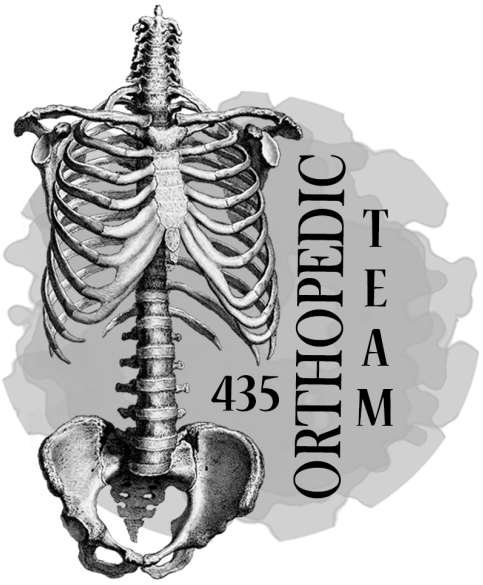

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peripheral nerve injury

Objectives:

Not given

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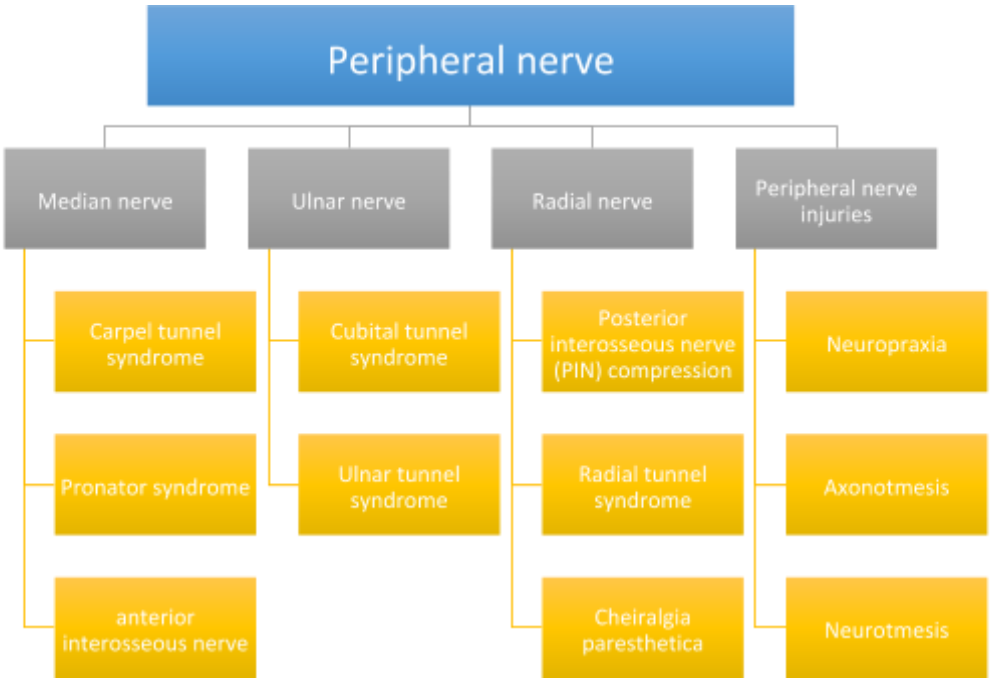
Sources: Apley's+Slides+Notes+433Team.

Mind map



Nerve injury: It is a condition, in which the conductivity of the nerve is affected, can be partial or complete.

Compression Neuropathy

Nerve compression impairs epineural blood flow and axonal conduction, giving rise to symptoms such as numbness, paraesthesia and muscle weakness; the relief of ischaemia explains the sudden improvement in symptoms after decompressive surgery. Prolonged or severe compression leads to segmental demyelination, target muscle atrophy and nerve fibrosis; symptoms are then less likely to resolve after decompression. Apley.

- It is a condition with sensory, motor, or mixed involvement.
- if mixed pathology, sensory function is affected first and then motor is affected "this is because Motor fibers have thick myelin sheath".
- As a result, first symptom to appear is hypoesthesia and lastly atrophy of the muscles which means severe disease.
- The sensory functions lost are as follows "in order" :
    - First lost ⟶ light touch – pressure – vibration (mild)
    - Last lost ⟶ pain sensation loss – temperature (severe)
- The pathophysiology of compression neuropathy:

  Microvascular compression due to any cause ⟶ neural ischemia ⟶ paresthesia ⟶ Intraneural edema ⟶ more microvascular compression ⟶ demyelination ⟶ fibrosis ⟶ axonal loss.

Common Systemic Conditions Leading To Compression Neuropathy	
Anatomic	**Inflammatory**
Synovial Fibrosis Lumbrical Encroachment Anomalous Tendon Median Artery Fracture Deformity	Rheumatoid Arthritis Infection Gout Tenosynovitis
Mass	**Systemic**
Ganglion Lipoma Hematoma	diabetes Alcoholism Renal Failure Raynaud
Fluid Imbalance	
Pregnancy Obesity	

symptoms

“Rule out systemic causes” ALARMING

- Night symptoms “Sign of advanced disease and indication to surgery”
- Dropping of objects
- Clumsiness
- Weakness

Physical examination

- Examine individual muscle power ⟶ grades 0 to 5 ⟶ pinch strength - grip strength.
- Neurosensory testing:
  - Dermatomal distribution
  - Peripheral nerve distribution

Special tests

Semmes-Weinstein monofilaments	Two-point discrimination
- **The best test** - can detect very early neuropathy - Cutaneous pressure threshold ⟶ function of large nerve fibers which is first to be affected in compression neuropathy. Sensing 2.83 monofilament is normal.	- **Static** function is lost first and then dynamic. - Performed with closed eyes - Inability to perceive a difference between points > 6 mm is considered **ABNORMAL** “Late finding”.

Electrodiagnostic testing:

- Sensory and motor nerve function can be tested through EMG and NCS
- operator dependent (disadvantage)
- Objective evidence of neuropathic condition
- Helpful in localizing point of compromise
- In the early disease, there is a High false-negative rate.

NCSs (nerve conduction studies)	**EMG** (Electromyography)
- Measure conduction velocity and distal latency and amplitude (nerve takes time to respond) - Demyelination ⟶ ↓ conduction velocity + ↑ distal latency - Axonal loss ⟶ ↓ potential amplitude	- Test muscle electrical activity - In case of muscle denervation ⟶ fibrillations, Positive sharp waves, fasciculations

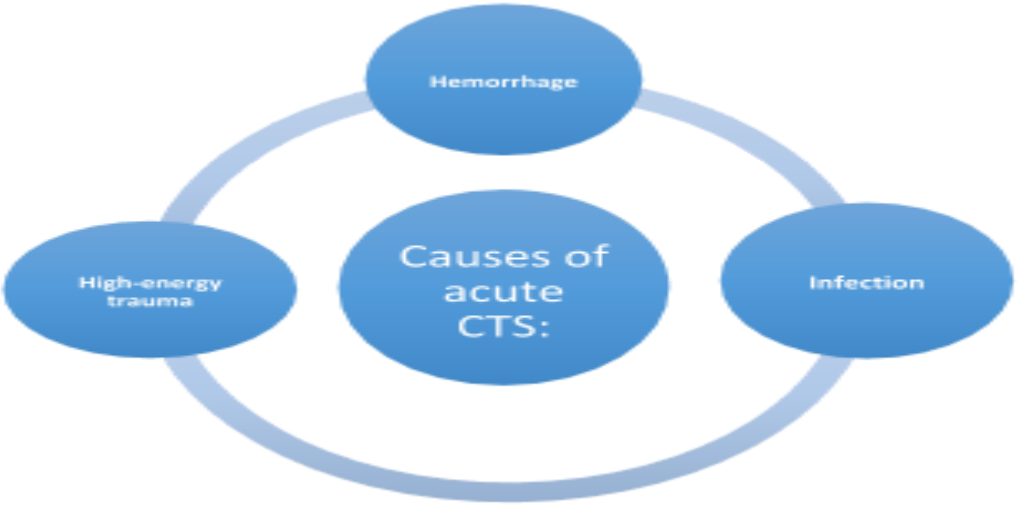
MEDIAN NERVE COMPRESSION

Carpal tunnel syndrome

This is the best known of all the entrapment syndromes. In the normal carpal tunnel there is barely room for all the tendons and the median nerve; consequently, any swelling is likely to result in compression and ischaemia of the nerve. Usually the cause eludes detection; the syndrome is, however, common at the menopause, in rheumatoid arthritis, pregnancy and myxoedema.Apley.

- **Most common compressive neuropathy in the upper extremity.**
- **Anatomy of the carpal tunnel:**
    1. Volar aspect →TCL (transverse carpal ligament)
    2. Radial aspect →scaphoid tubercle + trapezium
    3. Ulnar aspect → pisiform + hook of hamate
    4. Dorsal aspect → proximal carpal row + deep extrinsic volar carpal ligaments
- **Content of the carpal tunnel:**
    - Median nerve + FPL (flexor pollicis longus) + 4 FDS (flexor digitorum superficialis) + 4 FDP (flexor digitorum profundus) = 10
    - Normal pressure in the tunnel → 2.5 mm Hg "not measured or used clinically".
    - >20 mm Hg → ↓ ↓ epineural blood flow + nerve edema
    - >30 mm Hg → ↓ ↓ nerve conduction
- **Forms of carpal tunnel syndrome:**
    - Idiopathic → most common in **adults**
    - Mucopolysaccharidosis → most common cause in **children**
    - Anatomic variation: (rare)
        - Persistent median artery
        - Small carpal canal
        - Anomalous muscles
        - Extrinsic mass effect
- **Risk factors:**
    - Obesity
    - Pregnancy
    - Diabetes
    - Thyroid disease
    - Chronic renal failure
    - Inflammatory arthropathy
    - Vitamin deficiency
    - Storage diseases
    - Alcoholism
    - Advanced age
    - Vibratory exposure (using drills and electrical saws) during occupational activity

Acute CTS requires emergency decompression



Diagnosis of:

- **Symptoms**:
    - Paresthesia and pain, often at night involving the volar aspect of thumb, index, middle fingers + radial half of ring finger.
    - **REMEMBER: Sensory function loss in order:**

        Affected first ⟶ light touch + vibration
        Affected later ⟶ pain and temperature

    - Symptoms of late findings :
        - Weakness - loss of fine motor control - abnormal two-point discrimination
        - Thenar atrophy ⟶ severe denervation

- **Provocative test** ⟶ carpal tunnel compression test
    - Durkan test ⟶ The Most sensitive test.
    - Other provocative tests include Tinel and Phalen

- **Semmes-Weinstein monofilament testing** :
  ⟶ Early CTS diagnosis
- **Electrodiagnostic Testing:**
    - Not necessary for the diagnosis of CTS (just to reassure the patient)
    - Distal sensory latencies > 3.5 msec
    - Motor latencies > 4.5 msec
    - NCS: ↓ conduction velocity and ↓ peak amplitude ⟶ less specific
    - EMG: ↑ insertional activity - sharp waves -fibrillation – APB (abductor pollicis brevis) fasciculation.

Differential diagnosis of CTS:

- Cervical radiculopathy (C5/C6)
- Brachial plexopathy
- TOS (Thoracic Outlet Syndrome)
- Pronator syndrome (most likely)
- Ulnar neuropathy with Martin- Gruber anastomosis
- Peripheral neuropathy of multiple etiologies

Double-crush Phenomenon
Blockage of axonal transport one point makes the entire axon more susceptible to compression elsewhere along the nerve

Treatment of CTS	
Non-operative	**Operative**

• Activity modification
• Night splints(less pressure)
• NSAIDS
• Single corticosteroid injections (transient relief):
• 80% after 6 weeks
• 20% after one year
• ineffective corticosteroid injection is an indication of poor prognosis therefore,less successful surgery rate.
	can be in different approaches:
	• open - mini open - endoscopice
	• internal median neurolysis or flexor tenosynovectomy shows benefit
	  ○ Too ulnar surgical approach > ulnar neurovascular injury
	  ○ Too radial surgical approach > recurrent motor branch of median nerve injury
	• recurrent motor branch variations
	  ○ extra-ligamentous →50%
	  ○ subligamentous →30%
	  ○ transligamentous → 20%
	• Endoscopic release : (more expensive)
	➢ **short term (within 4 to 6 weeks):**
	• less early scar tenderness
	• Improves short-term grip/pinch strength
	  ○ Better patient satisfaction scores
	➢ **Long-term:**
	  ○ No significant difference (after one year)
	  ○ May have slightly higher complication rate
	  ○ Incomplete TCL (transverse carpal ligaments release)

- E.g. In thoracic outlet syndrome the lower trunks of the brachial plexus are compressed which makes the ulnar nerve vulnerable to compression.

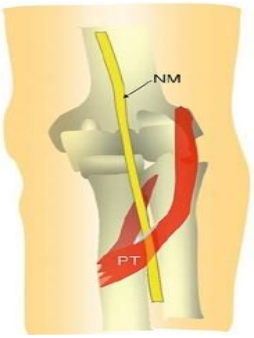
- E.g. If a patient with thoracic outlet syndrome comes with ulnar nerve symptoms and you treat the ulnar nerve, **the patient will still have the symptoms or be slightly better because you did not treat the cause (thoracic outlet).**

Release outcome :

- Pinch strength → will come back in 6 weeks
- Grip strength →will come back in 3 months
- Persistent symptoms after release indicate one of the following :
  - Incomplete release
  - Iatrogenic median nerve injury
  - Missed double-crush phenomenon
  - Concomitant peripheral neuropathy
  - Space-occupying lesion
- Revision of success →identify underlying failure cause

Pronator Syndrome

Median nerve compression at arm and forearm (entrapment of the median nerve between 2 heads of the pronator muscle).



Symptoms:

- Proximal volar forearm pain
- Sensory symptoms → palmar cutaneous branch

Anterior interosseous syndrome

motor weakness without sensory symptoms.

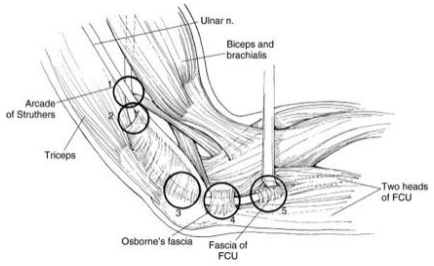
The anterior interosseous nerve can be selectively compressed at the same sites as the proximal median nerve. However, spontaneous (and usually temporary) physiological failure (Parsonage–Turner syndrome) is a more likely cause. Apley.

Ulnar nerve compression

Cubital Tunnel Syndrome

The ulnar nerve is easily felt behind the medial epicondyle of the humerus (the 'funny bone'). It can be trapped or compressed within the cubital tunnel (by bone abnormalities, ganglia or hypertrophied synovium), proximal to the cubital tunnel (by the fascial arcade of Struthers) or distal to the cubital tunnel as it passes through the two heads of flexor carpi ulnaris to enter the forearm (Osbourne's canal). Sometimes it is 'stretched' by a cubitus valgus deformity or simply by holding the elbow flexed for long periods.Apley.

- Second most common compression neuropathy of the upper extremity
- Cubital tunnel borders:
  - floor →MCL and capsule
  - Walls → medial epicondyle and olecranon
  - Roof → FCU fascia and arcuate ligament of Osborne

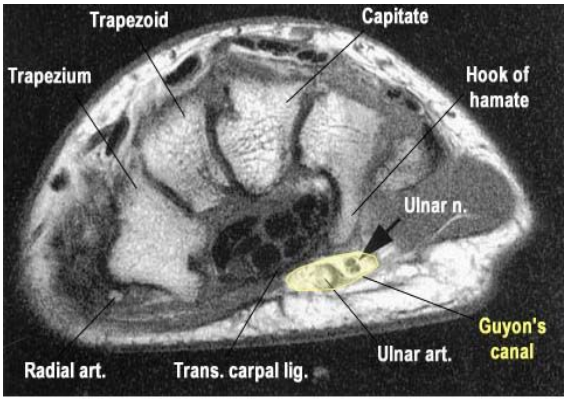


- **Clinical presentation:**
  - Paresthesia of ulnar half of ring finger and small finger
  - **Provocative tests:** (if the numbness developed, it means +ve test)
    - Direct cubital tunnel compression
    - Tinel sign
    - Elbow hyperflexion (increase the tension for the nerve. So, in mild symptoms casting with extended elbow will release the nerve)
  - **Froment sign**: flexion of the thumb and Interpharngeal joint when a sheet/credit card is held between the fingers (weak adductor pollicis)

- **Treatment:**
  - **Electrodiagnostic tests** → diagnosis and prognosis
  - **Nonoperative treatment：**
    - activity modification (avoid putting their hands on hard object and prevent hyperflexion of the elbow)
    - night splints → slight extension
    - NSAIDs
  - **Surgical Release:** Numerous techniques
    - In situ decompression, Anterior transposition, Subcutaneous, Submuscular, Intramuscular, Medial epicondylectomy
    - No significant difference in outcome between simple decompression and transposition.

Ulnar Tunnel Syndrome

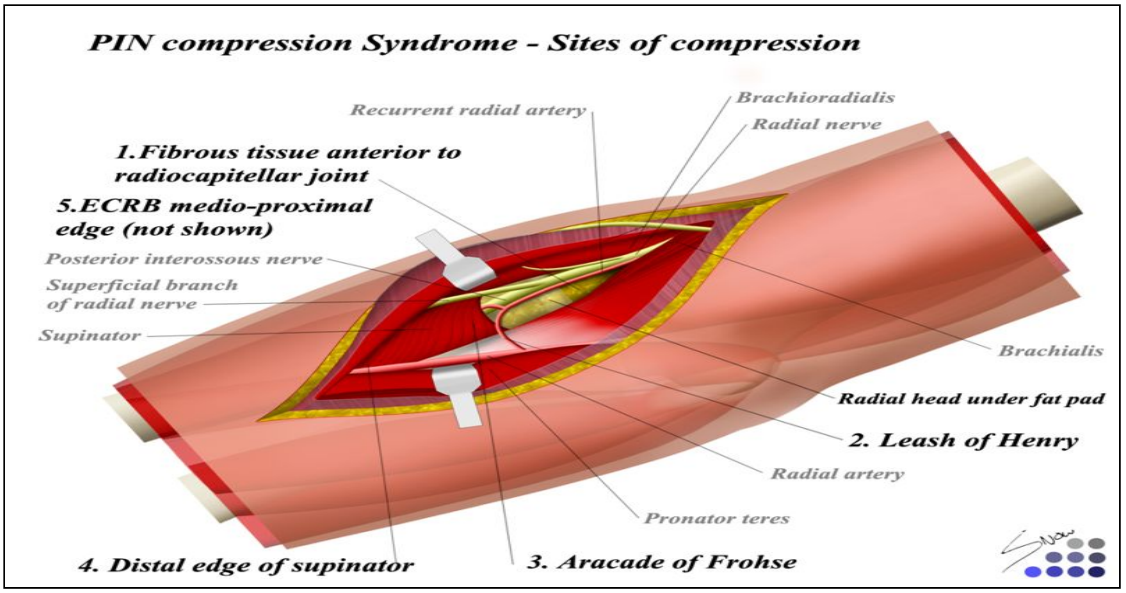
- Compression neuropathy of ulnar nerve in the Guyon canal
- **Causes:**
  - Ganglion cyst (80%)
  - Hook-of-hamate nonunion
  - Ulnar artery thrombosis
- **Investigations:** (you have to do it all to exclude)
  - **CT:** for hook-of hamate non-union
  - **MRI:** in case of Ganglion cyst (the best test)
  - **Doppler:** for ulnar artery thrombosis
  - **Nerve conduction study**
- **Treatment** (you have to identify cause)
  - **Non-operative treatment**
    - activity modification splints NSAIDs
  - **Operative treatment** → decompressing by removing underlying cause.



Radial nerve compression

Rarely compressed and mainly presented with motor symptoms and very vague symptoms.

- **Posterior interosseous nerve (PIN) compression**
  - lateral elbow pain
  - muscle weakness
- **Radial Tunnel Syndrome**
  - lateral elbow and radial forearm pain
  - no motor or sensory dysfunction
- **Cheiralgia paresthetica:** compression or trauma to the superficial-sensory branch of the radial nerve.
  - pain
  - numbness
  - paresthesia over dorso-radial hand



Causes (important to know it):

- **Compression** (acute fracture of the distal radius is associated with carpel tunnel syndrome)
- **Stretch** (After surgery, for example humorous surgery and the patient present post-op with drop-wrist due to stretching of the nerve)
- **Blast** (Gunshot wound or explosion)
- **Crush** (Heavy object fell down on the patient and usually present with multiple injuries)
- **Avulsion** (Gunshot wound when the bullet perpetrates the body it pulls some tissue with it or with retraction in the OR)
- **Transection** (Knife stab or iatrogenic in the OR)
- **Tumor invasion**

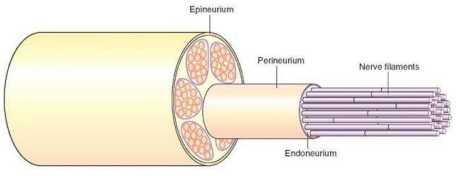
Prognostic factors for recovery	
Good factors	**Poor factors**
Young age (most important factor)	Crush or blast injuries
Stretch injuries (better than transection and crush etc.)	Infected or scarred wounds
Clean wounds	Delayed surgical repair (more than 2 weeks)
After direct surgical repair	----------

Classifications for peripheral nerve injury

1. **Neuropraxia**

   A reversible physiological nerve conduction block in which there is loss of some types of sensation and muscle power followed by spontaneous recovery after a few days or weeks. It is due to mechanical pressure causing segmental demyelination and is seen typically in 'crutch palsy', pressure paralysis in states of drunkenness ('Saturday night palsy') and the milder types of tourniquet palsy.Apley.

   - Mild nerve stretch or contusion
   - Focal conduction block
   - No wallerian degeneration (will be explained)
   - Disruption of myelin sheath
   - Epineurium, perineurium, endoneurium intact
   - Prognosis excellent with full recovery (best prognosis)



2. **Axonotmesis**

The term means, literally, axonal interruption. There is loss of conduction but the nerve is in continuity and the neural tubes are intact. Distal to the lesion, and for a few millimetres retrograde, axons disintegrate and are resorbed by phagocytes. This wallerian degeneration takes only a few days and is accompanied by marked proliferation of Schwann cells and fibroblasts lining the endoneurial tubes. The denervated target organs gradually atrophy, and if they are not reinnervated within 2 years they will never recover.
These axonal processes grow at a speed of 1–2 mm per day, Eventually they join to end-organs, which enlarge and start functioning again. Apley

- Associated with trauma and fractures
- Incomplete nerve injury
- Wallerian degeneration distal to injury (explained in the next page)
- Disruption of axons
- Sequential loss of axon, endoneurium, perineurium
- May develop neuroma-in-continuity
- Recovery unpredictable

3. **Neurotmesis**

As in axonotmesis, there is rapid wallerian degeneration, but here the endoneurial tubes are destroyed over a variable segment and scarring thwarts any hope of regenerating axons entering the distal segment and regaining their target organs. Instead, regenerating fibres mingle with proliferating Schwann cells and fibroblasts in a jumbled knot, or 'neuroma', at the site of injury. Even after surgical repair, many new axons fail to reach the distal segment. Apley

- Complete nerve injury (complete cut)
- Focal conduction block
- Wallerian degeneration distal to injury
- Disruption of all layers, including epineurium (painful)
- Proximal nerve end forms neuroma
- Distal end forms glioma
- Worst prognosis

Wallerian Degeneration

- Starts in distal nerve segment
- Degradation products is removed by phagocytosis
- Myelin-producing Schwann cells proliferate and align to form a tube to receive regenerating axons
- Nerve cell body enlarges and increased structural protein production
- proximal axon forms sprouts connect to the distal stump → migrate 1 mm/day

Surgical repair:

- Best performed within 2 weeks of injury
- Repair must be **free of tension** (any tension force will decrease blood supply)
- Repair must be within clean, well-vascularized wound bed
- Nerve length may be gained by neurolysis or transposition (neurolysis: release the proximal and distal tension of the nerve)
- **Repair techniques** (No technique deemed superior)
    - Epineurial (simple suture between the epineurium)
    - Individual fascicular
    - Group fascicular
- Nerve conduits → popular for digital nerve gaps >8 mm → polyglycolic acid and collagen based
- Larger gaps → grafting
- Autogenous → sural - medial/lateral antebrachial cutaneous - terminal/PIN
- Vascularized
- Growth factor augmentation → insulin-like and fibroblast → promote nerve regeneration
- Chronic peripheral nerve injuries → neurotization and/or tendon transfers
- Use of nerve transfers for high radial and ulnar nerve injuries gaining popularity