

Postoperative Nerve Injury Not Due to the Nerve Block

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INTRODUCTION

There are two reasons why a nerve injury discovered after a peripheral nerve block is a problem. The surgeon reflexively assumes that the nerve block caused the neurological deficit, and it is implied in any subsequent malpractice legal action that follows that the nerve block was negligently performed. These assumptions may be mostly untrue.

Peripheral regional anaesthesia has a well proven outcome general safety record observed for over 100 years in humans. However catastrophic injuries do occur rarely, and their mechanism of injury is often unclear. This suggests that other unrecognized factors (“factor-X”) were present.

Sixteen percent of all human anaesthesia closed claims are for nerve injuries. Two thirds of nerve injuries occurred after general anaesthesia only, and only one third after regional anaesthesia. The majority of regional anaesthesia associated nerve injuries are from epidural and spinal anaesthesia, and peripheral nerve blocks are a lesser source of nerve injuries. Upper limb nerve injuries mostly are positioning related and pressure related. Lower limb injuries are nearly exclusively associated with lumbar epidurals and spinal anaesthesia in humans.

The incidences of nerve injuries reported after peripheral nerve block range greatly from 0/2400 (0%) to cases to 18/300 (5.7%). This may reflect differences in technique and operator skill.

The biggest human peripheral nerve injury problem lies with (1) axillary blocks, and (2) nerve blocks for shoulder surgery. Severe long term nerve injuries after interscalene plexus block has been documented at up to 0,4%. There are some other more likely factors causing nerve function deficits discovered after shoulder surgery, and than the nerve block itself. Shoulder joint pathology is hard to differentiate from concomitant brachial plexus disease. Shoulder surgery positioning and traction of the arm place unusual severe traction and pressure on the brachial plexus from the roots to the axilla and mid-humerus, where the arm is tightly held and manipulated. The incidence of nerve block associated injury for nerve blocks unrelated to shoulder surgery is very small. The commonest injury is neuropraxias and is of temporary nature.

This presentation will focus on some other process that can cause discovery of postoperative nerve injury that are unrelated to nerve blocks.

The diagnosis of a nerve block induced injury can very seldom be made specifically. More usually the diagnosis of nerve block injury is a diagnosis of elimination. This requires that multitudinous, perhaps rare, other nerve injury pathologies be considered investigated and eliminated first before the nerve can be considered as the etiology of the nerve deficit.

ILLUSTRATIVE CASES

Case #1: A lady presents with the complaint of having a severe numb right thumb after undergoing surgery. It affected her ability to type, and thus her ability to earn a living. She sued the anesthesiologist. The anesthesiologist referred the former patient, now litigant, to a neurologist for evaluation.

The neurologist uncovered the following information. The lady’s numb thumb had troubled her prior to the surgery and anesthesia, but to a lesser degree. She had also had multiple unsuccessful injections for symptoms resembling those of tennis-elbow syndrome. The neurologist

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final diagnosis was that the lady suffered from an idiopathic brachial plexus neuritis. After a course of oral steroids the thumb and elbow symptoms and other minor symptoms of the plexitis improved. The litigant dropped the case against the anesthesiologist. The reason the lady had chosen to sue the anesthesiologist had been because she recalled the pulse oximeter had been placed upon her thumb. Of note the lady had never had a nerve block, and had only had a 30 minute long gynecological surgery. The deterioration of her thumb numbness complaints was likely purely coincidental to the surgery and anesthetic that she had received. Had the lady undergone a shoulder surgery instead, together with an interscalene nerve block it is possible the severe postoperative numb thumb could have been successfully litigated as a consequence of the nerve block.

Case #2: A patient presents with a numb left thumb after surgery. The patient attributed the symptoms to the anesthesiologist and initiated legal proceedings. Of note; the only surgical-anesthetic intervention applied to the left arm had been the application of an automated blood pressure measuring tourniquet cuff on a 6 minute pressure measuring cycle. A neurologist was consulted. The neurologist noted a positive Tinel test. The test involved tapping the carpal tunnel which elicited a shooting discomfort along the arm. A carpal tunnel syndrome was identified, and the tunnel pressure measured. The severity of symptoms and the noted high pressure and poor response to conservative treatment measures led to the patient undergoing successful carpal tunnel surgery. Of note, the patient admitted to having had minor symptoms of carpal tunnel prior to the first surgery, but had not suffered enough to make him consult a physician. Had the man undergone shoulder surgery instead, together with an interscalene nerve block it is possible the severe postoperative numb thumb could have been successfully litigated as a consequence of the nerve block.

LESSON: Just because a neurological injury becomes apparent following a nerve block does not prove the nerve block was the etiology.

Case #3: A sixteen year old needed surgical repair of a patella and knee joint injury. She was given a femoral nerve block and general anesthesia. She was found post-operative to have persistent numbness of the anterior thigh skin and a substantial but not total thigh muscle weakness. The sensory loss recovered after 6 weeks. The patient developed persistent deep muscle pain in the thigh for six months. The full thigh strength took one year to recover. The nerve block was blamed initially.

Final opinion was that that is highly likely not a nerve block injury but rather tourniquet induced demyelinating axon injury. It is a well described phenomena in animal research and occurred with the earliest automatic blood pressure monitoring devices. If tourniquet pressure is excessive at the interface between the tissues under pressure from the tourniquet and the adjacent normal pressures axons can have their myelin layers stripped off them in a degloving type of injury. This occurs more likely and worst with more superficial lying nerves like cutaneous sensory nerves, than with the deepest motor nerves. Once the myelin is regenerated in single weeks the sensory nerves become normal again. Motor recovery takes longer for two reasons. Severe muscle wasting occurs secondary to two events. The first event is the demyelination of the motor axons. Muscle recovery can only start after demyelination is fully repaired. The second event is that typically a secondary complex regional pain syndrome (CRPS) develops that hinders recovery. Full and final recovery can only occur after the CRPS involving deep muscle hyperalgesia has recovered.

In this patient a tourniquet inflation pressure for the surgery of 300 mmHg was used. In addition the young girl had a very asthenic build with notably thin thighs. That represented the maximally worst combination of risk factors to develop this type of injury. Persons with large thighs, are relatively protected from this syndrome.

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Case #4: A young man underwent knee surgery for an injury and received a femoral nerve block and general anesthesia. Two units of blood were transfused. One day after surgery his whole foot on the surgical side became severely painful and the skin was tender to touch. The surgeon blamed the femoral nerve block. It was pointed out that the femoral nerve does not supply the foot. It also

became apparent that there were similar signs and symptoms, although much less severe, in both hands and in the opposite side foot.

A neurologist proposed that this was post-surgical inflammatory neuropathy. As sacrifice-able sensory cutaneous nerve was biopsied showing nerve inflammation, and it confirmed the diagnosis, thus fully exonerating the coincidental nerve block. This syndrome (i) affects multiple nerves, (ii) only manifests a day or later after surgery, (iii) is associated with pain and allodynia, (iii) blood transfusions increase the risk, (iv) responds positively to corticosteroids, (v) affects distal limb parts of a limb or nerve more than the proximal limb or nerve part, and (vi) the prognosis is good. It is thought this relatively unknown syndrome is also underdiagnosed.

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Case #5. Six months after undergoing an awake shoulder repair under interscalene block the patient developed a respiratory infection with shortness of breath. A chest X-ray revealed evidence of phrenic nerve paralysis on the side of the surgery and prior nerve block. The anesthesiologist was sued for causing injury to the phrenic nerve.

During the ensuing legal proceedings the anesthesiologist proposed that the late discovered phrenic nerve deficit had not been adequately investigated to exclude other causes of phrenic nerve injury. Suggestions including performing a MRI scan of the base of the neck to exclude a lung apical tumor infiltrating the phrenic nerve and causing a Pancoast syndrome. This was done. By serendipity the scan was performed low enough to include a view of the upper pole of the kidney on that side. A renal cancer was seen and soon operated. The phrenic nerve subsequently recovered full function.

The final diagnosis was that the patient had had malignancy associated neuropathy which had affected the phrenic nerve. It is an auto-immune based illness and typically the affected nerve or nerve recovers upon cure of the cancer. This is a well documented, and described disease.

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FUNCTIONAL ANATOMY OF PERIPHERAL NERVES.

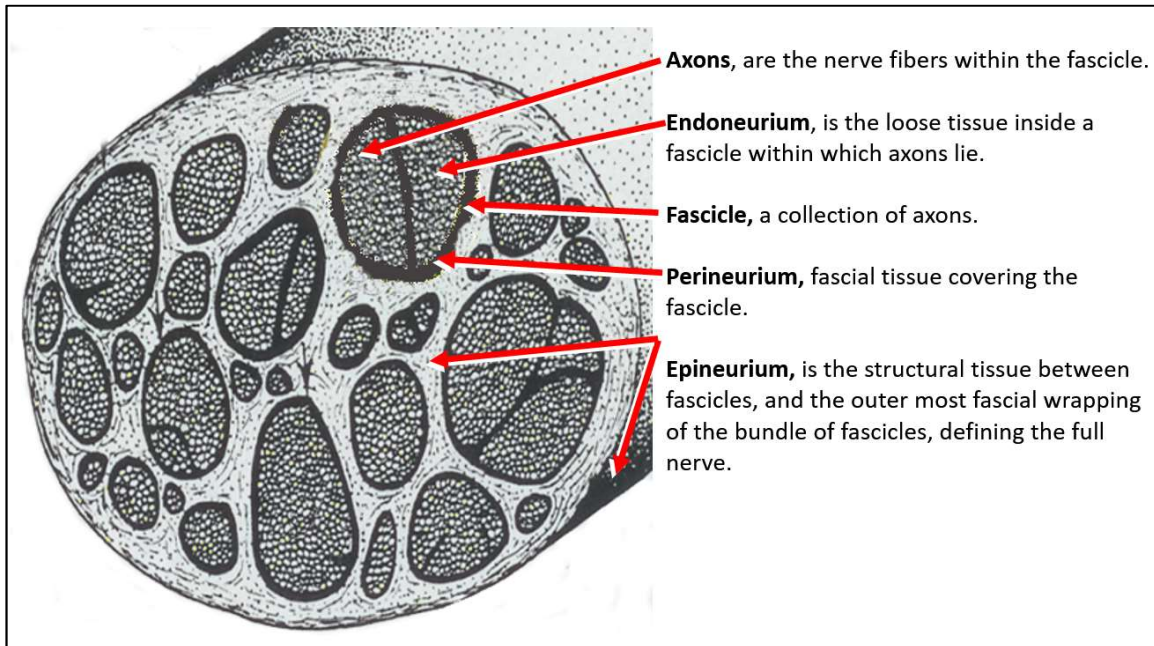
TERMINOLOGY;

Epineurium = the connective tissue layer surrounding the entire nerve trunk, and is tough and thick.

Perineurium = the connective tissue layer covering anatomical individual sub-groups of nerve fibres in a funiculus or fascicle within the epineurium. The groups have mixed sensory and motor fibres destined for one anatomical area. It is fairly tough.

Endoneurium = the connective tissue layer covering individual nerve fibres, and is loose and thin.

Funiculus or fascicle = small collection of sheathed axons of either motor or sensory type.



Axons have functions of (1) electrical impulse transmission and (2) axonal transport of proteins, receptors, and neurotransmitter precursors from the soma (cell body) to the periphery. These processes are highly oxygen and energy dependant, and only slight the axon pressure arrests this transport.

The axon exists in a specialized milieu controlled by the epineurium and the nerve blood barrier (analogous to the blood brain barrier). The nerve blood barrier anatomically is the endoneurium capillary. The epineurium also physically aids in creating a different intra-neuronal milieu from that of the extra neuronal interstitial milieu. Disruption of the specialized milieu effects axonal function and survival.

While the epineurium which is of ectodermal origin similar to epithelium, has lymph vessels, the intraneural tissues has no lymph drainage and is prone to edema and edema takes a long time to resolve.

PRE-EXISTING NERVE INJURY AND PERIPHERAL NERVE BLOCKS

More injuries are reported with brachial plexus blocks than any other peripheral nerve block group. The reason may be because (1) the brachial plexus is the most blocked peripheral nerve, (2) the highly mobile human arm puts the human plexus under more strain positions than other peripheral nerves, (3) shoulder surgery puts severe surgical strain onto the brachial plexus, (4) brachial plexitis is largely unrecognized and may be camouflaged as being part of other apparently painful arm or shoulder conditions. Placing a nerve block into an unrecognized brachial plexitis may result in the patient wrongly attributing persistent anaesthesia, discomfort or pain to the nerve block.

Part of plexitis may be an unrecognized Complex Regional Pain Syndrome (CRPS). Commonly CRPS is unrecognized, because the radiologically visible and operable orthopaedic disease dominates clinical assessment. The presence of CRPS may result in both a disappointing surgical result to reduce the pain, and also a worsening CRPS with symptoms removed from the surgical field which will be blamed on the nerve block. While regional anaesthesia is not contraindicated in the presence of CRPS the hypersensitivity of skin needs to be sought, and documented and highlighted to the patient who must give prior informed consent for the nerve block.

Also avoid performing brachial plexus nerve block in the presence of a diagnosed acute brachial plexitis. *Acute Brachial plexitis* is characterized by the *acute onset of shoulder pain, weakness, and paralysis in patients following a variety of surgical procedures and this occurs without any regional anaesthesia involvement*. This illness is rare. The addition of a nerve block to a patient developing unrecognized brachial plexitis invites attributing the etiology to the nerve block. The etiology of brachial plexitis may include recent/current viral and bacterial infections and giant cell arteritis, and polymyalgia

rheumatica. It is advisable to avoid performing brachial nerve block in patients with unexplained pain in the forearm elbow or shoulder unrelated to the surgical pathology, particularly if they have current myalgic or febrile infection or connective tissue disease such as polymyalgia rheumatica and giant cell arteritis.

PATHOPHYSIOLOGICAL CLASSIFICATION OF PERIOPERATIVE DIAGNOSED NERVE INJURY (Based on Seddon 1943)

1. False new nerve injury.
 - a. Pre-existing unrecognized *neuropathy* or injury.
2. True new nerve injury.
 - a. *Neuropraxia*. There is axonal sideration (acute loss of neurological functioning, of seemingly no visible cause). This represents minor neuronal insult, has the feature of decreased conduction, and reverses over time.
 - b. *Axonotmesis*. There is selective axonal interruption, but with preservation of supporting connective tissues. Sideration last 6 weeks where after axonal regrowth from to injury point to the distal end of the nerve occurs at 1-2 mm per day. Good health and youthfulness favours better recovery. The cause is typically compression, stretching or topical depositing of toxic substances or drugs.
 - c. *Neurotomesis*. There is both axonal interruption and supporting tissue disruption. Loss of continuity of proximal and distal tissue alignment makes recovery is impossible.
3. Combination injury and neuropathy. There has been a suggestion that a minor injury causes a worse deficits in the presence of neuropathy. There is however little objective evidence that routine regional anaesthesia influences underlying neuropathy at all. Any deterioration of the neuropathy is probably coincidental to the intervention.

SUNDERLAND CLASSIFICATION OF NERVE INJURY (1951)

- Grade 1:** Conduction along axon is interrupted, but axon is intact. Function loss is variable and recovery occurs in days and weeks. Prognosis is good. Motor loss exceeds sensory losses. Order of sensory loss from most severe to least severe injury is proprioception, touch, temperature, and pain. Recovery is in reverse order. Paresthesia lasting several days follows. Sympathetic function is durable, and returns soon if affected at all. Characteristically the distal axon can be stimulated. Also characteristic is the simultaneous return of both proximal and distal motor function. Full recovery is usual.
- Grade 2:** Axon is disrupted but the Schwann cell basal lamina forming the endoneural tube is intact. Full motor and sensory loss occurs initially. Axons can recover within their own neural tubes. Some axons and their Cell somas never recover. Motor recovery progresses from proximal to distal and near full recovery is a feature.
- Grade 3:** The neural tube is disrupted and the perineurium is disrupted. This leads to disorganization with regenerating axons entering “incorrect” neural sheaths. Recovery is slow and is incomplete.
- Grade 4:** The endoneurium is disrupted, but also some of the epineurium and perineurium is disrupted. Retrograde degeneration is severe and many soma die. Scar tissue is all that approximates the trunk ends of the nerve. Scar also hinders axons re-entering distal endoneural sheaths. Prognosis for recovery without surgery is poor.
- Grade 5:** There is total transection of the nerve trunk, usually associated with open wounds. No recovery occurs.
- Grade 6:** (added by McKinnon) A mixture of Sunderland grades injury within one trunk. Recovery is mixed and poor.

INVESTIGATION OF NERVE INJURIES

The patient with a suspected nerve injury after regional anaesthesia needs (1) a neurological assessment to exclude generalized neuropathy, (2) full clinical examination to exclude systemic diseases, and (3) Neurophysiological studies.

There are two electrophysiological test groups;

- A. **NERVE CONDUCTION VELOCITY TESTS.** Stimulating electrodes are placed both proximal and distal to the injury site, as well as sensing electrodes further distal on a target muscle, of the nerve under investigation. Proximal axon function, as assessed distally, will be absent until axonal integrity is restored. Immediately after injury the distal axons will conduct normally, but the distal axons will cease to show conduction from 5 to 10 days after axonotmesis and neurotmesis due to Wallerian degeneration of the axons distal to the injury site. With neuropraxia distal axon function remains normal throughout and prognosis is good for spontaneous recovery. Accordingly it is essential to perform these tests well within 10 days of the suspected injury as they will determine good or poor prognosis early. By shifting electrodes the site of injury can be determined. Injury site may not correspond with the nerve block site.
- B. **ELECTROMYOGRAPHY.** Spontaneous resting myoelectrical activity is assessed with needle electrodes will be normal after acute injury. Activity will be abnormal if there an older injury. This assists diagnose cause-and-effect relationship when litigation is a possibility. Abnormal spontaneous rest potentials appear later after injury; (1) *Positive sharp waves* appear between 10 to 14 days later in myotomes corresponding with injured axons, (2) *fibrillations* appear between 14 and 18 days after injury and remain present until recovery or degeneration of the muscle into fibrosis over years. There is general amplitude increase of potentials between 2 and 6 months after injury as neuronal sprouting occurs with reinnervation, after which the patterns normalize.

The sympathetic nervous system preservation can also be tested with the sweat test, and skin resistance testing. Active sympathetic function indicates moderate or less neuropraxia. Absence sympathetic function means severe neuropraxia or severe injury. In the case of severe neuropraxia, sympathetic function returns before sensory function and motor function returns very late. .

The objectives of electrophysiological tests for nerve deficit associated with nerve blocks is thus;

1. Determine the congruence of the nerve injury site with the nerve block site.
2. Determine if proceeding nerve damage existed.
3. Determine the prognosis of the nerve injury.
4. Assist in the decision whether to operate the injury or not (relieve some anatomical factor causing compression, or anastomose the nerve ends).

NEUROTOXICITY OF DRUGS.

1. Injection of incorrect drugs. The possibilities are endless for erroneous injection of drugs. The most catastrophic anecdotal case report drug known to the author, was injection of calcium chloride misidentified as saline for dilution of the local anaesthetic. It caused an untreatable total nerve loss, with sloughing of all the overlying skin. Most drug errors may be unrecognized.
2. Local anaesthetic drugs. There is the intriguing phenomenon that local anaesthetic drugs are neurotoxic in isolated free floating animal nerves in ex-vitro laboratory studies. Lidocaine in particular can fully destroy a bullfrog nerve in 15 minutes bathing in 1.5 % concentration. Bupivacaine 0.75% in the same study caused a 50% permanent loss function loss. Factors associated with local anaesthetic toxicity are added adrenaline, added glucose, higher concentrations, long duration exposure, and removal of the nerve from surrounding support tissues with their regular blood supply. There may be calcium mechanisms involved as use of calcium channel blockers protects against local anaesthetic induced neuronal injury in the laboratory research. Outside of the phenomenon of Transient Neurological Irritation seen with use of high concentration lignocaine in dextrose and spinal anaesthesia, common clinical use of local anesthetics is seemingly totally devoid of evidence of neuronal injury. No single clinical study on perineuronal infusions even hinted LA infusions cause nerve damage. All nerve injury problems seem to be due needle insertion and injection and never the infusion of local anaesthetic in clinical settings. It seems the viable surrounding tissues and active nerve blood supply prevent intra-neuronal concentrations getting as high as those seen in free floating nerve in laboratory animal studies.

3. Indirect neurotoxicity of systemic drugs. These many drugs are associated with peripheral nerve injury, especially in disease states like renal failure. Examples are oncology drugs, penicillins, other beta-lactam antibiotics, small-pox vaccine, mercury, indocyanine green, thalidomide, organophosphor esters, isoniazid and methanol. Cisplatin, oxaliplatin, carboplatin cause painful neuropathy. Vincristine, taxol, and suramin cause a mixed sensorimotor neuropathy with or without involvement of the autonomic nervous system. Neurotoxicity depends on the total cumulative dose and the type of drug used. Be cautious performing regional anaesthesia simultaneous to the administration of these drugs.
4. Direct neurotoxicity of systemic drugs not usually injected onto nerves. Neurotoxicity has been seen with midazolam, amitryptaline, and the preservative sodium bisulphate. Intraneuronal pethidine (mepridine) was not neurotoxic in studies. It is advisable NOT to experimentally administer drugs onto nerves despite some theoretical hope of a unique benefit, until animal and human volunteer studies exclude the possibility of direct neurotoxicity. Remember there is no other tissue in the body where such high drug concentrations are achieved, as with nerves after direct injection onto the nerve so the effects of drugs under these high local concentrations may be bad, despite apparent safety when administered IV or orally.

PHYSICAL INJURY TO NERVES

This is a major cause of injury in the peri-operative period. It is due to patients taking up unusual positions for long periods. Persons taking up unusual positions while awake or during physiological sleep, normally continuously reposition themselves.

Animal experiments show that stretching lowers the nerve vulnerability to compression injury greatly.

After shoulder surgery there can be up to 30% brachial plexus transient injuries unrelated to nerve blocks. A concurrent nerve block would seem to be more likely innocent should the patient get a neuropraxia. Other limb surgeries are also associated with neuropraxias.

Many old studies indicate that intraneuronal injection of drugs are particularly damaging compared to peri-neural injection of the same drug. This was observed with various non-local anaesthetic drugs. The evidence of damage with local anaesthetic intraneuronal injection is however lacking, but it has been suggested to avoid injecting adrenaline containing local anaesthetics intraneuronal.

In animal research, intra-fascicular injections cause far worse damage than extrafascicular injections regardless of drug injected. Some study drugs were neurotoxic intrafascicular but harmless extrafascicular. Also is the thickest myelinated nerve fibres that are most susceptible to injury, and the thinnest and unmyelinated fibres the least.

The debate on whether intentional soliciting of paresthesia (in humans) with nerve block injuries is harmful or not, rages on. In the era before electro-locator needles were available it was said “No paresthesia no anaesthesia”. The use of electro-locator needles has not been proven to have reduced nerve injuries incidences in a clinically significant degree. Studies also have proven that to elicit paresthesia the needle tip has to be intra-neuronal. It thus seems that intraneuronal injection of local anaesthesia is in the large majority of patients without ill consequence as this was standard practice for 80 years. Placing a needle intra-neuronal may be different to additionally injecting intraneuronal drug. Also intra-neuronal injection itself may have different effects at different nerve sites and in different patients. This may depend on the local nerve surrounding tissues and the local nerve health to dissipate the drug and the effects they have on the nerve physiological milieu and also in what time period the nerve is restored to its healthy energy supply state. This is argument to be extra cautious about high pressure injections in the “problem” patient with neuropathy or some other disease. It is also apparent that injection of local anaesthetic perineural results in perfect conduction block anaesthesia and intra-neural injection of local anaesthesia is not essential to successful nerve blockade.

HOW TO AVOID INTRANEURONAL INJECTION

This is probably the biggest source of conflict of opinion amongst regional anaesthesia experts. The following options exist;

1. Using the patient’s degree of pain as indicator of damaging intra-neuronal injection. There is no single trial or scientific data offering guidance here. This is not possible in veterinary anaesthesia.
2. Use the lowest stimulating current as indicator of intraneuronal positioning of the needle. It is widely believed that when a needle can elicit a motor response with a current of 0.2 mAmp or lower that it is probably within the nerve. Withdraw the needle until 0.2 mAmp current does NOT elicit a motor twitch, but keep the needle close enough so that a current of 0.6 mAmp or less DOES stimulate the nerve. This is probably an **important indicator** of potential intraneuronal injection. This is especially important when the injection volume will be large (exceed 10 ml) and large proximal nerves are being injected. It seems this advice does not apply to round tip nerve catheters (Stimucath) placed onto (? into nerves) as currents of down to 0.05 mAmp have often been seen after easy catheter placement with no injury following on the drug injection.
3. Avoid paresthesia techniques of nerve location. Use nerve stimulators as in point 2 above. The Urmev and Choyce studies prove that paresthesia nearly only occurs with an intra-neuronal needle tip position.
4. Avoid repeated needle insertions into a nerve. Abandon difficult nerve blocks. The worst injuries are associated with persistent stabbing with a needle.
5. Avoid added adrenaline
6. Avoid added corticosteroids especially hydrocortisone and triamcinolone. Dexamethasone seems safer.

7. Avoid injecting nerves in poor compliance areas where injection pressure may become high due to of the surrounding tissues (tough osseo-fascial tunnels) inability to expand or dissipate the injected volume.
8. Avoid high pressure when doing nerve block injections. This is in case an intraneuronal needle placement is unrecognized. Thin needles with long thin injection side-tubing are good, because they only permit slow injection. Research shows nerve damage occurs if intra-neuronal pressures exceed certain thresholds. It is possible that intraneuronal injection in some (maybe smaller) nerves and in some sites is harmless as the perineurium is sufficiently fragile to allow easy extra-neuronal transfer of the drug. It is possible that most intra-neuronal injections are unrecognized and inconsequential. However when a regional anaesthesia technique reliably suggests intra-neuronal injection then it is best avoided. An injection rate under 20 ml per minute is also recommendation. It is also known that the perineural “sheath” around nerves is thicker at some places than in others. The sheath is probably thickest, probably where the nerve will slide with limb movement. At such places large volume intra-neuronal injection may be less safe than intra-neuronal injection at other sites at other sites where flimsier supportive tissues allow drug escape to peri-neural planes. Even short periods of raised intraneuronal pressure, cause neuronal oedema which can last 24 hours. Nerves have no internal lymphatic drainage so oedema is sustained. Neuronal oedema is associated with axonal degeneration and demyelination. This oedema sustains the intraneuronal pressure for hours after the causative pressure source is removed. Intraneuronal blood flow can be reduced for two weeks after a pressure induced injury. Experimentally an intraneuronal pressure of 118 mmHg completely stops intra-neuronal blood flow and causes an immediate conduction block, that reverses with release of the pressure. Keeping intra-neuronal pressure down is achieved by
 - a. Ceasing injection if there is unusual resistance to injection.
 - b. Using a nerve stimulator to identify intra-neuronal needle tip positions.
 - c. Avoid performing nerve blocks in zones the surrounding tissues have poor compliance (e.g. ulnar nerve groove of humerus)
9. Avoid sharp shallow bevel needle for nerve blocks. These needle cut nerve fibres more easily than blunter needles. Microscopy research by Selander suggested the blunter steep bevel type needles (as in common nerve locator designs) cause more extensive ragged type trauma than sharp needles when forced into nerves in laboratory studies. However thought these clinically blunter needles are more likely to also push a nerve aside rather than penetrate and remain preferred. Standards insulated nerve locator needles are only available with shallow bevels and have been used in millions of nerve blocks with acceptable results.
10. Use aseptic techniques.
11. Beware of nerve plexitis. When regional anaesthesia is planned for the shoulder surgery, beware of patient complaints of pain at the elbow and downwards. This may indicate a pre-existing brachial plexitis. While the presence of such plexitis does not absolutely contra-indicate a nerve block, it is advised that the symptoms be documented, and the patient be informed that change in symptoms will more likely be due to disease progression than from the local anaesthetic. Also beware the patient coming for multiple repeat surgery, who has a high risk of having an unrecognized CRPS and may have persistent pain complaints after surgery related to the CRPS.
12. Use needles of shortest possible length needle to perform the block. This is a rule for fools. One of the causes of greatest interscalene brachial plexus complications is inexpert doctors battling with long needles that reach into the spinal chord via the intervertebral foramen. Experience, knowledge and good teaching are far more important.
13. Fractionate large volume injections. This facilitates both (1) timeous recognition of local anaesthetic toxicity before the injected dose is fatally large, (2) allows multiple aspiration checks for IV injection, and (3) slows the total injection rate allowing drug dissipation keeping intra-neuronal pressure low.

ADDITION OF ADRENALINE (EPINEPHRINE) TO L. A. SOLUTIONS.

A 1 in 100 000 solution reduces nerve blood supply 35%. Adrenaline significantly extends the duration of lignocaine infiltration nerve blocks, but hardly influence the long acting local anaesthetics. The adrenaline causes vasoconstriction of the support tissues of the nerve thus delaying removal of the drug by

absorption into capillary blood vessels. Anecdotally there is a suggestion that addition of adrenaline increases the risk of nerve injury.

It seems thus logical to avoid use of adrenaline in all local anaesthetic infusions, and also in all solutions used for major direct nerve block. Adrenaline may however be safer and also beneficial when added to infiltrative field type blocks or to cavity blocks such as the intra-peritoneal block and the intra-pleural block.

SHOULD NERVE BLOCKS BE DONE IN THE PRESENCE OF NEUROPATHY

Examples of causes of neuropathy are diabetes mellitus, Charcot-Marie tooth disease, and Systemic Lupus Erythematosus. No definitive evidence exist of regional anaesthesia causing a problem, Many case reports utilize regional anaesthesia as a option rather than general anaesthesia which has its own associated morbidity/mortality.

MANAGING A NERVE INJURY AFTER NERVE BLOCK

DEFINITION OF NERVE INJURY = a limb that after a nerve block remains paralysed or anaesthetic for longer than expected, or there is pain in a neurotome not corresponding with surgery after the surgical pain is resolved. .

1. Reassure animal owner that some nerve blocks take longer. Keep contact and interest.
2. Prescribe a NSAID from the earliest time possible, if safe.
3. Personally examine patient. Seek signs of sympathetic nerve damage (it will predict very slow recovery, inform patient). Seek signs of other mechanisms of nerve injury other than the nerve block.
4. Arrange for neuro-physiological studies (to be done before 6 – 10 days after nerve block have passed).
5. Consult neurologist.

Indications for neurolysis; If a known “wrong drug” was accidentally administered into nerve early neurolysis as strongly advised, as it will accelerate the rate of healing. Some benefit will result from neurolysis in such cases even if done weeks and months later.

Neurolysis is generally not recommend after local anaesthetic nerve block associated injury, unless it is the severest neuropraxia type injury (signs of sympathetic nerve damage) and no signs of recovery at all are seen after many days .

Neurolysis that opens the perineurium (outermost layer) is safe and harmless generally, however fascicular neurolysis may disrupt the complex intra-neuronal blood supply and loose more than it could gain.

SUMMARY:

When a peripheral nerve injury is seen after regional anaesthesia it is often unclear whether there is underlying nerve disease, a surgical cause or other unidentified cause. The fact a nerve block was performed does not automatically implicate it as a negligent cause.

Routine easy correctly performed nerve blocks without negligence are rarely associated with injury. The incidence of catastrophic nerve injury after regional an aesthesia approaches near zero incidence and the incidence of non-incapacitating nerve injury one in tens of thousands, of cases.