5 June 2009

Regional Anaesthesia in Patients with Pre-existing Neurological Disease

B Daya

Commentator: H Ekwueme

Moderator: AD Maharaj

Department of Anaesthetics

CONTENTS

INTRODUCTION

The use of regional anaesthesia (RA), either alone or as an adjunct to general anaesthesia, is at an all-time high. The benefits of regional anaesthesia are well documented and include reduced patient morbidity and mortality, superior postoperative analgesia with enhanced patient satisfaction, and enhanced cost effectiveness.

Despite being extremely rare, neurological complications, including permanent neurological injury, are complications feared by both patients and anaesthesiologists. There is also concern that pre-operative neurological and neuromuscular diseases may be exacerbated or worsened following regional anaesthesia; hence present potential management dilemmas for anaesthesiologists.

Historically, the recommendations of Vandam and Dripps in 1956 *(1)* to avoid spinal anaesthesia in patients with pre-existing central nervous system disorders have greatly influenced the clinical management of patients for several decades.

However, the rarity of these conditions results in a paucity of data that are often conflicting. Existing scientific literature and expert opinion can neither confirm nor refute adverse effects of regional anaesthesia in patients with pre-existing neurological disease.

Therefore, the decision to use regional anaesthesia in these patients is determined on a case-by case basis, and involves the understanding of:

1) the overall incidence of neurological complications after regional techniques.
2) pathophysiology of neuraxial and peripheral nerve injury
3) pathophysiology of neurological disorders.

This review aims to address the issues mentioned above, and provide guidelines regarding the perioperative management of patients with underlying neurological disease.

INCIDENCE OF REGIONAL ANAESTHESIA-RELATED NEUROLOGICAL INJURY

Because of the infrequency of which anaesthesia-related neurologic injuries occur (or are reported), it is extremely difficult to obtain reliable and consistent incidence data. Medicolegal and insurance-based data are biased by the presence of injury, hence may overestimate the occurrence. Furthermore, underreporting may bias clinical-based data, hence may potentially underestimate the true incidence.

A comprehensive study of neurological complications after central neuraxial (CNB) block was published in 2004. *(2)* This retrospective study from Sweden between 1990-99 included 1 260 000 spinals and 450 000 epidurals being performed. The authors reported an overall frequency of severe neurological complications (without including epidural haematoma and abscess) after spinal of 0.4 per 100 000 and after epidural 1.6 per 100 000.

A prospective study from France between 1998-1999 included 41 079 spinals and 35 293 epidurals. *(3)* The authors reported an incidence of severe neurological complications after spinals to be 3.7 per 100 000, and 0.3 after epidurals.

It is clear that in the recent years that randomized-controlled-trials and meta-analyses have led to conflicting conclusions and interpretations regarding the risks and benefits of central neuraxial block techniques. The National Audit Project performed by the Royal College of Anaesthetists over 12months, between 2006 and 2007, identified the performance of 707 455 central neuraxial blocks. *(4)*

Major complications of central neuraxial block: report on the Third National Audit Project of the Royal College of Anaesthetists†

T. M. Cook[1]*, D. Counsell[2] and J. A. W. Wildsmith[3] on behalf of The Royal College of Anaesthetists Third National Audit Project

The authors identified an incidence of 2.0 – 4.2 per 100 000 patients of permanent injury due to CNB.The incidence of epidural and combined spinal-epidural techniques were twice those of spinals and caudals. This large prospective study provides some re-assurance ,with the incidence of permanent injury being lower than other related studies; and almost complete recovery in majority of the patients experiencing nerve injury.

Table 4 Summary of cases reviewed and their classification by review panel. Exclusion from review was due to wrong diagnosis, minor injury, full recovery before notification, and procedure performed outside the dates of the audit or in a non-NHS hospital. See text for definitions of 'pessimistic' and 'optimistic' categories

Category	Total	Excluded from review	Excluded from incidence calculation: full recovery during follow-up	Included: pessimistic incidence calculation	Included: optimistic incidence calculations
Epidural abscess	20	5	7	8	3
Meningitis	6	3	3	0	0
Vertebral canal haematoma	8	2	1	5	4
Nerve injury	18	4	7	7	3
Spinal cord ischaemia	6	2	0	4	0
Wrong route error	11	10	0	1	1
Cardiovascular collapse	6	0	3	3	2
Miscellaneous	9	6	1	2	1
Total	84	32	22	30	14

There are a limited number of contemporary prospective studies examining the risk of neurological injury after peripheral nerve blocks (PNB). Most of the available data involves upper extremity PNB, reflecting the preference for brachial plexus blockade in contemporary regional anaesthesia practice, with axillary and interscalene blocks carrying the highest risks of transient neurological deficit.

In the 2 large prospective studies performed by Auroy et al, the incidences of neurological injury were 3.8 per 100 000 in 1997 and 2.7 per 100 000 in 1999, with persistence of symptoms more than 6months in seven out of the twelve patients in the latter study. *(3)(5)*

Unfortunately, however, there is insufficient detail or research done to determine the overall frequency of permanent neurological deficit with PNB.

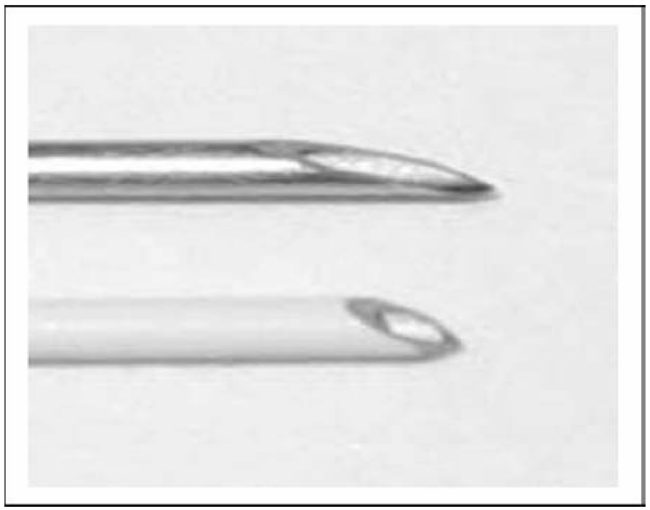
The incidence of neurological injury with pre-existing neurological disease is unknown; however, as the practice of regional anaesthesia continues to gain popularity, knowledge of the risk of neurological injury associated with the most common RA techniques is imperative.

PATHOPHYSIOLOGY OF NEURAXIAL INJURY

Neuraxial injury may involve the spinal cord, spinal nerve roots, spinal nerves or spinal vasculature. Neuraxial injuries are linked to mechanical damage due to needle or catheter techniques, mass lesions within the spinal canal, ischaemia, drug-related neurotoxicity.

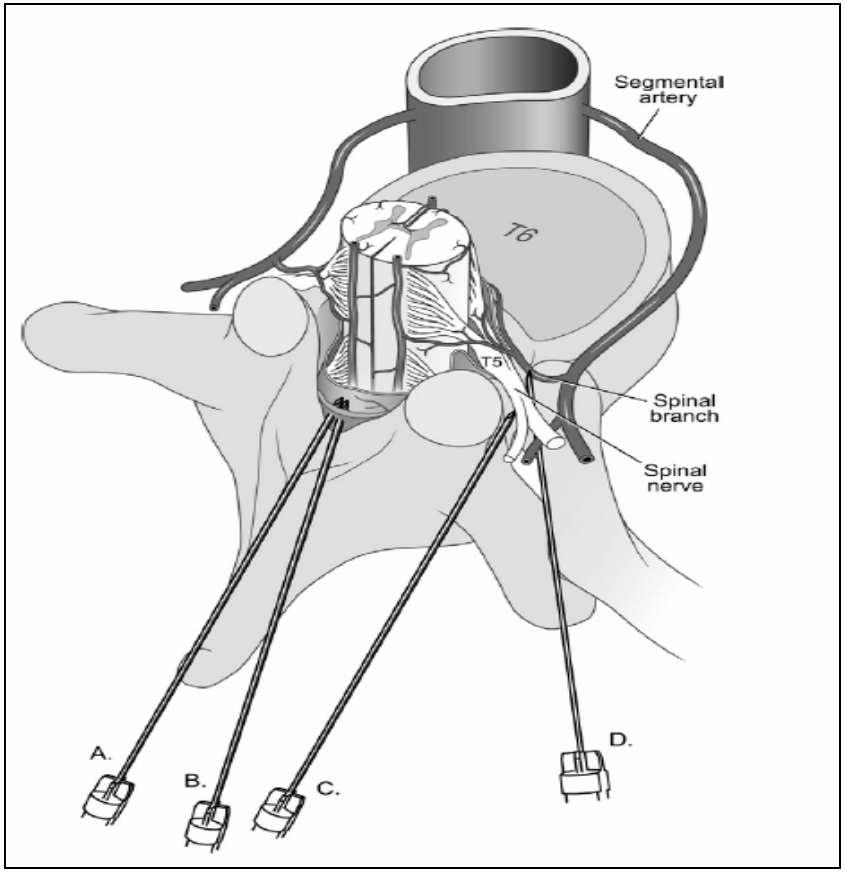
a) Mechanical damage
This may be associated with anatomical variations in the terminal portion of the conus medullaris, abnormalities of the ligamentum flavum and epidural space.

Figure 1 A comparison of 21 gauge Quincke (upper) and 'B-bevel' 45° (lower) needles



The 'B-bevel' (lower) needle is blunt enough to make direct nerve penetration less likely, as per D.E. Selander [7].

The use of 12-15 degree long-bevelled needles (upper needle) is thought increase the risk of neural injury.
Injection techniques may also cause mechanical injury to neuraxis:



Laterally directed needles (C) may injure spinal nerves or blood vessels near the medial aspect of the intervertebral foramina.
Transforaminal medially directed needles (D) may also injure the same structures.

Needle entry into the spinal cord may not be recognized as it has no sensory receptors and sensory input from the meninges is inconsistent. Actual injection may elicit pressure-related paraesthesia.

b) Neural Ischaemia

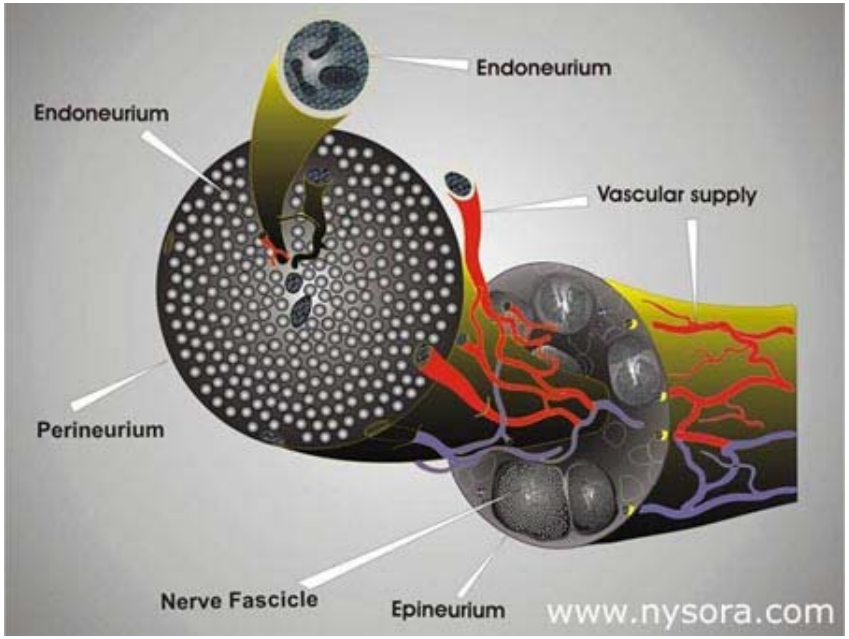
Impediments of blood flow to nerves may occur due to high tourniquet inflation pressures, systemic hypotension and increases in intraneural pressure. The use of vasoconstrictors, for example adrenaline, theoretically reduces spinal cord blood flow.

c) Local Anaesthetic Toxicity

Local anaesthetics are seldom neurotoxic when given in recommended doses. The potential for toxicity is heightened when mechanical damage to the protective outer connective tissue barriers occurs; when injection or catheter insertion physically disrupts the structural integrity of the spinal cord; or when vasoconstrictors impede local anaesthetic clearance.

Theoretically, the cauda equina is more susceptible to injury as it is partially unmyelinated and has a large surface area.

PATHOPHYSIOLOGY OF PERIPHERAL NERVE INJURY



Anatomy of a Peripheral Nerve

Peri-operative peripheral nerve injury can occur during general and regional anaesthesia, and as a consequence of surgical nerve injury, postural compression or traction, and neural stretch. Furthermore, similar to neuraxial injury, it is commonly linked to mechanical trauma from needles or catheters, ischaemia and drug toxicity.

The prognosis of peripheral nerve injury depends largely on whether the axon is preserved.

Neuropraxic lesions, which damage the myelin sheath(axon preserved) are associated with stretch or compressive injuries and are more likely to resolve. However, if the axon is disrupted, recovery is slower and may be incomplete.

The "Double-Crush" theory suggests that patients with pre-existing peripheral nerve damage are likely to sustain further damage if a second injury occurs.

Peripheral nerve injuries require the breach of connective tissue barriers (perineurium) which surround the fascicle. Disruption of the perineurium with a needle or catheter is difficult, and is compounded by ultrasound-guided evidence showing that nerves tend to move away from approaching needles. However, if penetration of the fascicle occurs, neurons are exposed to local anaesthetics that can cause time and concentration-dependent injury.

It is important to note that the recommendations made decades ago have greatly influenced the clinical management of patients with neurological diseases. The risk of mechanical trauma, local anaesthetic toxicity or neural ischaemia, combined with chronic underlying neural compromise, was thought to place patients at an increased risk of further neurological injury.

However, the aetiology of postoperative neurological deficits is often difficult to evaluate because of the many patient, surgical and anaesthetic risk factors that may play a role:

eg. Patient age or body habitus;
intra-operative surgical trauma;
tourniquet inflation pressures;
prolonged or difficult labour;
improper patient positioning or
anaesthetic technique.

Furthermore, progressive CNS diseases, eg multiple sclerosis (MS) may co-incidentally worsen peri-operatively, independent of anaesthetic or surgical technique.

Therefore the abundance of contributing factors makes it difficult for clinicians to reliably isolate the effect of anaesthetic technique on neurological outcome.

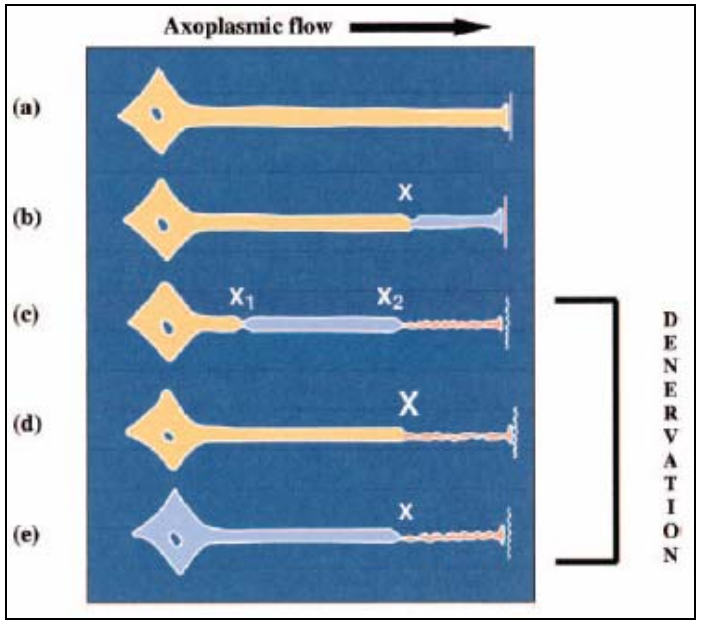
REGIONAL ANAESTHESIA AND THE PATIENT WITH PRE-EXISTING NEUROLOGIC DISEASE

Pathophysiology

Pre-existing disorders of the CNS and PNS present challenges to patients and anaesthesiologists.

Upton and McComas *(15)* first described the "Double Crush" Phenomenon in 1973, and suggested that patients with pre-existing neural compromise may be more susceptible to injury at another site, when exposed to a secondary insult.
Secondary insults may include a variety of mechanical, ischaemic or toxic risk factors associated with regional anaesthesia.

Osterman *(16)* emphasized that not only are 2 low-grade nerve insults along a peripheral nerve worse than a single insult, but the damage far exceeds the expected additive damage caused by each isolated insult.



Double-Crush Phenomenon

(a) Normal neuron
(b) Mild neuronal injury single site (X)
(c) Mild neuronal injury at 2 sites (X1 & X2) – may cause distal denervation
(d) Severe neuronal injury at a single site (X) – may cause distal denervation
(e) Axon with diffuse, pre-existing underlying disease with impaired axonal flow – predisposes axon to distal denervation following single minor neural insult at (X) (ie "double crush")

It was concluded from these studies that the performance of regional techniques in patients with pre-existing CNS/PNS disorders, may theoretically increase the risk of a double-crush phenomenon.

However, the current literature, despite often being limited to small retrospective studies and case reports, neither confirms, nor refutes the safety of neuraxial or peripheral nerve blocks in these patients. It also does not definitely address the relative safety of spinal versus epidural anaesthesia or analgesia.

Furthermore, the data is often conflicting:

Neuraxial block and Pre-existing Neurological Disease

A retrospective investigation of 139 patients with pre-existing CNS disease (multiple sclerosis, post-polio syndrome, spinal cord injury), who underwent neuraxial anaesthesia and analgesia, was published in 2006. *(17)*

96% of patients had underlying neurological conditions which had been stable for at least 6months. Bupivicaine 0.75% at a mean dose of 14+/-2mg was the most commonly used drug for spinals. Results showed that a worsening of neurological condition was not observed in any patient at a 3 month postoperative follow up. (0%-0.3% 95%CI).

Another retrospective study of neuraxial anaesthesia in 567 patients with pre-existing sensorimotor neuropathy or diabetic polyneuropathy was performed. The authors noted the onset of new or progressive neurological deficit in 0.4% (0.1% -1.3% 95%CI) of patients – an incidence which is higher than expected within the general population.

Enthusiasm related to favorable outcomes must be tempered by adverse outcomes reported by case series by Aldrete et al in 2004, and Bader et al in 1998.

Aldrete *(19)* reported 7 cases of unrecognized or undiagnosed spinal or cerebral symptoms which may have been exacerbated by neuraxial anaesthesia techniques.

Bader noted an increase in relapse rate of multiple sclerosis after spinal anaesthesia in surgical patients versus a statistically similar incidence of relapse rate in pregnant women receiving neuraxial analgesia and anaesthesia.

Peripheral Nerve Blocks and Pre-existing Neurological Disease

Peripheral sensorimotor neuropathies may occur secondary to a variety of underlying aetiologies including metabolic, autoimmune, infectious or hereditary abnormalities.

Conditions that may Predispose Patients to Neuropathies:

Acromegaly
Amyloidosis
Carcinoma
Cryoglobulinaemia
COPD
Diabetes Mellitus – most common
Drugs – incl cancer chemotherapeutics (eg vincristine,cisplatin)
Hypothyroidism
Liver disease
Lymphoma
Multiple Myeloma
Porphyrias
Uraemia

Wide clinical experience suggests that neural blocks in these patients rarely exacerbate pre-existing nerve injury.

A study reporting the presence of underlying sensorimotor peripheral neuropathy in 18 of 607 patients undergoing axillary block showed that none of the 18 patients developed postoperative neurological symptoms.

Dhir et al reported successful outcomes with 4 ultrasound-guided axillary and brachial plexus blocks in 3 patients with Charcot-Marie-Tooth disease.

However, recently Koff et al *(20)* reported a case of severe brachial plexopathy in a patient with multiple sclerosis, after an ultrasound-guided interscalene block.

In addition, another case report describes a patient with presumed subclinical neuropathy from cisplatin, 48 hours after an interscalene block with 0.5% bupivicaine with adrenaline was performed.

Unfortunately this relatively sparse and conflicting evidence leaves the anaesthesiologist with more questions than answers:
Who is right and who is wrong?

Is it reasonable to assume a small but unknown added risk of performing regional anaesthesia in patients with underlying inflammatory or congenital neurological disease?

Will ultrasound-guided techniques offer a higher degree of patient safety?

REGIONAL ANAESTHESIA CONCERNS FOR SPECIFIC NEUROLOGICAL CONDITIONS

Regional Anaesthesia and Previous Spinal Surgery and Chronic Back Problems

Patients with a history of prior spinal surgery present unique challenges for neuraxial techniques. Most instrumentation involved in spine stabilization and fusion does not alter the architectural integrity of the posterior spinal elements, making neuraxial techniques frequently possible. Plain radiographs and fluoroscopy can be beneficial in planning neuraxial techniques. Because of surgically-induced epidural space narrowing, local anaesthetic spread may be abnormal in these patients, thus spinal anaesthesia may be preferred, when appropriate.

Reports have recently raised growing concerns about neuraxial techniques in patients with spinal stenosis (or suspicion of compressive aetiology). Cauda equina, persistent motor deficit and sensory deficit have been reported after neuraxial blocks in patients in whom the diagnosis of asymptomatic spinal stenosis was evidenced postoperatively.

Major Spinal Cord Injury

The use of RA offers the advantage above GA of less haemodynamic perturbations especially in patients with autonomic hyperreflexia. A titratable RA technique has been used most frequently in obstetric patients, although success is variable. Determination of the sensory block level may be difficult; autonomic hyperreflexia may indicate wrong catheter position.

Myelopathies : Multiple Sclerosis

Multiple sclerosis (MS) is a chronic, degenerative disease of the central nervous system, characterized by focal or segmental demyelination within the brain and spinal cord. This produces a conduction blockade which fluctuates in severity, resulting in a characteristic waxing and waning disease course.

The use of regional anaesthesia or analgesia in patients with MS has been debated for some time.

Local anaesthetics given systemically, neuraxially or locally may transiently worsen or unmask MS symptoms. One postulated explanation is that the lack of a protective nerve sheath around the spinal cord may render it more susceptible to the potential neurotoxic effects of local anaesthetics.

Although epidural anaesthesia or analgesia has been used safely in obstetric patients with MS, there are case reports of worsening of symptoms. Hence, although not specifically contra-indicated, neuraxial anaesthesia using local anaesthetics should be used with caution in these patients.

Peripheral Neuropathies: Diabetes Mellitus

Distal symmetrical sensorimotor neuropathy is the most common neuropathy associated with diabetes mellitus. It involves axon loss in peripheral nerve trunks and possibly spinal cord. Diabetic nerve fibres may be more susceptible to the toxic effects of local anaesthetics because of:

i) they are exposed to larger concentrations because of decreased blood flow.
ii) the nerve is already ‘stressed’ by chronic ischaemic hypoxia secondary to microangiopathy.
iii) addition of epinephrine may reduce nerve blood flow.

Neuromuscular Disorders: Myasthenia Gravis

Myasthenia gravis is an autoimmune disease resulting in the destruction of postsynaptic acetylcholine receptors at the neuromuscular junction, causing varying degrees of weakness and fatiguability. These patients may be challenging to he anaesthesiologist as both the disease process and medications used to treat it cause difficulties in anaesthetic management.

Central neuraxial blocks have been used, however weak concentrations of local anaesthetic, causing the least motor impairment, should be selected. Peripheral nerve blocks are also not without risk, and respiratory distress due to phrenic nerve block after interscalene block has been reported.

Amide local anaesthetics are theoretically better choice than esters since amide local anaesthetic metabolism is unaffected by cholinesterase.

Pregnancy has an unpredictable effect on myasthenia and exacerbations should be anticipated.

Myopathies

Little is known about RA techniques in patients with myopathy. RA will not treat nor prevent the occurrence of myotonic contractures. Spinal and epidural anaesthesia has many advantages over general anaesthesia, although technical difficulties may be experienced owing to scoliosis. Another point to consider is that the potential myotoxicity of local anaesthetic agents on abnormal muscles is unknown and needs further clarification.

CLINICAL APPROACH TO REGIONAL ANAESTHESIA IN PATIENTS WITH NEUROLOGY

Pre-Operative Evaluation

1. A careful history and physical examination, including neurological examination and review of relevant investigations, must be performed.

2. Clear documentation of the region of pathology and nerves or dermatomes involved.
   Distribution of weakness, sensorimotor loss and other neurological symptoms must be documented.

3. Informed consent: patients must be informed of potential technical difficulties; and possible relapse or progression of the disease, associated with the combination of stress, surgery and anaesthesia.

4. Case-by-case analysis of the risk-benefit ratio regarding the choice of anaesthetic technique must be considered.

Intra-operative Management

A: Anaesthetic Considerations

1. Minimize needle trauma and intraneural injection. This is facilitated by the use of blunt level block needles.
   The safety of catheter techniques in these patients has not been determined.

2. Smaller volumes of less potent local anaesthetic solutions should be used when feasible, to decrease the risk of local anaesthetic toxicity with pre-existing neural compromise.

3. The use of vasoconstrictor agents eg epinephrine-containing solution in patients with pre-existing neurological deficit is controversial.

   The potential risk of vasoconstrictor-induced nerve ischaemia must be weighed against the advantages of improved quality and duration of the block.

Because the vasoconstrictors prolong the block and neural exposure to local anaesthetics, the appropriate concentration and dose of local anaesthetic solutions must be considered.

ASRA* Recommendations on Limiting Neuraxial Injury

- Careful evaluation of vertebral level and avoidance of lateral needle placement, especially in patients with challenging anatomy.
- Initial dosing or redosing of subarachnoid local anaesthetics greater than the maximum recommended dose may increase the risk of spinal cord or nerve root toxicity, and should be avoided
- Avoid neuraxial blocks in patients with space-occupying extradural lesions (eg. Severe spinal stenosis)

ASRA Recommendations Regarding Peripheral Nerve Blocks

- Nerve Localization techniques:
  - Avoid elicitation of paraesthesia
  - No data to support the superiority of one technique over another ie nerve stimulator or ultrasound
  Ultrasound does not prevent intraneural and hence possible intrafascicular injection.
  Monitoring injection pressures and/or impedence may be other alternatives.
- For PNB proximal to the spine (interscalene, psoas, paravertebral blocks), avoid orientation of the needle and/or inserting the catheter towards the spine.
- Avoid flexible needles
- If damage to the perineurium is suspected from an abnormally painful paraesthesia or pain on injection of local anaesthetic, consider needle repositioning or aborting the procedure.

B: Surgical Considerations

1. Patient positioning must be considered to limit further nerve injury.

2. Tourniquet us may cause nerve damage by ischaemia or mechanical deformation or compression of nerves; especially of fast-conducting myelinated fibres.

High inflation pressures (>400mmHg) and long tourniquet times increase the likelihood of tourniquet-induced-neuropathy.

In patients with pre-existing neuropathy, it may be prudent to forego the use of the tourniquet, if possible, or alternate between 2 cuffs.

Postoperative Evaluation of Suspected Exacerbation of Pre-existing Neurological Pathology

1. History and Examination
   History of patients pre-existing pathology
   Formal neurological examination, determining the anatomical basis of the damage : motor/sensory/autonomic
   Upper/ lower motor neuron
   Root/plexus/trunk/branch
   Dermatome

2. Electrophysiological Studies: EMG and Nerve Conduction studies
   Early EMG (within first few days postoperatively) may present changes until 2 weeks after nerve injury.
   However, it provides a baseline to which later studies can be compared.
   EMG should be repeated at 2-3weeks and 3-5months postoperatively.

3. Radiological Studies
   MRI and CT are useful for determining any space-occupying –lesions which can be surgically treated.
   New ultrasound or Magnetic Resonance Neurography techniques may be useful in the diagnosis of nerve lesions.

4. Treatment
   Most treatment is supportive.
   Long term rehabilitation and physiotherapy support is the mainstay of treatment for many of the patients.
   Surgical treatment is limited to the early relief of spinal cord compression due to haematoma or epidural abscess.
   Nerve grafting may have a small role where damage is limited to a single nerve.

NERVE BLOCK PROCEDURE RECORD

THE JOURNAL OF THE NEW YORK SCHOOL OF REGIONAL ANESTHESIA (WWW.NYSORA.COM) | UNIVERSAL DOCUMENTATION SHEET FOR PERIPHERAL NERVE BLOCKS

Institution Name:

Nerve Block PROCEDURE RECORD

Date & Time:_____

Referring M.D.:_____

Surgical Procedure:_____

Preoperative Diagnosis:_____

Indication: ☐ Surgical ☐ Pain Management

ANESTHETIZING LOCATION:_____

Patient Name

Medical Record #

Age

(Patient name plate stamp)

☐ **SITE** MARKED: Left/Right ☐ **CONSENT** on chart: ☐ Surgical ☐ Anesthesia ☐ **TIMEOUT** performed

Nerve Block Procedure		**Single**	**Catheter**
☐ **ULTRASOUND GUIDED 76942**	☐ Interscalene	☐ 64415	☐ 64416
	☐ Supraclavicular	☐ 64415	☐ 64416
	☐ Infraclavicular	☐ 64415	☐ 64416
	☐ Axillary	☐ 64417	☐ 64416
	☐ Lumbar Plexus	☐ 64483	☐ 64449
	☐ Femoral	☐ 64447	☐ 64448
	☐ Sciatic/Popliteal	☐ 64445	☐ 64446
	☐ Ankle/Wrist	☐ 64450	
	☐ Paravertebral	☐ 64520	
	☐ Other_____	☐ _____	

ICD-9 Pain Diagnosis		**Technique**
☐ Shoulder	☐ 719.41	☐ Single Injection
☐ Upper Arm/ Elbow	☐ 719.42	☐ Continuous
☐ Forearm/Wrist	☐ 719.43	☐ Tunneled
☐ Hand	☐ 719.44	☐ Ultrasound ☐ Nerve Stimulator
☐ Hip/Thigh	☐ 719.45	
☐ Knee/Leg	☐ 719.46	
☐ Foot/Ankle	☐ 719.47	
☐ Other	☐ _____	

Approach ☐ Anterior ☐ Posterior ☐ Lateral ☐ Other: _____

Monitors: ☐ Blood Pressure ☐ EKG ☐ Other ☐ Pulse Oximetry ☐ ETCO2 _____

Oxygen _____ (L/min) ☐ Nasal cannula ☐ Mask ☐ Other:_____

Premedication (in last 30mins): ☐ Midazolam ___ mg ☐ Propofol ___ mg ☐ Alfentanil ___mcg ☐ Hydromorphone ___ mg ☐ Fentanyl ___mcg ☐ Morphine ___mg ☐ Other _____

Level of Sedation
☐ Patient awake.
☐ Patient sedated. Easily aroused and conversant.
☐ Patient under general anesthesia.
☐ Patient under spinal/epidural/PNB.

Needle: ☐ Manufacturer:_____ ☐ Model:_____ ☐ gauge:_____ Size: ☐ 50mm ☐ 100mm ☐ Other:_____ Catheter: ☐ Stimulating ☐ Non-stimulating

Prep: ☐ Sterile prep ☐ Sterile drape ☐Betadine/Chlorhexidine ☐ Sterile gloves

Local Anesthetic	Type and Concentration	Vol(mL)	Additives
	☐ Chloroprocaine _____% ☐ Lidocaine _____% ☐ Mepivacaine _____% ☐ Ropivacaine _____% ☐ Bupivacaine _____% Other:_____%	_____	☐ Epinephrine (1:___ 00,00) ☐ Bicarbonate (0.1meq/ml) ☐ Other_____

Procedure Notes:
Start time:_____End time: _____Length of Procedure:_____
☐ Skin anesthetized with local anesthetic. Pt Position:_____
Needle depth: ____ cm Minimal current: ____ mA Number of attempts: _____
Type of motor response (describe): ☐ Catheter depth @ skin: ____cm
Blood aspirated ☐ No ☐ Yes- Action Taken:_____
Pain on injection ☐ No ☐ Yes- Action Taken:_____
Injection pressure > 20 PSI ☐ No ☐ Yes- Action Taken:_____

☐Attending performed the procedure **Signature:** (Date & Time)

Print:_____

☐Attending was present for the critical portions of the procedure **Attending Resident(s) Signature:** (Date & Time)

Print:_____

*To bill for US guidance, a permanent image of nerve block should be attached to the documentation. Please document patient name, target nerve, and local anesthetic spread on attached image.

CONCLUSION

Existing scientific literature and expert opinion can neither confirm nor refute an adverse effect of regional anaesthesia in patients with pre-existing neurological disease. However, most existing data suggest that an increased risk, if present, is likely to be of minimal magnitude. Most importantly, patients with pre-existing disease may develop new or progressive during the peri-operative period, independent of anaesthetic technique – from surgical factors, peri-operative stress or underlying comorbidity. These factors must be taken into consideration when deciding on the peri-operative management of the patient with underlying neurological disease.

* = American Society of Regional Anesthesia and Pain Medicine

REFERENCES

(1) Vandam LD, Dripps RD. Exacerbation of pre-existing neurological disease after spinal anaesthesia.
NEJM 1956; 255: 843-849

(2) Moen V, Dahlgren N. Severe neurological complications after central neuraxial blockade in Sweden 1990-1999.
Anaesthesiology 2004; 101: 950-959

(3) Auroy Y, Benhamoud D. Major complications of regional anaesthesia in France: the SOS regional anaesthesia hotline service.
Anaesthesiology 2002; 97: 1274-1280

(4) Cook TM, Counsell D. Major complications of central neuraxial block: Report on third national project of Royal College of Anaesthetists.
BJA 2009; 102: 179- 190

(5) Auroy Y, Narchi P. Serious complications related to regional anaesthesia and results of a prospective survey in France.
Anaesthesiology 1997; 87: 479-486

(6) Lee LA, Postner KL, Domino KB. Injuries associated regional anaesthesia in the 1980's and 1990's: a Closed Claims Analysis.
Anaesthesiology 2004; 101: 143-152

(7) Brull R, McCartney CJL. Neurological complications after regional anaesthesia: Contemporay estimates of risk.
Anaesthesia and Analgesia 2007; 104: 965-974

(8) Neal JM, Bernards CM. ASRA practice advisory on neurologic complications in regional anaesthesia and pain me.
Regional Anaesthesia and Pain Medicine; 33(5): 404-415

(9) Neal JM. Anatomy and pathophysiolgy of spinal cord injury associated with regional anaesthesia and pain medicine.
Regional Anaesthesia and Pain Medicine; 33(5): 423-434

(10) Guay J. First do no harm: balancing the risks and benefits of regional anaesthesia in patients with underlying neurological disease.
Canadian Journal of Anesthesia 2008; 55(8): 489-494

(11) Macfarlane AJ, Brull R. Regional anaesthesia and pre-existing neurological disease – I (editorial).
Canadian Journal of Anesthesia 2008; 55(12): 875-876

(12) Nelson T. Regional anaesthesia and pre-existing neurological disease – II (editorial).
Canadian Journal of Anesthesia 2008; 55(12): 876-878

(13) Fischer B. How to avoid neurological damage –ESRA recommendations for good clinical practice
SAJRA Vol 17-24

(14) Horlocker TT. Peripheral nerve injury following regional anaesthesia: diagnosis, prognosis and prevention.

(15) Upton AR,McComas AJ. The double crush in nerve entrapment syndromes.
Lancet 1973; 2: 359-362

(16) Osternam AL. The double crush syndrome.
Orthopaedic Clinics of North America 1988; 19: 147-155

(17) Hebl JR, Horlocker TT. Neuraxial anesthesia and analgesia in patients with pre-existing central nervous system disorders.
Anaesthesia and Analgesia 2006; 103:223 – 22

(18) Hebl JR, Kopp SL. Neurologic complications after neuraxial anesthesia and analgesia in patients with pre-existing sensorimotor neuropathy or diabetic polyneuropathy
Anaesthesia and Analgesia 2006; 103: 1294-1299

(19) Aldrete JA. Exacerbation of pre-existing neurological deficits by neuraxial anaesthesia: report of 7 cases.
Journal of Clinical Anaesthesia 2005; 17: 304-313

(20) Koff MD. Severe brachial plexopathy after an ultrasound-guided single-injection nerve block for total shoulder arthroplasty in a patient with multiple sclerosis.
Anaesthesiology 2008; 108: 32-328

(21) Vercauteren M, Heytens L. Anaesthetic considerations for patients with a pre-existing neurological deficit: are regional techniques safe?
Acta Anaesthesiology Scandanavica 2007;51 : 831-838

(22) Hogan Q. Pathophysiology of peripheral nerve injury during regional anaesthesia.
Regional Anaesthesia and Pain medicine; 33(5): 435-441

(23) Rubino F. Perioperative management of patients with neurologic disease.
Neurology Clinics 2004; 22: 261-276

(24) Greensmith J, Bosseau Murray W. Complications of regional anaesthesia.
Current Opinion in Anaesthesiology 2005; 19: 531-537