated sciatic nerves when sharp needles were used. The damage with sharp needles was also more extensive when the orientation of the sharp bevel was perpendicular to the fibers. With short bevel needles, the damage was less frequent as the fibers were pushed away by the advancing needle.

This concept has been challenged by Rice et al. According to these authors it may be more difficult to penetrate a nerve fascicle with a short-bevel needle than with a sharp needle, but should it occur, the lesions may be more severe. Recently in 2009 Sala-Blanch and collaborators published in Regional Anesthesia and Pain Medicine a study in which sharp long beveled versus blunt short beveled (30 degrees) needles were introduced into a sciatic nerve of a human cadaver. After the punctures the specimen was investigated under the microscope for evidence of fascicular damage. They demonstrated that with either needle was very difficult to penetrate the fascicles. In fact they found no histological evidence of fascicular damage with short beveled needles and only 3.2% of fascicular damage (4 fascicles) with sharp needles.

4. **Ultrasound:** It is the latest and most sophisticated piece of technology introduced to the practice of regional anesthesia and has already caused a revolution. It is the only method that can provide real time assessment of the position of the needle with respect to the nerve, as well as an image of the surrounding structures. An added advantage is that the practitioner is able to make an assessment of local anesthetic spread, giving him /her the chance to more accurately predict the success of the technique as well as the need for supplementation.

   Ultrasound could theoretically produce warming of tissues or gas formation. This technology is still expensive, and requires competency on interpretation of cross-section anatomy from "grainy" images. However, it has been rapidly progressing and in many centers, including ours, has become the gold standard method to perform regional blocks of every kind.

**Characteristics of ultrasound**

The human ear can hear sounds between 20 and 20,000 Hz (cycles per second) or 20 KHz. Ultrasounds waves travel at a higher frequency than the highest frequency detectable by the human ear. Ultrasound waves used in medicine usually are in the 1 to 20 MHz range (1 MHz = 1 million Hz).

Ultrasound waves travel easily through fluids and soft tissue, but have problems traveling through bone and air. Ultrasound is better reflected at the transition between two different types of tissues like soft tissue-air, bone-air and soft tissue-bone. This transition plane is seen as a hyperechoic line on the screen.

The ultrasound is delivered from a small probe that contains piezoelectric crystals that under the influence of an electric current are made to vibrate producing a wave of ultrasonic sound. The ultrasound waves in the form of a narrow beam travel through tissues at a speed that depends on the nature of the human tissues, but for calculations and image production is assumed to be an average value of 1,540 m/sec. This value closely approximates the speed of ultrasound through soft tissue (1,540 m/sec), muscle (1,580 m/sec), blood (1,560 m/sec), but differs to the speed through bone (4,000 m/sec), lung (500 m/sec) or air (330 m/sec).

Part of the ultrasound waves are reflected back to the transducer, especially at tissue interfaces, where the mechanical energy is converted back to electrical energy. The information is then processed by the software of the ultrasound machine to generate an image. Therefore, the transducer delivers ultrasound for part of the time and for part of the time it "listens" for the returned waves. The distance is calculated as a function of the time it takes for the waves to return. Tissues with high density like bone reflect most of the waves and produce a bright image, known as **hyperechoic**. A tissue like blood that permits easy passage of the ultrasound waves through it appears dark or **anechoic**. The rest of tissues present intermediate characteristics between anechoic o hypoechoic to hyperechoic.

Better images are obtained when the probe is perpendicular to the structure being searched (e.g., nerve, needle). This is because more bouncing sound waves can be detected by the transducer. Changes as small as 10 degrees from the perpendicular can distort the echogenicity of a nerve, reducing the amount of waves returning to the transducer and decreasing the quality of the image. This is known as **anisotropy**, the change of the quality of the echo image as a result of change in the angle of incidence of the probe with respect to the target structure. Tendons characteristically have higher anisotropy than peripheral nerves.

**Short versus long axis views**

The most common way to identify a peripheral nerve is through a transverse scan of it, also called "short axis view". This provides a cross section image of the nerve(s) and surrounding structures. A "long axis view" of a nerve is also possible, although sometimes more challenging, because the nerves trajectories are not necessarily linear. In addition, in a long axis view the operator looses the ability to readily recognize lateral and medial sides of the nerve on the 2-dimensional image obtained.

**In plane versus out of plane techniques**

The needle can be advanced "in-plane" or "out-of-plane" with respect to the main axis of the probe. In the in-plane approach the needle is advanced in coincidence with the long axis of the probe, in other words, in the same plane of the ultrasound beam. This makes possible the visualization of the needle as it advances toward the target nerve(s).

Good needle visualization depends on its angle of insertion, with the best visualization obtained when the needle trajectory is parallel to the probe. As the angle of penetration increases (deeper targets) the difficulty to visualize the needle also increases. When the insertion angle is more than 45 degrees with respect to the plane of the probe the needle is only visualized as a faint "shade". At this point tissue movement and injection of small amounts of local anesthetics can help determine the location of the needle tip.

With the out-of-plane approach the needle is advanced perpendicular to the main axis of the probe, so only the tip of the needle can be visualized at the point where it crosses under the ultrasound beam. The tip is seen as a very hyperechoic bright point on the screen. As the tip of the needle approaches the plane of the ultrasound beam a "tissue disruption" is observed on the screen, which helps to locate the needle. The main advantage of an out-of-plane technique is the shorter trajectory that the needle needs to travels to its target, a very important point in deeper blocks. Regardless of the approach the goal is to bring the tip of the needle into the proximity of the nerve(s) for injection.

### High versus low frequency probes

High frequency probes (8-15 MHz) are usually linear probes that provide good resolution, but limited penetration (3-4 cm). These probes are used at different levels of the brachial plexus, abdominal wall and at different locations in the lower extremity. For deeper structures, lower frequency (4-7 MHz) curved probes are needed providing a wider field and deeper penetration at the expense of resolution. Deep scanning of intra abdominal organs requires frequencies of 3-5 MHz. The quality of the image is also affected by other factors like compound imaging (the capture of different views of structures before producing an image) and color Doppler.

## Nerve injury

Persistent paresthesias can occur after regional anesthesia, although severe neurologic injury is extremely rare. Neal estimates the incidence of persistent neuropathy after regional anesthesia to be less than 0.4%.

A large survey by Auroy et al in France in 1997, involving 71,053 neuraxial blocks and 21,278 peripheral nerve blocks, showed a low incidence (0.03%) of nerve complications after regional anesthesia. The survey showed that neurological deficits although low, were relatively more frequent after spinal (70%) than either epidural (18%) or peripheral nerve block (12%). In two thirds of the cases of neuropathy after spinal, and 100% of the cases after epidural, a paresthesia was elicited either by the needle or during the injection. Among the neurological deficits that developed after non-traumatic spinals, 75% of them were in association with the use of 5% hyperbaric lidocaine.

Cheney et al in 1999 reviewed the American Society of Anesthesiologist closed-claims database and found that out of 4,183 claims, 670 (16%) were considered "anesthesia-related nerve injury". Injury to the ulnar nerve represented 28% of the total, and in 85% of the cases it was associated to general anesthesia. Other nerve injuries were brachial plexus in 20%, lumbosacral trunk in 16% and spinal cord 13% and these were more related to regional anesthesia. In 31% of the brachial plexus injuries the patient had experienced a paresthesia with the needle or during injection. They concluded that prevention strategies are difficult because the mechanism for nerve injury, especially of the ulnar nerve, is not apparent.

Lee et al in 2004 conducted a new review of the Closed Claims Data for the 1980 to 1999 period focusing in regional anesthesia. A total of 1,005 regional anesthesia-related claims were reviewed. These claims were 37% obstetric related and 63% non-obstetric. All regional anesthesia, obstetric claims were related to neuraxial anesthesia/analgesia. In 21% of the non-obstetric claims, peripheral nerve blocks were involved. The most common block was axillary block (44%). Upper extremity blocks were more involved in claims than lower extremity blocks. Nerve injury temporary or permanent was claimed in 59% of the peripheral nerve injury claims.

Death or brain damage was usually the result of cardiac arrest associated with neuraxial block. Pneumothorax accounted for 10% of the claims and "emotional distress" was claimed in 2% of the cases. Eye blocks accounted for 5% of the claims.

Regional anesthesia could result in nerve damage directly from a needle or catheter or be the result of ischemia or other unknown mechanism. Ischemia could be the potential result of vasoconstrictor use or by an intraneural injection that produces an increase of the intraneural pressure leading to nerve ischemia. Local anesthetic toxicity could play a role in cauda equina syndrome and transient neurological symptoms. Another mechanism of nerve injury could be hematoma and infection leading to scar formation.

It has been a common belief in regional anesthesia that nerve puncture and intraneural injection lead to nerve damage. In 2006 Bigeleisen published in Anesthesiology a study that seems to discredit this notion. In his study conducted under ultrasound guidance 21 of 26 patients had nerve punctures of at least one nerve, and 72 out of 104 nerves had intraneural injection (2-3 mL). A 6 month follow up failed to demonstrate nerve injury. Incidentally it is important to notice that the local anesthetic mixture injected (bupivacaine plus lidocaine) contained 3 microgr/mL of epinephrine.

Since peripheral nerves are formed by neural tissue (fascicles) and connective tissue, it is possible to penetrate the nerve (intraneural), but still be extrafascicular. In 2004 Sala-Blanch et al reported in Anesthesiology two cases of inadvertent intraneural, extrafascicular injection after anterior approach of the sciatic nerve block with nerve stimulation performed in two diabetic patients, as evidenced by CT scan. These two cases also demonstrate that painless nerve punctures and even intraneural (although extrafascicular) injections are possible without apparent sequelae.

**A preexisting neurological injury should always be documented. It is important to realize that nerve damage can occur perioperatively for a reason other than regional anesthesia. Nerves can be injured during surgery by direct trauma, use of retractors and tourniquets and by improper positioning. Nerves can also be damaged postoperatively by a tight cast or splint, wound hematoma or surgical edema.**

## *Use of epinephrine*

Epinephrine-containing local anesthetic solutions may theoretically produce nerve ischemia by vasoconstriction of the epineural and perineural blood vessels. Patients at increased risk would be those with previous impaired microcirculation (e.g., diabetics). There is no evidence at this time to suggest a detrimental effect of epinephrine in regional anesthesia, as used in clinical practice. Epinephrine has been used extensively and presumably safely in regional anesthesia for over 100 years. The 2010 ASRA Practice Guidelines on Local Anesthetic Toxicity, cited elsewhere, recommends the use of epinephrine in nerve blocks as an intravascular marker considering that the benefits outweigh the risks in the majority of patients. We use local anesthetic solutions containing epinephrine 1:400,000 (2.5 micrograms/mL) in all kind of patients, and we appreciate its role as an indicator of inadvertent intravascular injection (for more information on the subject please see the local anesthetics chapter).

**Persistent paresthesia, Clinical presentation**

The symptoms can appear within 24 h after the injury, but sometimes they do not present until days or weeks after the offending procedure took place. The degree of symptoms is usually related to the severity of the injury. The cases are usually mild with symptoms like tingling and numbness that usually disappear within weeks, or more rarely they can progress to severe cases of neuropathic pain and motor involvement that can last months and even years.

**Pre-existing neurologic condition and regional anesthesia**

A pre existing neurologic condition per se is not a contraindication to regional anesthesia. However a careful preoperative assessment must be performed and any neurological deficit must be documented in the patient's chart. A thorough discussion with the patient and the surgeon is always important.

Certain progressive neurologic conditions like multiple sclerosis, acute poliomyelitis, amyotrophic lateral sclerosis and Guillian Barre syndrome are relative contraindications to regional anesthesia, because the development of new symptoms postoperatively may be confused with complications from the nerve block. In these cases the risks and benefits must be carefully evaluated before proceeding with regional anesthesia. In 2006 Koff et al published in Anesthesiology a case of severe plexopathy after an ultrasound-guided interscalene block in a patient with multiple sclerosis.

There are other stable neurologic conditions like a preexisting peripheral neuropathy, inactive lumbosacral radiculopathy and neurologic sequelae of stroke that can be adequately managed with regional anesthesia, provided that all preexisting neurological deficits are well documented in the chart.

## Persistent paresthesia prevention and management

In order to minimize the risk of neurologic injury after regional anesthesia the anesthesiologist needs to consider several factors, including procedure, patient and surgeon. A meticulous nerve block technique, avoiding direct trauma to the nerve and appropriate selection of local anesthetic volume and concentration are important. The role of vasoconstrictors, especially low dose (1:400,000), on clinical development of neural ischemia, has not been elucidated.

When a neuropathy develops in the post operative period, a prompt evaluation is necessary and a multidisciplinary approach, with participation of neurology, radiology, and surgery, is recommended. A detailed history must be obtained including the timing and nature of symptoms. A physical exam should look for any signs of hematoma or infection. A neurological exam by a neurologist is also crucial.

**Electrophysiological testing**

Although electrophysiological studies remain normal for 14 to 21 days after the injury, ordering them early could help establish a baseline and rule out any preexisting condition. These tests have limitations, as they only assess large motor and sensory fibers and not small unmyelinated fibers. They usually include nerve conduction velocity studies and electromyography and sometimes may include evoked potentials.

1. **Sensory Nerve Conduction Studies**
   They assess functional integrity of sensory nerves by measuring amplitude and velocity of peripheral nerve conduction. Injuries involving fascicular damage primarily show a decrease in the amplitude of the action potential, a sign that the impulses are being transmitted by a reduced a mount of fibers. Conduction velocity in these cases may be minimally affected. When the lesion is demyelinating, like the ones seen after tourniquet compression, nerve conduction velocity is greatly affected while the amplitude remains normal.

2. **Electromyography**
   It records electrical activity in the muscles helping to locate the denervated muscles in reference to the level at which the nerve damage has occurred. Within 2-3 weeks post injury, spontaneous activity can be recorded from the muscle, in the form of sharp waves and muscle fibrillation. After 3 months the pattern may change, as nerve regeneration by "sprouting" takes place. In permanent injuries, electromyography remains abnormal.

**Tourniquet**

Use of crude compression devices to control surgical bleeding from the extremities, can be, according to Bailey, traced back to ancient Rome. The tourniquet was apparently introduced by Petit, a French surgeon, in 1718. The device was a mechanical screw-strap contraption that he used to provide surgical hemostasis in amputations of the extremities. It was Lister though in 1864 the first surgeon to use a tourniquet to produce a bloodless surgical field. Modern tourniquet devices have a microprocessor, use an air pump and are able to accurately and safely maintain the desired pressure. A fail-safe mechanism protects from pressure ever exceeding 500 mmHg.

**Tourniquet time:** Recommended tourniquet time varies, but the most commonly accepted limit is 2 hours. This recommendation is based on a work by Wilgis, published in 1971 in which he demonstrated more acidosis after 2 hours of ischemia. Surgeons should be made aware when the 2-hour limit has been reached and the tourniquet should be deflated at that time, unless the procedure is at a crucial time. This communication with the surgical team needs to be documented in the chart.
Despite the widely accepted 2-hour limit, Klenerman, as cited by Bailey (1994), saw minimal evidence of muscle damage under electron microscopy with tourniquet times up to 3 hours.
Some people advocate deflating the tourniquet at 1.5 h for 5-15 minutes followed by an additional 1.5 h of inflation time.

**Tourniquet inflation pressure:** It is believed that inflation pressure is more important of a factor than time in influencing injury. It is recommended to use the minimum inflation pressure that accomplishes ischemia. In general 100 mmHg above the systolic pressure is a common setting. Roekel and Thurston in 1985 showed that 200 mm Hg for the upper extremity and 250 mm Hg for the lower extremity were adequate parameters. Adding layers of padding is important. Wrinkles in the padding should be avoided, since they may become pressure points.

**Tourniquet associated problems:** The exsanguination with an Esmarch bandage prior to tourniquet inflation causes an increase in preload, which can be significant when bilateral tourniquets are used in the lower extremities. Eliminating circulation in part of one extremity also can lead to an increase in afterload. This may cause problems in patients with cardiac problems and decreased cardiac output. Exsanguination of lower extremities has also been associated with pulmonary embolism and cardiovascular collapse.
Some patients may develop post-tourniquet nerve palsy, affecting more frequently larger motor fibers than sensory fibers. These lesions are usually reversible. The magnitude and duration of the compression dictate the severity of the injury.
Patients can also develop **"post-tourniquet syndrome"**, a clinical picture characterized by interstitial edema, arm weakness and numbness secondary to cell injury and alteration or permeability. It usually resolves within a week.
When the tourniquet is deflated blood pressure drops (sudden drop in preload and afterload) and heart rate increases as blood rushes into an ischemic, vasodilated bed (reactive hyperemia). Carbon dioxide and potassium levels increase and so does lactic acid leading to acidosis. These effects peak at about 3 minutes post deflation. There is also a decreased in patient's body temperature.

**Tourniquet pain:** It is commonly observed despite signs of otherwise good anesthesia of the extremity. Unpremedicated volunteers refer intolerable pain by 30 minutes. Signs of tourniquet pain, manifested as a gradual rise in blood pressure, are also observed under neuraxial blocks and general anesthesia. Patients report this pain under the tourniquet and distal to it.
Controversy exists as to how this pain is transmitted. De Jong and Cullen in 1963 proposed that tourniquet pain was transmitted by small non-myelinated sympathetic fibers. However tourniquet pain can arise even when high thoracic levels of anesthesia are present.
It seems that tourniquet pain is transmitted, as other painful sensations, by A-delta myelinated fibers and C unmyelinated fibers. Tourniquet pain is usually described as burning, cramping or heaviness. The burning and aching sensations, characteristics of ischemia, are believed to be conducted by unmyelinated fibers (MacIver and Tanelian, 1992), while the sharp pain, usually a small component of tourniquet pain, is transmitted by A-delta fibers.
MacIver and Tanelian proposed that C fiber activation by ischemia-induced alterations are responsible for tourniquet pain. They studied in an in-vitro model the effects of ischemic alterations (i.e., hypoxia, hypoglycemia, lactic acid, and decreased ph) on A-delta and C pain fibers. They showed that hypoxia and hypoglycemia induced under ischemia, increased C fiber tonic action potential activity, but did not affect A-delta fibers. Increased lactate and decreased pH did not alter the discharge frequency of C fibers in this model. The activation of C fibers by ischemia products seems crucial in tourniquet pain. Whether these C fibers eventually enter the spinal cord at a level above the somatic nerve block is debatable.

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