

PERI OPERATIVE NEUROPATHIES

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Introduction

Perioperative nerve injury (PNI) is not new.

- ❑ Lately it has caught the attention of the lay-press, Lawyers, the Plaintiff, and ofcourse the principal character- the doctor.
- ❑ Third Leading cause of anaesthesia related claims according to American Society of Anaesthesia (ASA) claims registry data.
- ❑ There is the need for an in-depth knowledge of the causes and prevention of PNI and management protocol
- ❑ Legal defence in case the inadvertent happens.
- ❑ It is almost impossible to absolve the care giver of negligence/mismanagement if a patient comes into a health facility without nerve lesion, but emerges with one.
- ❑ Nigeria is now a medico-legally evolving state
 - Due to Increased knowledge
 - And also the return of some of our foreign based colleagues

AIM OF PRESENTATION

1. To remind us of what we already know
2. To enable us give best practice to our patients
3. In the unfortunate scenario of litigation, to enable us prepare our defence to some reasonable extent.

HISTORICAL BACKGROUND

- ❑ 1894 Budinger and 1897 Garriques reported neuropathies occasioned by improper anaesthetics and patients malpositioning.
- ❑ 1973 Wadsworth and Williams in a retrospective study of just 12 patients – ulnar nerve injury only 2 of these were due to external nerve compression.
- ❑ 2000 – Warner et al reported in a prospective study at Mayo Clinic found that both medical and surgical patients develop neuropathies during In and Out patient care.
- ❑ Also Warner et al, retrospective study found that ulnar neuropathy is;
 - 70-90% common in male patients.
 - BMI $\geq 38\text{kg/m}^2$
 - Common in patients with prolonged bed rest.
- ❑ In 1987 – Alvine and Schurrer found that many patients with peri operative neuropathies have a high frequency of contr-alateral ulnar nerve dysfunction.

INCIDENCE

Exact incidence is difficult, this is because of the quality and heterogeneity of available studies.

- ❖ One study gave a figure of 0.14% when all patients are considered.
- ❖ For ulnar another study quoted 0.037%
- ❖ Another study which looked into anaesthesia related PNI from 380,680 patients is 112 which is 0.03%

Incidence by specialty

Khan & Birch (1991-1998); 612 PNI cases review. 291 of these were explored, amazingly about 144 were subject of litigation.

Incidence by specialty:

Specialty	Incidence	
Orthopaedics	174/291	59.8%
General Surgery	70/291	24.1%
Vascular Surgery	11/291	3.78%
Plastic Surgery	7/291	2.41%
Cardio-thoracic	5/291	1.72%
ENT	5/291	1.72%
Anaesthesia	4/291	1.37%
Neurosurgery	4/291	1.37%
Maxillofacial	2/291	0.69%

AETIOLOGY AND RISK FACTORS

-Direct nerve damage

- Surgical

Ligation, Laceration

Anaesthetics Technique

- Needle trauma 2^o regional technique
- Peripheral nerve block and catheterization

Stretch and Compression

- Poor padding
- Abnormal limb positioning
- Use of tourniquet and surgical retractors

Bruner's ten rules for safe use of tourniquet

Rule	Description
Size of tourniquet	Arm 10cm Leg 15cm or wider especially in the obese
Site of Application	Upper arm Mid thigh
Padding	At least 2 layers of orthopaedic wool
Skin prep	Cover pad up to prevent soaking
Pressure	50-100 mmHg above patient's systolic pressure or 200-250 mmHg in the arm 250-350 in the leg. We know that pneumatic tourniquet is the best.
Time	Absolute maximum 3hrs. Generally do not exceed 2hrs.
Temperature	Avoid heating e.g Hot light cool if feasible and keep tissues moist
Documentation	Duration time-in and time-out mandatory
Calibration and maintenance	Do it regularly at least weekly against mercury manometer and teeth gauge
Maintenance	3 monthly to keep equipment optimal

AETIOLOGY AND RISK FACTORS

Cont'd

■ Ischaemia

Common pathway of nerve injury

-Prolonged use of tourniquet

-prolonged immobility

-Oedema

LA drugs especially those with epinephrine

AETIOLOGY Cont'd

- Toxicity of LA - This is not a problem when used in clinical concentration and injected extravascular, but injection in high concentration intraneurally causes nerve damage.
- Preservatives, some are neurotoxic
- Use of LMA (Laryngeal mask)
 - Associated with lingual nerve damage
- Patient factor
 - Hypertension
 - DM and smoking
 - Anatomical abnormalities
 - Thoracic outlet obstruction
 - Severe contracture deformity at the elbow

PERI OPERATIVE FACTORS

- ☐ Hypovolaemia
- ☐ Dehydration which causes perfusion deficit
- ☐ Hypotension
- ☐ Hypoxia
- ☐ Electrolyte imbalance
- ☐ Hypothermia (Peripheral vasoconstriction)

CLASSIFICATION OF PNI

Sedon's classification

Sunderland's classification

Sedon's is most widely accepted. It is simple

- ✓ Neuropraxia
- ✓ Axonotmesis
- ✓ Neurotmesis

Sunderland described a more complex stages 1-5 nerve injury

Table 1

Degree of Injury	Cause	Pathology	Symptoms	Action Potential	Recovery
Neuropraxia	Compression or Ischaemia	There is no wallerian degeneration. Ischaemia of myelin sheath distal to the point of injury. Nerve is intact	Sensory loss in areas supplied by the nerve. Distal to injury site	Proximal to injury for motor	Re-myelination of distal segments. Normal AP for motor expected. Distal to the site of injury by day 10. FR 2-12 weeks
Axonotmesis	A more severe compression crush. Disruption of Axon. Myelin sheath support structures are intact. Nerve sheath/schwann cells	Wallerian degeneration occurs both ways proximal and distal to injury site to the nearest node of ranvier nerve cells - loss of conduction distal segment 3-4 days	Sensory and motor loss in both directions	Partial or none for sensory or motor due to Axon loss EMG –Fibrillation potential +sharp waves 2-3 weeks after injury.	Axonal regeneration at 1-2 mm per day. full recovery 2-6 months. This depends on age and the amount of scarring
Neurotmesis	Complete disruption of peripheral nerve by any means. Most often surgical - Laceration -Diathermy	Wallerian degeneration. Distal to lesion. NC loss of conduction. Distal segment 3-4 days	Sensory loss, motor loss. Distal to the injury site. Pain.	2-3 weeks after injury EMG fibrillation potential and sharp waves 2-3 weeks	Surgical intervention is required to repair the nerve 2-18 months onward

CLINICAL PRESENTATION

➤-Parasthesia

➤-Hypo and Hypersthesia

➤-Pain

In the area supplied by the affected nerve.

Motor deficit .

■Parexis

■Trophic changes

- Muscle loss

INDIVIDUAL NERVE NEUROPATHIES

- ✓ Ulnar
- ✓ Brachial plexus

Ulnar nerve

- commonest causes:
 - External compression of ulnar nerve
 - Elbow flexion when it is over 100 degrees and for a long time.
 - The cubital tunnel retinaculum is made taut compressing the nerve against the olecranium
 - Fore arm rotation especially pronation

INDIVIDUAL NERVE NEUROPATHIES cont'd

- ✓ Compression within the bony groove posterior to medical epicondyle is possible.
- ✓ Direct external compression at distal to the medical epicondyle where the nerve and the artery are superficial in the post condylar groove.

Figure 1

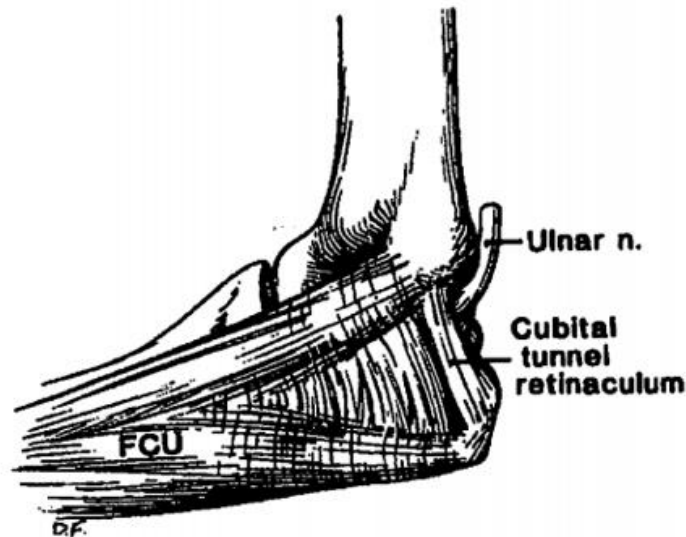


FIG. 1. The proximal edge of the roof of the cubital tunnel is formed by a retinaculum that originates on the medial epicondyle and inserts on the olecranon. It is distinct from the aponeurosis of the flexor carpi ulnaris (FCU) with which its distal margin blends. Reproduced with permission and copyright © of the British Editorial Society of Bone and Joint Surgery, from O'Driscoll SW, Horii E, Carmichael SW, Morrey BF: The cubital tunnel and ulnar neuropathy. *J Bone Joint Surg [Br]* 1991; 73:613-7.

Figure 2

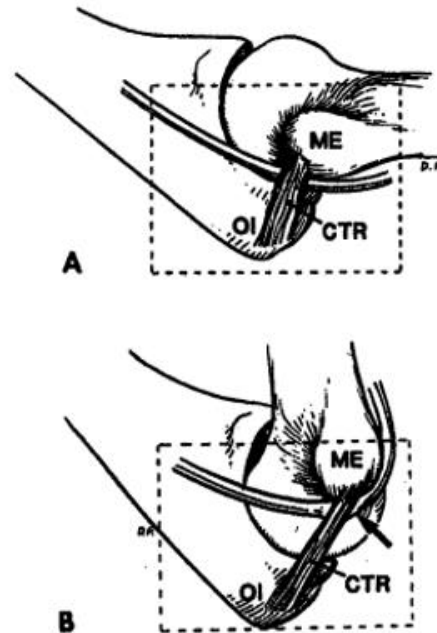


FIG. 2. In this medial-to-lateral view of the right elbow, the cubital tunnel retinaculum (CTR) is lax in extension (A) as it stretches from the medial epicondyle (ME) to the olecranon (OI). The retinaculum tightens in flexion (B) and can compress the ulnar nerve (arrow). Reproduced with permission and copyright © of the British Editorial Society of Bone and Joint Surgery, from O'Driscoll SW, Horii E, Carmichael SW, Morrey BF: The cubital tunnel and ulnar neuropathy. *J Bone Joint Surg [Br]* 1991; 73:613-7.

BRACHIAL PLEXUS

causes:

- ✓ Median sternotomy
- ✓ Direct trauma by fractured 1st rib.
- ✓ Stretch of the nerve in prone position and head turned to contralateral side. Ipsilateral shoulder abducted and elbow bent on the same side

Figure 3

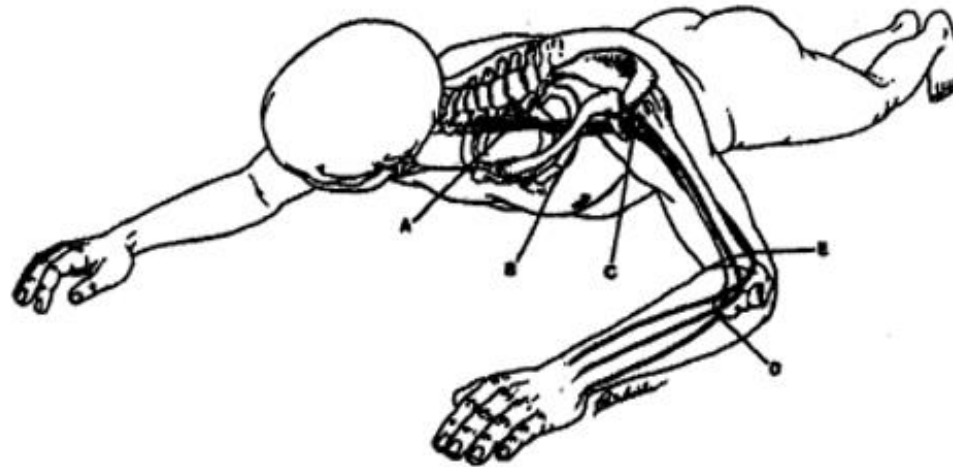


FIG. 6. Sources of potential injury to the brachial plexus and its peripheral components in a prone patient. Head position stretching plexus against anchors in shoulder (A). Closure of the retroclavicular space by chest support with the arms at the side; neurovascular bundle trapped against the first rib (B). Head of the humerus thrust into the neurovascular bundle if the arm and axilla are not relaxed (C). Compression of the ulnar nerve in the cubital tunnel (D). Area of vulnerability of the radial nerve to compression above the elbow (E).

LOWER EXTREMITIES

The following nerves can be affected.

- Common Peroneal 81%
- Sciatic 15%
- Lateral femoral cutaneous nerve 4%

Mode of Injury

Nerve injury in the lower extremities can occur in so many patients postures the commonest is Lithotomy position.

It is presumed to be preventable post operative problem that result mainly from

(1) Prolong Lithotomy (2) Inadequate padding and Malpositioning.

Hyperflexion of the hip followed by extension at the knee will cause a strain on the sciatic nerve.

Abduction of the thigh > 30 degrees without concomitant flexion at the hip puts strain on the obturator nerve.

- Any positioning that put the knee flexors the hamstrings muscles on tension put a strain on sciatic nerve.

It is important to observe that the hamstrings is not taut in Lithotomy.

COMMON PERONEAL NEUROPATHY

- ✓ Direct compression of nerve against the head of fibula as it is most superficial here in leg support in lithotomy
- ✓ Injury can be due to pressure from firm leg stockings

CRITERIA FOR DIAGNOSIS

1. Early diagnosis is very important.

-For anaesthetic causes especially positional. Take a good history in the immediate post op

- Any numbness
- Parasthesia
- Pain

It must be new development within 48hours of either sedated or anaesthetized.

2. Loss of sensory impulse along the distribution of a particular nerve or loss of motor.

3. Reduced reflex

4. Surgical

When an incision is made along a nerve and patient complain of loss of function in a particular limb, then laceration of the nerve must have occurred.

MANAGEMENT IN THE EVENT OF NEUROPATHY

1. Determine the type of neuropathy;

sensory or motor.

Sensory damage is transient and symptoms are numbness and tingling.

-symptoms most likely to disappear in the 1st 5days post op.

Plan – Reassure, plan to see pt frequently in the period.

2. If the neuropathy has a motor component. A neurologist consult is mandatory. Who will determine the location of injury by EMG sometimes 3 Teslar imaging.

The study may also demonstrate chronic abnormality of the nerve, or if applicable the contralateral nerve.

ELECTRODIAGNOSIS

Base line 0-7 days.

- ❖ Determine nerve injury
- ❖ For comparison with later studies.
- ❖ If fibrillation and sharp waves are present in the first week then there must have been a pre-existing nerve injury.

Initial study 10-21 days

- ❖ Differentiates between just demyelination from substantial axonal loss.

Follow up 3-6 months

- ❖ Shows extent of reinervation
- ❖ Axonal regeneration can be demonstrated.

INDICATION FOR NON OPERATIVE INTERVENTION

- ❖ Delay in presentation
- ❖ Clinical improvement on presentation
- ❖ Patient prefers palliative treatment
- ❖ Pain relief possible by non-op means

MISMANAGEMENT

- ❖ Inadequate informed consent.
- ❖ Avoidable damage to
- ❖ Delay in diagnosis/ miss-diagnosis
- ❖ Delay referral/ treatment
- ❖ Inappropriate treatment

Informed Consent

Risk of damage to a particular nerve by a procedure should be discussed in detail if the risk is more than a remote one. E.g;

- excision of lump from the posterior triangle of neck.
- Fracture fixation/ removal of metal work
- Spinal decompression
- Excision of lump close to a nerve. E.g parotid

MANAGEMENT

Neurotmesis

- Clear, accurate documentation of the injury and action taken with the help of neurologist for adequate assessment.
- Prompt referral to nerve surgeon
- Discuss with patient with full truthful explanation

Indication for Surgery

- ✓ Clinical evidence of Neurotmesis
- ✓ Failure to recover within the presumed time of axonotmesis.
- ✓ Deterioration of lesion while under observation
- ✓ Persistent intractable pain

Aim of Surgery

- ✓ Establish diagnosis
- ✓ Relief pain
- ✓ Reverse disease progress by improving function

POST OPERATIVE BLINDNESS

- Reported especially in patients in prone position for multi-level spinal decompression surgery, some cardiac procedure that went on for a long time also record some cases of post procedural visual impairment.
- Pathology- it is usually due to either ischaemia of the posterior or anterior branch of the optic nerve.

Blindness due to post spinal decompression surgery usually is caused by posterior ischaemic optic neuropathy (PION) while blindness after a prolonged cardiac procedure is due to both anterior and posterior ischaemic optic neuropathy.

It is postulated that this injuries can be due to:

1. Increased intraocular pressure, anemia and hypertension
2. Engorged veins around the optic nerve leading to compartment compression and decreased arterial perfusion, this is unsubstantiated

RISK FACTORS FOR POST OP BLINDNESS

Advanced age

Arteriosclerosis— Patients with peripheral vascular disease and Diabetics

Procedural issues

- Prolonged pump perfusion
- Deliberate post op anaemia
- Deliberate intraoperative hypotension

All these produce inadequate perfusion of the optic nerve.

Evaluation of ASA claims registry recently deduced that most cases of post op blindness after spinal surgery occur —operation lasted for 6hrs or more, pt. had severe haemorrhage, and pt is in prone position.

PREVENTIVE CONSIDERATION FOR POST OP BLINDNESS

- Head position at level or higher than heart position when possible in high risk patient
- head in neutral without significant neck flexion, extension or rotation
- staged spine procedure for patient who require multi-level decompression surgery.

A case of Ulnar Neuropathy I'm involved in

- ✓ A foreign based gynecologist who recently relocated to Nigeria.
- ✓ Refused to heed my advice of him not hyperextending the right arm at the shoulder while performing laparoscopy surgery.
- ✓ The procedure took a while. Post op, the patient, a banker developed paresis in the finger and can't have her signature done rightly.
- ✓ This surgeon could not discuss the case with me because he is culpable. The patient had to go as far as USA to seek redress.
- ✓ Patient got better.
- ✓ There was no case instituted.

A CASE OF ULNAR NEUROPATHY.

Cont'd

✓ Lesson here

Theatre session is a team effort.

The surgeon, is the head of the team, being the principal actor.

All participants should be respected and their views objectively considered.

✓ Why

Because every team member work in the interest of the patient.

Conclusion

The incidence of perioperative nerve injury though low is significant because it has to do with a situation of “this thing speaks for itself”. An individual with normal nerve function enters the hospital and came out with some sensory loss and motor dysfunction. To avoid being caught in a legal issue, it is important for all medical personnel to be conscious of the probability of a perioperative neuropathy and do all that is necessary to prevent it.

- Detailed documentation of precaution taken.
- Prompt diagnosis and treatment

When this is done, there will be a window of escape.

THANK YOU



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