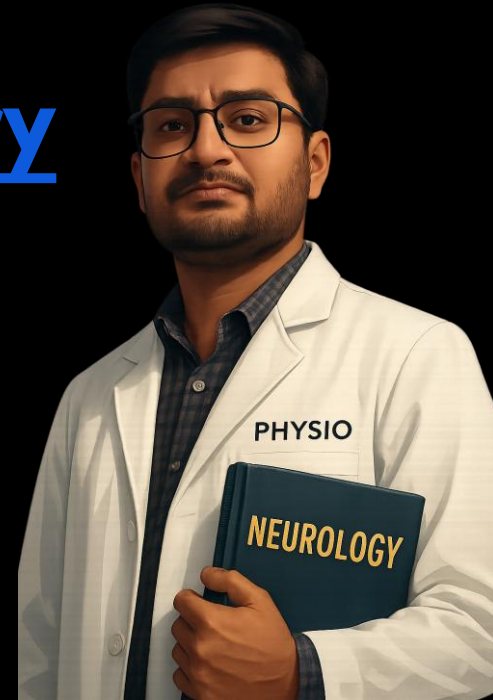




Peripheral Nerve Injury

Dr. Preet Mehta (PT)

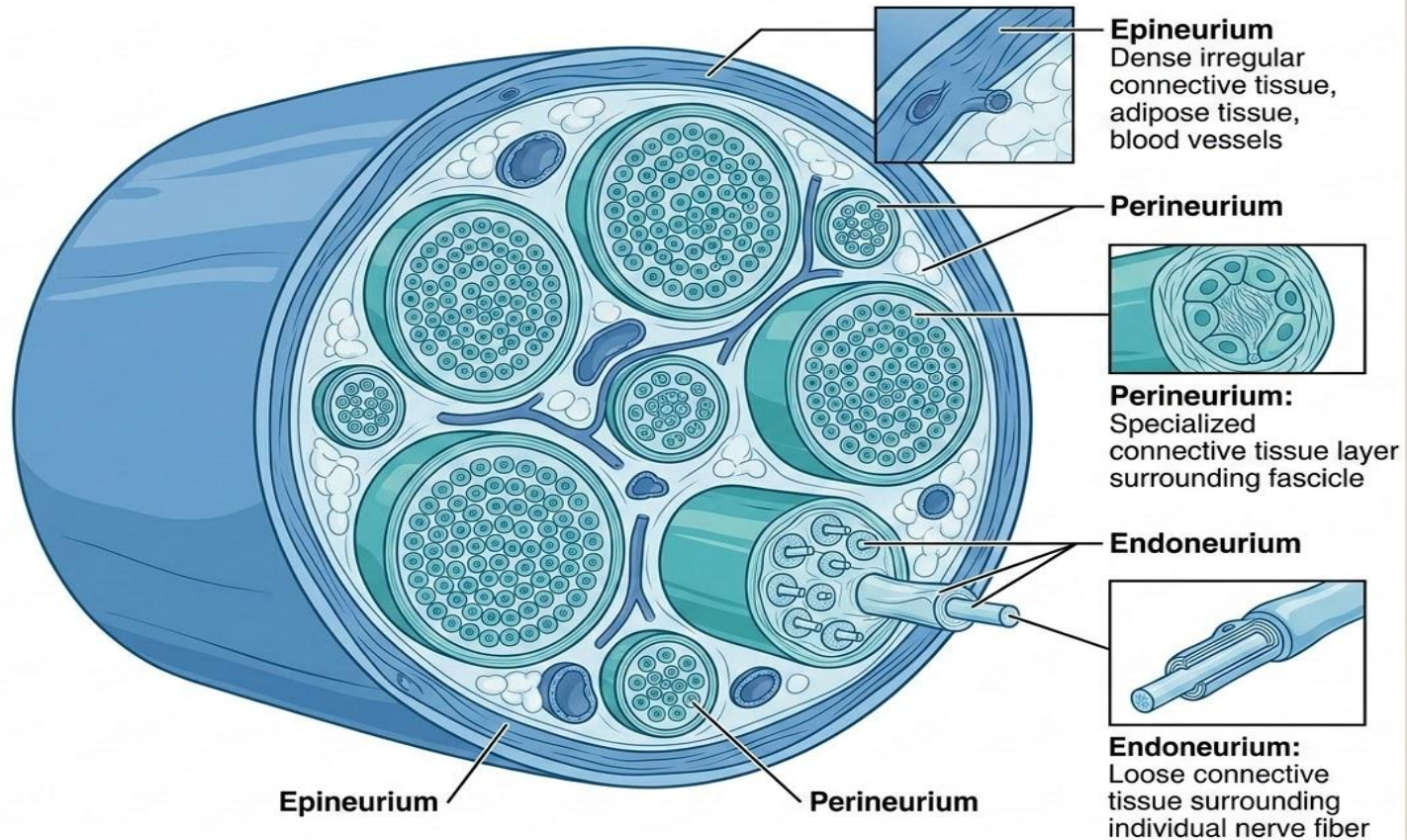


Introduction to the Peripheral Nervous System (PNS)

The peripheral nervous system consists of nerves **branching from the brain and spinal cord, forming the communication network with the body.**

Nerves contain bundles of fibers: **sensory nerves utilize afferent** fibers to provide skin sensation, while **motor nerves rely on efferent fibers** to supply muscles. **Mixed nerves** involve both functions.

Peripheral nerve injuries range from **minor damage to full transection**, presenting clinically with pain, numbness, muscle weakness, and loss of active movement, making prompt assessment critical.



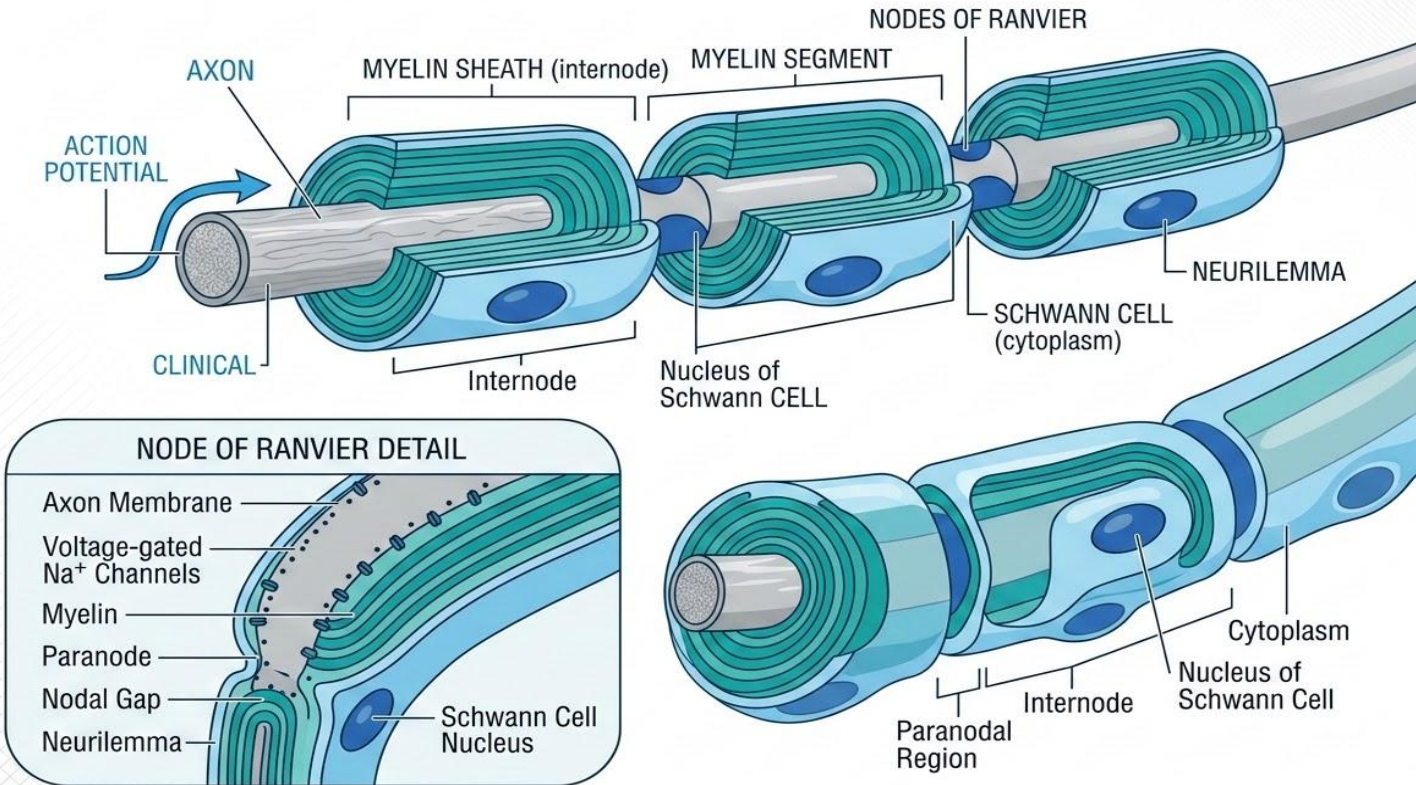
Protective Layers of Peripheral Nerves

Peripheral nerves are structured with three distinct protective connective tissue layers.

The EPINEURIUM is the outermost sheath surrounding the entire nerve trunk.

Inside, nerve fibers are grouped into **bundles called fasciculi** , which are individually encased by the **PERINEURIUM** . Within each fasciculus, every individual nerve fiber is wrapped in the **ENDONEURIUM** .

MYELINATED NERVE FIBER (PNS)



Nerve Fiber Structure & Injury Mechanisms

Cellular Physiology

A nerve contains bundles of nerve fibers functioning **as axons or dendrites**, surrounded by connective tissue.

Sensory nerves utilize long dendrites, while **motor nerves rely on long axons**. Individual nerve fibers are insulated by myelin.

Pathological Response to Injury

Mild Compression Injury

Focal demyelination occurs directly at the injury site without disrupting the underlying axon.

Severe Axonal Damage

Physical disruption occurs to both the axon cylinder and its insulating myelin sheath.

Pathophysiology of PNI

1. Traction & Stretch

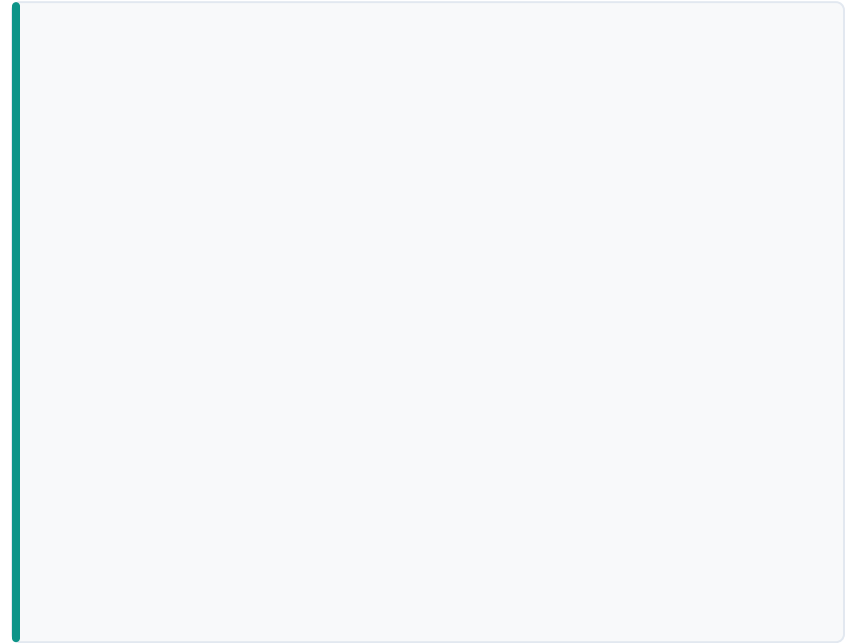
Primary mechanism where tensile forces stretch the nerve, **common in high-energy trauma or joint dislocations.**

2. Laceration

Physical transection of nerve architecture, often caused by **sharp objects, penetrating wounds, or surgical accidents.**

3. Compression & Ischemia

Direct pressure leading to microvascular obstruction.



Nerve Stretch Mechanics & Traction Trauma

Pathophysiology of Tensile Strain

Peripheral nerves possess natural length redundancy, allowing them to **stretch up to 10 percent during normal limb movements** with minimal consequence. However, exceeding these limits triggers severe internal and external structural lesions.

High-Risk Susceptibility & Etiology

Brachial Plexus Vulnerability

Nerves around the brachial plexus, particularly the **axillary nerve**, are **highly susceptible** to acute stretch-induced trauma.

10%
DISCOUNT

Nerve Laceration Pathophysiology

Mechanism of Injury

Laceration injuries involve a nerve being **partially or completely transected**, typically resulting from penetrating trauma such as knife wounds, metal shards, or flying debris.

This mechanism completely disrupts axonal continuity. Because the nerve is severed, spontaneous regeneration is anatomically impossible without targeted clinical intervention.

Consequently, these open, high-severity injuries absolutely require specialized surgical treatment to restore pathways.

Surgical Intervention

Primary end-to-end repair is the gold-standard surgical option to reconnect the severed nerve ends and re-establish a viable pathway for regenerating axons.

Coaptation must be meticulously executed to avoid introducing **stretch or tension** across the repair site, which heavily compromises axonal regrowth.



Surgical Coaptation Mandatory

Spontaneous recovery is anatomically impossible for complete lacerations. Requires direct end-to-end surgical repair with zero tension.

Pathophysiology of Nerve Compression

Pathology & Clinical Etiology

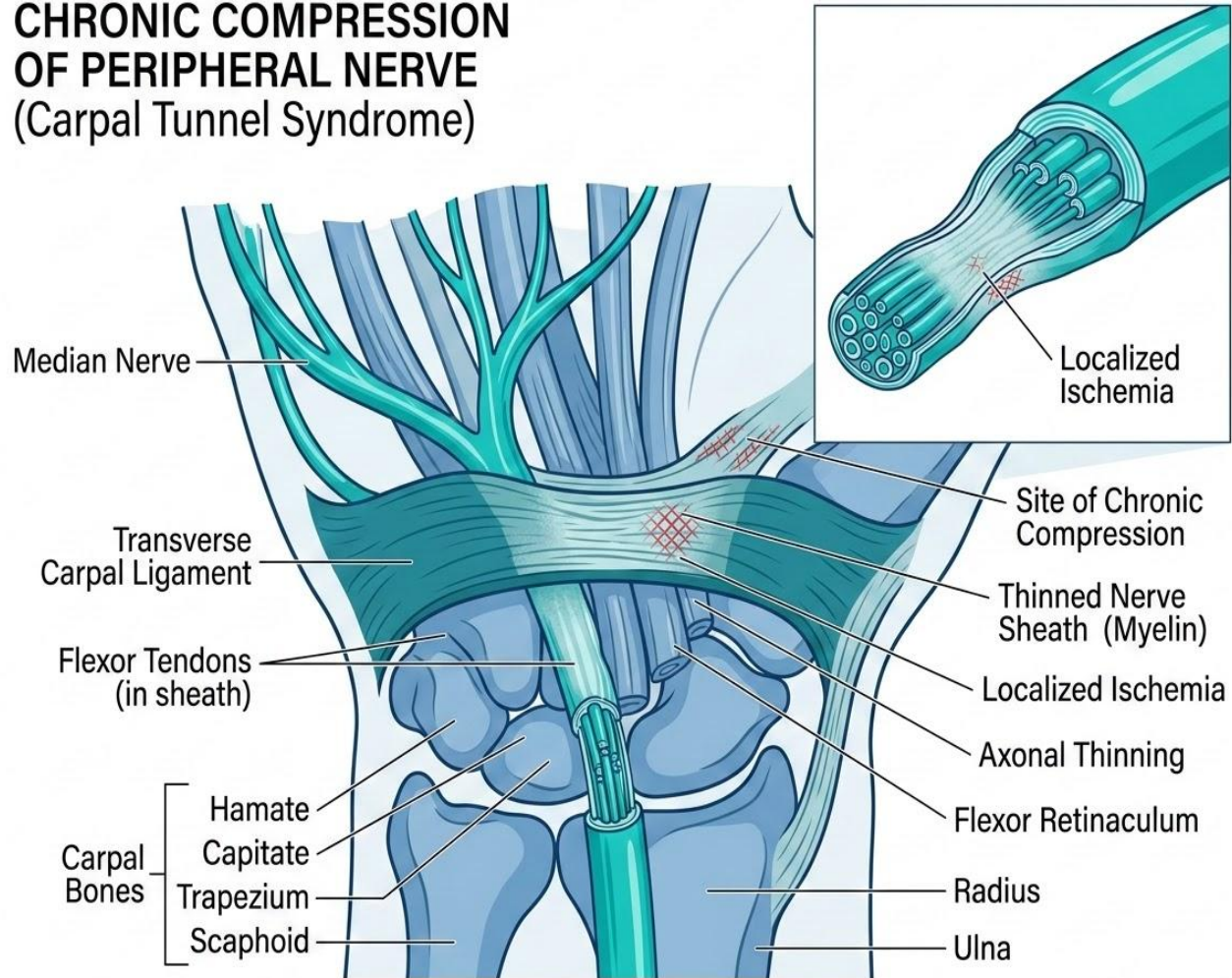
Anatomical Vulnerability: Compression injuries affect nerves crossing over bony structures or passing through rigid, unyielding fibro-osseous spaces.

Onset & Presentation:

- **Acute Compression:** Caused by continuous pressure over hours, such as in "Saturday Night Palsy" or crush injuries.
- **Chronic Compression:** Develops progressively inside narrow anatomical canals, typical of Carpal Tunnel Syndrome.

Ischemic Cascade: External pressure triggers mechanical compression and localized ischemia, compromising the nerve's microcirculation. This vascular compromise leads to localized myelin damage or complete functional loss, even if internal nerve fibers remain structurally intact.

CHRONIC COMPRESSION OF PERIPHERAL NERVE (Carpal Tunnel Syndrome)



+

Classifying Nerve Injuries: Seddon vs. Sunderland

Seddon's Model of Peripheral Nerve Injury

Seddon's model categorizes peripheral nerve injuries into three distinct classes:

Class I

Neuropraxia

The mildest injury, featuring focal demyelination usually secondary to compression. **The whole nerve remains intact, allowing complete recovery within months.**

Class II

Axonotmesis

The axon and myelin sheath are severed, triggering **Wallerian degeneration**. **Because the endoneurium remains intact,** spontaneous recovery is possible but protracted.

Class III

Neurotmesis

Complete physiological disruption and **transection of the entire nerve trunk. No spontaneous regeneration occurs,** making surgical reanastomosis absolutely required.

Sunderland's Classification of Nerve Injury

DEGREE	SEDDON CLASS	COMPROMISED STRUCTURES
1st Degree	Neuropraxia	Local myelin damage with the nerve structurally intact

DEGREE	SEDDON CLASS	COMPROMISED STRUCTURES
2nd Degree	Axonotmesis	Severed axon with an intact endoneurium

DEGREE	SEDDON CLASS	COMPROMISED STRUCTURES (PARTIAL SPARING)
3rd Degree	Axonotmesis	Disruption of both the axon and endoneurial tube, but the perineurium remains intact.

DEGREE	SEDDON CLASS	COMPROMISED STRUCTURES (PARTIAL SPARING)
4th Degree	Axonotmesis	Loss of continuity of the axons, endoneurial tubes, and perineurium; only the epineurium remains intact.

DEGREE	SEDDON CLASS	COMPROMISED STRUCTURES (FULL DISRUPTION)
5th Degree	Neurotmesis	Complete transection of the entire nerve trunk (Neurotmesis).

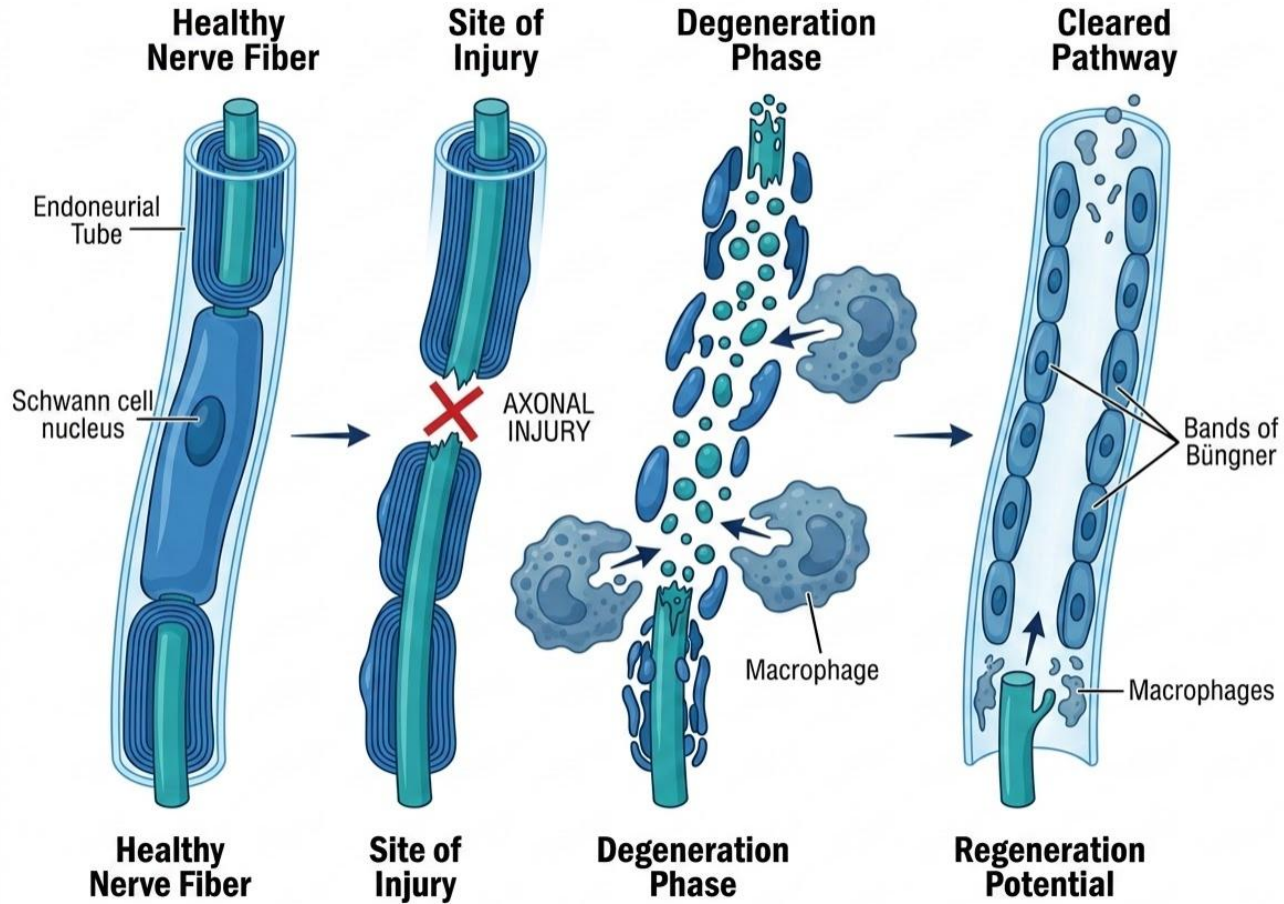


FIG.1. PATHOLOGICAL STAGES OF AXONAL DEGRADATION

Wallerian Degeneration

Distal Nerve Pathology

Following axonotmesis or neurotmesis, physical disruption of the axon compromises the nerve's internal structure and triggers a rapid degenerative cascade:

- + **Axonal Fragmentation:** The severed distal axon segment undergoes **rapid physical breakdown and begins to disintegrate within 24 to 48 hours.**
- + **Myelin Cleansing:** **Schwann cells and infiltrating macrophages actively phagocytose and clear myelin sheath** debris to prepare the pathway.
- + **Scaffold Preservation:** Crucially, **the outer connective tissues are preserved, forming essential pathways to guide regenerating sprouts.**

Physiological Nerve Regeneration

Regeneration Pathway & Progression

Following Wallerian degeneration, nerve regeneration is a slow and highly variable process. When the connective tissue pathways remain intact or are surgically repaired, axons can slowly regenerate from the **proximal stump toward the distal targets**.

Recovery occurs sequentially from **proximal to distal locations relative to the injury site**. Because of this slow physiological progression, successful functional recovery can take up to a maximum of two years.

PHYSIOLOGICAL RATE

1 mm/day

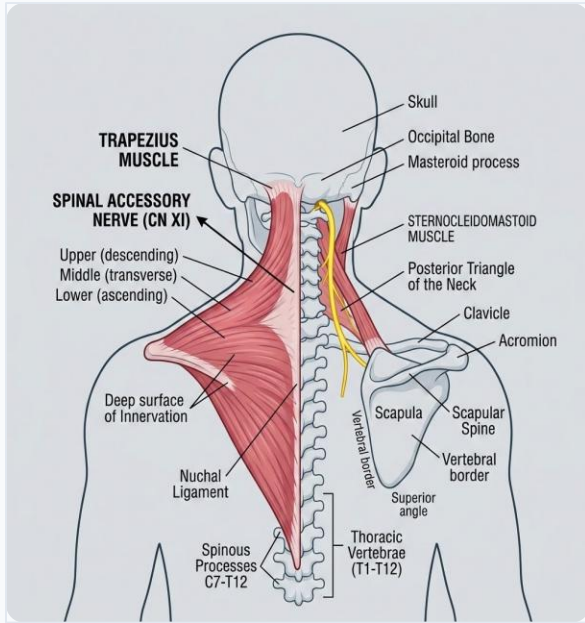
Average Physiological Rate of Axonal
Regeneration under Optimal Conditions

(0.5mm - 2mm daily range)



RECOVERY WINDOW TIMELINE

Surgical intervention may be required if pathways are disrupted, as functional viability decreases over the 2-year regenerative window.



AFFECTED: TRAPEZIUS

Peripheral Nerve Lesions

Spinal Accessory Nerve Impairments Present with Trapezius Dysfunction



RELATED INJURIES

**Fractured Atlas or Hyoid,
Neck Trauma**

MOTOR FUNCTION

- Elevates Pectoral Girdle
- Retracts Scapula
- Depresses Shoulder
- Ipsilateral Neck Flexion

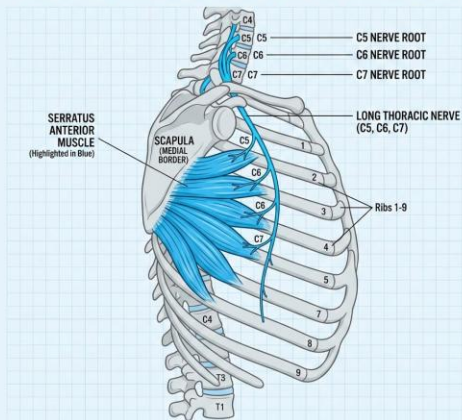
CLINICAL ASSESSMENT

Tests & Observations:

- Observe for scapular winging at rest & evaluate posture.
- Assess shoulder abduction.
- Test hand-behind-back lift away.

PATHOLOGY NOTE Major peripheral nerve injuries lead to severe muscle weakness and consequential atrophy, which can start 72 hours after injury.

ANATOMICAL VECTOR DIAGRAM:
SERRATUS ANTERIOR MUSCLE & C5-C7 NERVE ROOTS



Serratus Anterior Muscle

C5 - C7 ROOTS

Pathology & Clinical Signs

Long Thoracic Nerve Injury & Scapular Winging



Mechanisms of Injury

Commonly caused by stab wounds, sudden scapular depression, or axillary crutch use.

Motor Deficits

- Impaired scapular protraction
- Impaired upward scapular rotation
- Impaired shoulder abduction $> 90^\circ$

Clinical Assessment

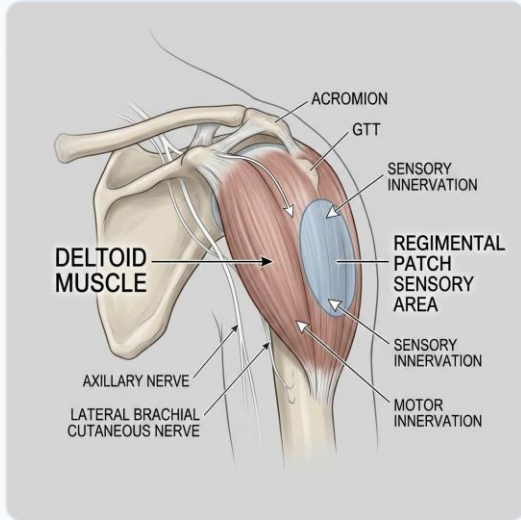
Serratus Anterior Test

Assess scapular winging & protraction strength against resistance (e.g., wall push).

Functional Movement

Record strength during active shoulder abduction $> 90^\circ$.

Sensory Distribution



SENSORY ZONE

Regimental Patch Area

Sensory loss over the lateral aspect of the shoulder is a hallmark sign of axillary nerve lesions.

MOST COMMON PERIPHERAL SHOULDER INJURY

Axillary Nerve Damage

C5-C6 ROOT



RELATED INJURIES

- Shoulder Fracture
- Shoulder Dislocation

MUSCLES AFFECTED

Deltoid, Teres Minor

MOTOR FUNCTION LOSS

- Impaired Abduction > 15°
- Impaired External Rotation
- Deltoid Atrophy (Flat Shoulder)

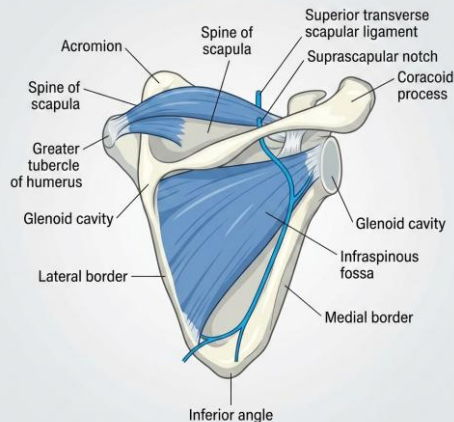


CLINICAL ASSESSMENT

Unable to Abduct Arm to 90°

Patient is unable to initiate or maintain resisted abduction at 90 degrees, highlighting marked impairment in deltoid strength.

C4 - C6 ROOT



ANATOMICAL PATHWAY

Posterior view of the scapular region showing the suprascapular nerve course.

Clinical Presentation

Suprascapular Nerve Lesions Compromise Initial Shoulder Abduction

AFFECTED MUSCLES

- Supraspinatus
- Infraspinatus

RELATED INJURIES



Shoulder, Clavicular, and Proximal Humeral Fractures

MOTOR IMPAIRMENTS

- Impaired Initial 15° Abduction
- Impaired External Rotation
- Shoulder Instability
- Atrophy of Supraspinatus & Infraspinatus

CLINICAL ASSESSMENT

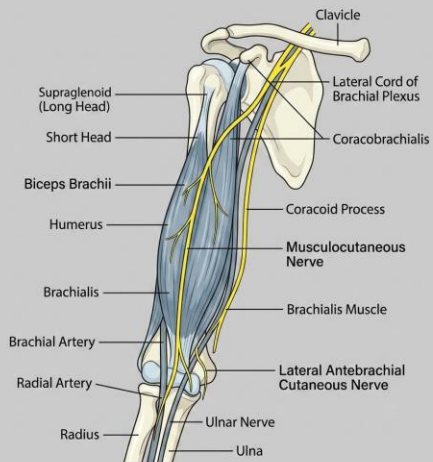
- Infraspinatus Test
- Suprascapular Nerve Stretch Test

NEURAL MOBILITY ASSESSMENT

Consider the path of the nerve and what limb movement puts it under stretch, while carefully avoiding over-stretching.

Musculocutaneous Nerve Injuries Impair Elbow Flexion and Supination

Anatomical Illustration: Anterior Compartment of the Right Arm



C5 - C7 Root



Related Injuries

Isolated injury is rare; typically caused by a Knife wound to the Axilla or Compression.

Motor & Muscles

Muscles Affected

Biceps Brachii, Brachialis, Coracobrachialis

Functional Deficits

- Impaired Elbow Flexion & Supination
- Weak or Absent Biceps Tendon Reflex
- Atrophy of Anterior Arm Compartment

Sensory Function

Cutaneous Branch

Lateral Antebrachial Cutaneous Nerve

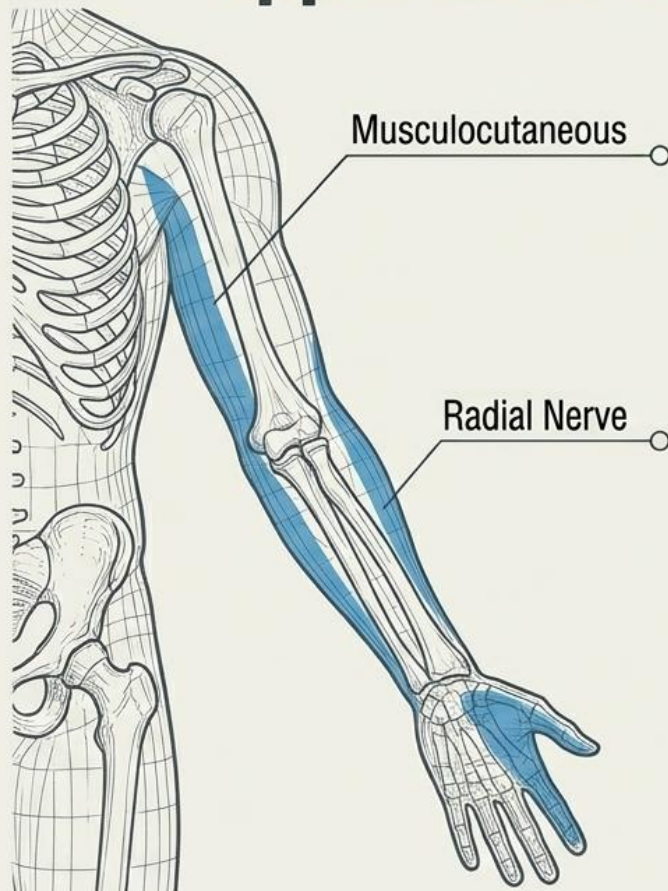
Sensation Loss Area

Loss of sensation over the lateral and volar aspect of the forearm, extending from the elbow down to the base of the thumb.

Clinical Assessment

Patient is unable to Flex the Elbow against resistance when the forearm is supinated.

Upper Limb Topography: Arm & Forearm



Musculocutaneous (Axilla Trauma/Compression)



Biceps, Brachialis, Coracobrachialis
(Impaired elbow flexion, supination).



Lateral and volar aspect of forearm (elbow to thumb base).



Unable to flex elbow; absent biceps reflex.

Radial Nerve (Midshaft Humerus/Elbow Fractures)



High Lesion (Arm): Affects Triceps/Anconeus. Total loss of elbow, wrist, and hand extension.



Mid/Low Lesion (Forearm): Affects Brachioradialis, Supinator, Extensors. Impaired wrist/thumb extension and radial deviation.

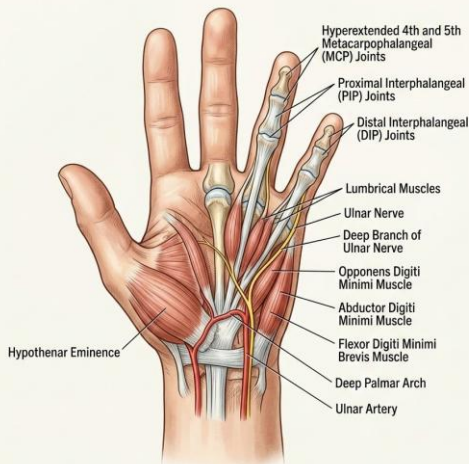


1st webspace; Dorsum aspect from 1st to ½ 4th digit. (No sensory involvement if Posterior Interosseous Branch is damaged alone).



Wrist Drop present; decreased grip strength.

Distinct Hand Deformities Based on Injury Level



ANATOMICAL DIAGRAM OF ULNAR CLAW HAND DEFORMITY.



Related Injuries

Fracture/Dislocation of Elbow,
Laceration at Wrist

High Lesion (Ulnar Paradox)

- Hyperextension of 4th & 5th finger at MCP joint
- Paralysis at IP joint
- Impaired ulnar deviation
- Loss of thumb adduction

Low Lesion (Distal)

- Partial Claw / Ulnar Claw presentation
- Flexion of 4th & 5th finger at IP joint

**Paradox: Low lesions present with more severe visual clawing due to intact FDP muscle action.*



CLINICAL ASSESSMENT & TESTS

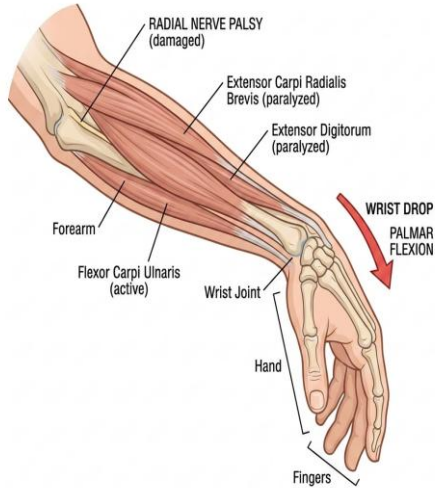
Evaluate hand coordination and motor integrity using targeted clinical maneuvers:

Wartenburg Sign (**abduction of fifth digit**) • Froment Sign (**weak pinch**) • **Inability to cross second and third finger** • **Poor grasp and release coordination.**

Radial Nerve Compression

Wrist Drop & Extension Loss

C5 - T1 ROOT



Related Injuries

- Axilla Compression
- Midshaft Humerus Fracture
- Elbow Fractures
- Lacerations
- Radius Fracture

High Lesion

- Loss of Elbow Extension
- Wrist Extension Loss
- Hand Extension Loss
- Radial Deviation Loss
- Forearm Atrophy

Middle & Low Lesion

- Impaired Wrist Extension
- Impaired Hand Extension
- Impaired Thumb Extension

Clinical Assessment

Wrist Drop is present with decreased grip strength

Sensory Distribution

- 1st Webspace
- Dorsum Aspect Hand from 1st to 1/2 4th Digit

Disrupted Flexion & Thumb Opposition

C5 - T1 ROOT

High Lesion (Proximal)

- Impaired Wrist Flexion: **Weakened flexion and loss of ulnar deviation.**
- Flexion Deficit: **Inability to flex the thumb and index finger.**
- Deformity: **Presents clinically as the 'Hand of Benediction'.**

Low Lesion (Distal)

- Impaired Opposition: **Selective loss of thumb opposition.**
- Thenar Wasting: **Severe muscular atrophy of the thenar eminence.**
- Deformity: **Presents clinically as the 'Median Claw'.**

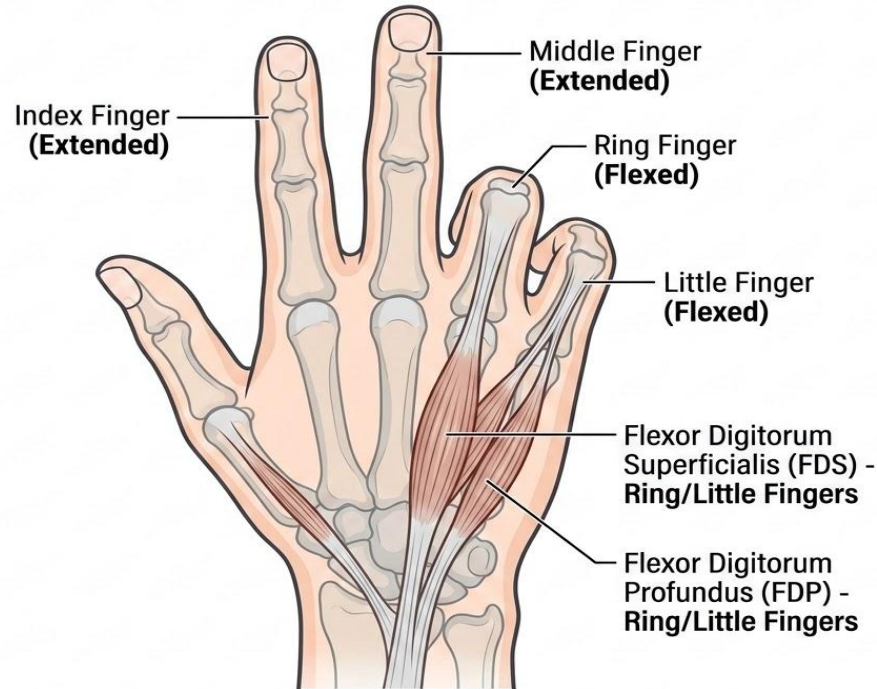
Carpal Tunnel Pathology

- Thenar Wasting: **Gradual loss of thenar muscle tissue density.**
- Motor Deficit: **Progressive weakness of Abductor Pollicis Brevis.**
- Deformity: **Advanced cases present as 'Ape Hand'.**

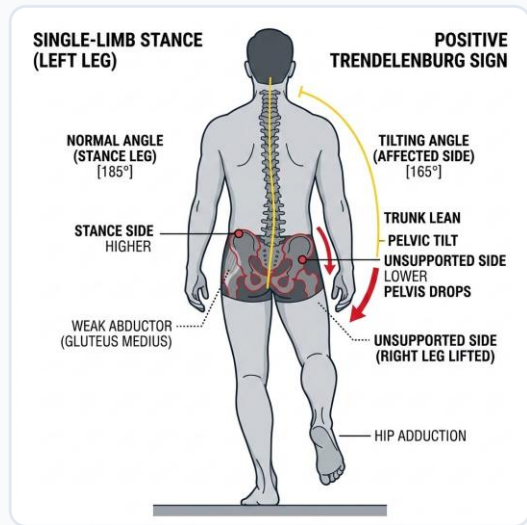
Clinical Assessment

- Sensation Loss: **Palmar aspect of 1st, 2nd, 3rd, and ½ of 4th digits.**
- Clinical Tests: **OK Sign and Pinch Test diagnostic evaluation.**
- Trauma Association: **Supracondylar fractures & wrist lacerations.**

HAND OF BENEDICTION DEFORMITY (Posterior View)



ASSESSMENT VISUALIZATION



TRENDELENBURG GAIT

Contralateral pelvic drop occurs during single-limb stance on the affected side due to weak hip abductors (gluteus medius).

SUPERIOR GLUTEAL NERVE PATHOLOGY

Gluteal Nerve Injury

Manifests as Impaired Hip Abduction & Gait Instability

AFFECTED: GLUTEUS MEDIUS



RELATED INJURIES

Hip Dislocation

High-risk in posterior joint disruptions and severe pelvic trauma.

CLINICAL ASSESSMENT

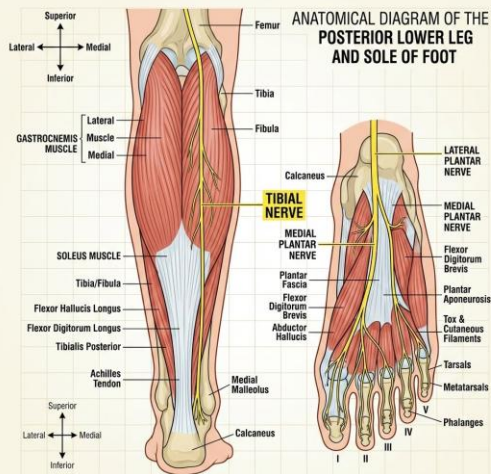
Trendelenburg Test

A positive sign is indicated when the pelvis sags on the unsupported side, demonstrating functional weakness of the stance-limb abductors.

TRAUMA CONTEXT & DIAGNOSTIC OUTLOOK

In disaster and conflict situations, major peripheral nerve injuries in the lower extremity are the most common site of the single most significant injury. Diagnosis is generally clinical, based upon neurologic symptoms consistent with nerve injury along with a history of acute or repetitive trauma.

L4 - S3 ROOT



⚠️ RELATED INJURIES

Dislocation of the Knee, Fracture of the Tibia or Fibula, Laceration Injury.

TIBIAL NERVE LESION

Damage Compromises Plantarflexion & Toe Function

MUSCLES AFFECTED

- ⦿ Biceps Femoris (Long Head)
- ⦿ Popliteus
- ⦿ Gastrocnemius
- ⦿ Soleus & Plantaris
- ⦿ Flexor Digitorum Longus
- ⦿ Flexor Hallucis Longus

MOTOR DEFICITS

- **Impaired Plantarflexion**
- **Impaired Inversion**
- **Unable to Curl Toes**

SENSORY LOSS

Bottom of the foot & toes
Loss of protective sensation on plantar surfaces.

CLINICAL ASSESSMENT

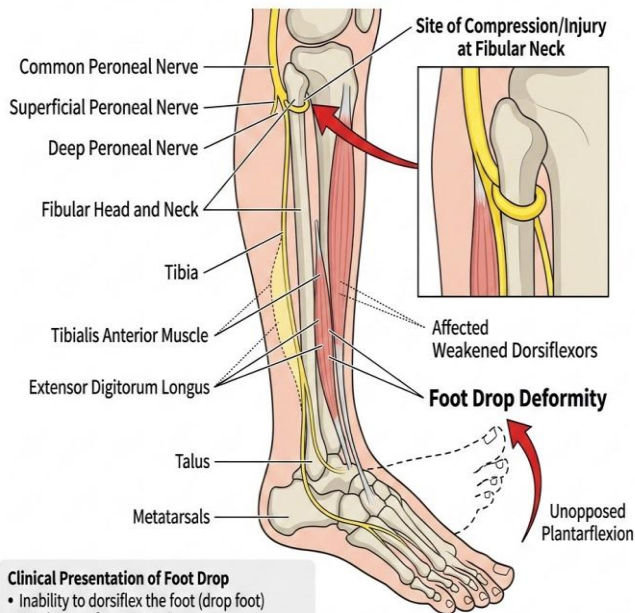
Patient is unable to walk on their toes.

Peroneal Nerve Injuries

L4 - S2 ROOT

CLINICAL ANATOMY:

FOOT DROP AND COMMON PERONEAL NERVE PALSY



Clinical Presentation of Foot Drop

- Inability to dorsiflex the foot (drop foot)
- Weakness of toe extension
- Loss of sensation over the dorsum of the foot
- Compensatory high-steppage gait



RELATED INJURIES & PATHOLOGY

Dislocation of the knee, fracture of the fibular head, and trauma or nerve entrapment at the fibular head/neck.

MOTOR FUNCTION

Muscles Affected:

Biceps femoris, Tibialis anterior

Deficits:

Impaired dorsiflexion, impaired eversion,

SENSORY FUNCTION

Sensory Loss Distribution:

- **Lateral surface of lower leg**
- **Dorsum of feet & toes (except 1st/2nd web space)**

CLINICAL ASSESSMENT

Heel-Walking Test: Patient is unable to walk on their heels, demonstrating marked weakness in ankle dorsiflexion.

Brachial Plexus Injuries Often Stem from Crush or Penetrating Trauma

MECHANISM OF INJURY

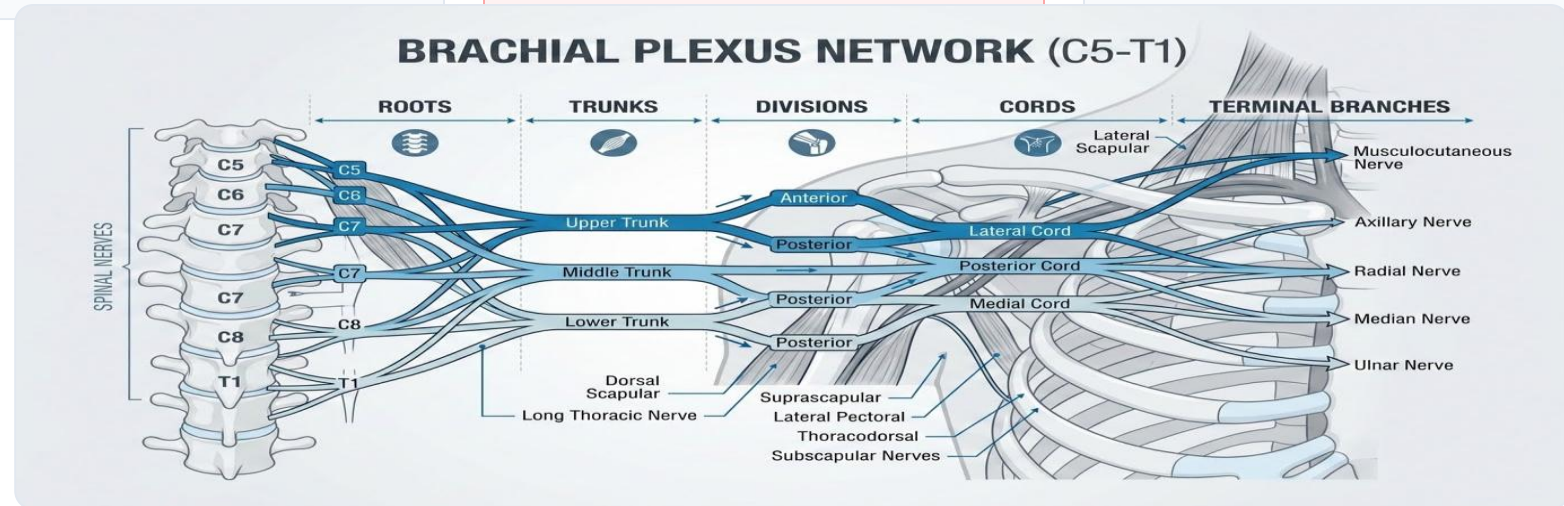
Brachial Plexus Injuries can be caused by penetrating injury and blunt mechanisms, such as in a crush injury.

CLINICAL PRESENTATION

Proximal nerve lesions are more likely to cause hand distortions (e.g., hand of benediction), which are only visible when the patient tries to flex the fingers or make a fist.

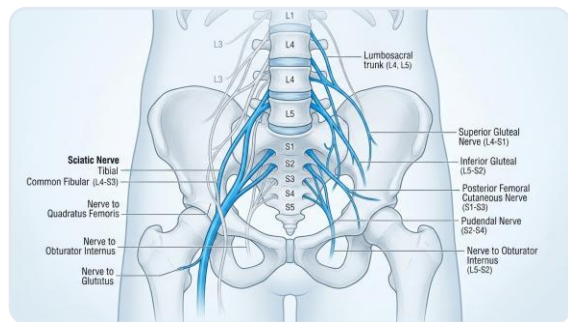
MANAGEMENT

An arm sling may be used to prevent subluxation in Brachial Plexus Injuries.



PELVIC & HIP TRAUMA COMPLICATIONS

Sacral and Lumbosacral Plexus Injuries Complicate Pelvic and Hip Trauma



MECHANISM OF INJURY

Sacral Plexus injuries can be caused by pelvic fractures, hip surgery, and the use of traction.

<3%

PELVIC FRACTURES

Injured in less than 3% of pelvic fractures

13%

POSTERIOR DISLOCATIONS

In 10% to 13% of posterior dislocations of the hip

REHABILITATION PRIORITY

Early Rehabilitation Focus: Early rehabilitation should focus on oedema management, pain management, range of movement, positioning, graded exercise, and weight-bearing once fractures are stabilized.

Urgent clinical red flags requiring immediate medical escalation



Compartment Syndrome

Trigger: Red, hot, swollen limb; severe pain worsened with passive movement.



DVT / Pulmonary Embolism

Trigger: Red, hot, swollen limb with lack of pulse; shortness of breath at rest.

**STOP
TREATMENT**



Unstable Fracture

Trigger: Severe pain with visible malalignment or abnormal bony movement/shape.

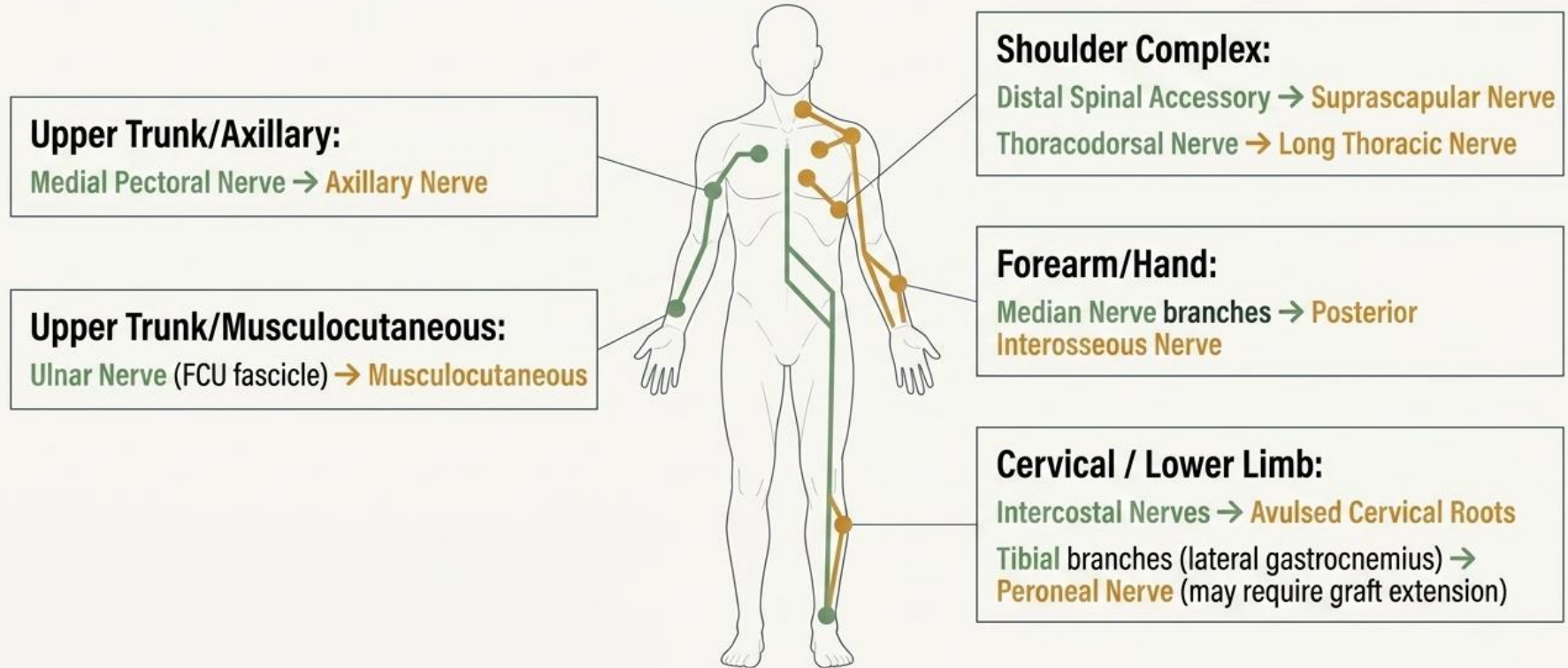


Cervical Spine Instability

Trigger: Bilateral pins/needles, weakness in hands/feet, cranial symptoms, inability to maintain posture.

The Surgical Matrix: Donor Nerve Routing

Autografts/conduits for delayed Grade V injuries



Physiotherapy Management



CLINICAL & ACADEMIC PHYSIOTHERAPY

Rehabilitation Principles & Objectives

Core Rehabilitation Aims

Rehabilitation aims to restore independent function and educate patients on realistic management strategies.

Long-Term Strategy

Since nerve regeneration can take up to two years, establishing long-term, independent patient management is essential.

Core Objectives Across Phases

- ✓ Oedema management
- ✓ Pain modulation
- ✓ Maintaining range of motion
- ✓ Preventing contractures through splinting
- ✓ Graded progressive exercise to preserve muscle function
- ✓ Sensory re-education to aid recovery



Phase 1 Rehabilitation: Protecting ICF Body Structures

Intervention Dashboard

Pain Modulation

(Targeting neuropathic pain)

TENS:

100Hz constant mode. Delay application until one-week post-trauma for maximum NMJ benefit.

Adjuvant Therapies:

Low-Level Laser Therapy (LLLT),
Aromatherapy Massage.

Oedema Control

(Preventing secondary compartment syndrome)

Positioning:

Elevate extremities. Facial oedema requires sitting at 45° for 24 hours/day.

Active Care:

Use active muscle pump action. Ensure dressings are firm but allow AROM.



WARNING: NO HEAT OR ICE. Due to insensate skin, thermal modalities carry an extreme risk of severe burns/tissue damage.

Edema & Soft Tissue Management

Swelling Risks: Excessive swelling can hinder wound healing, cause joint stiffness, and increase the risk of compartment syndrome.

'Peace & Love' Principle: Guides soft tissue management, emphasizing comprehensive patient education and active psychosocial support.

Acute Extremity Positioning: Requires appropriate elevation and utilizing muscle pump action through active range of motion exercises.



Never apply heat or ice due to sensory loss. Alert medical teams immediately for a hot, swollen limb and pain worsened by passive movement.

Neuropathic Pain Modulation

Clinical Presentation

Severe neuropathic pain can exhaust patients and hinder rehabilitation.

Effective pain modulation requires a multidisciplinary approach.

Pharmacological Therapies

Pharmacological treatments for neuropathic pain often include gabapentin, amitriptyline, and pregabalin.

100Hz

TENS pain-modulation frequency

Complementary Modalities

Physiotherapy modalities serve as complementary pain relief.

- Transcutaneous Electrical Nerve Stimulation (TENS) applied at 100Hz in a constant mode is beneficial for reducing neuropathic pain.
- Additionally, Low-Level Laser Therapy (LLLT) assists in pain relief and accelerates healing for neurological deficits.

Range of Motion & Safe Mobilization

4-6x

Daily Range of Motion Sessions

Physiological Benefits

Performing regular passive, active-assisted, and active exercises is crucial for healing, aiming directly to:

- Maintain joint and soft tissue mobility
- Minimize painful contracture formation
- Reduce risks associated with pressure ulcers
- Enhance healthy synovial fluid movement

Neural Mobilization Guidelines



Smooth Gliding: When performing mobilization, the nerve must be glided in a smooth, continuous movement.



Avoid Traction: It is critical to avoid movements that overstretch or place traction on the affected nerve.



CRITICAL SAFEGUARD

Therapeutic stretching must **never** induce paresthesia under any circumstances.

Splinting & Positioning Objectives

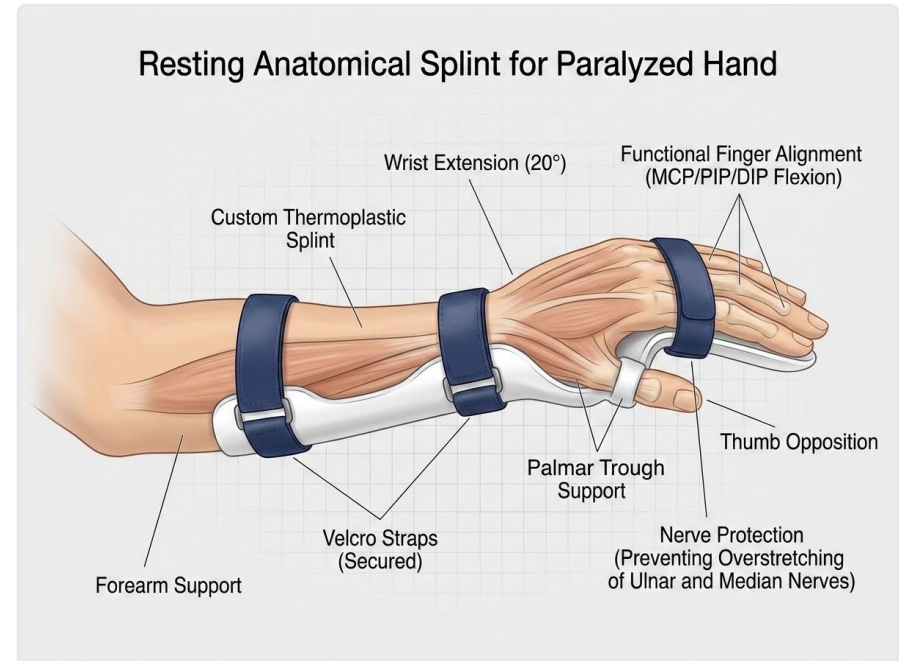
Immediate positioning and splinting are vital to maintain optimum joint alignment, reduce pain, and prevent compensatory movements.

Primary Clinical Objectives

- Protecting injured tissues and minimizing oedema.
- Preventing contracture formation.
- Compensating for lost motor function and facilitating daily activities by resting paralyzed muscles in optimal positions.

Clinical Precaution

It is essential to prevent any postures that could overstretch or place traction on the healing nerve.



Splinting Protocols for Peripheral Nerve Lesions

Specific splinting designs counteract motor loss and muscle imbalance associated with individual nerve lesions.

Upper Limb Orthoses

Radial Nerve Palsy: A Plaster of Paris splint prevents wrist drop.

Ulnar Nerve Lesions: A modified aluminium finger splint prevents the clawing of fingers.

Median Nerve Lesions: Adhesive tape can maintain the thumb in apposition during the night.

90°

Ankle Angle Required to Prevent Foot Drop

Lower Limb Orthoses

Sciatic & Lateral Peroneal Nerve Lesions: Lower limb injuries require night splints to maintain the ankle at 90 degrees, effectively preventing foot drop.



Dynamic Radial Outrigger Splint



The mechanics of field-expedient splinting

Clinical Objective	Field Material
Radial Nerve (Prevent Wrist Drop)	Plaster of Paris
Brachial Plexus (Prevent Subluxation)	Standard Arm Sling
Ulnar Nerve (Prevent Clawing)	Modified aluminum finger splints
Median Nerve (Thumb Apposition)	Adhesive tape at night
Peroneal/Sciatic (Prevent Foot Drop)	Night splint maintaining ankle at 90°

The Splint Care Rule of 3

- ✓ **Check:** Remove and check for red marks. Must fade in 30 mins.
- ✓ **Wash:** Clean with cold/lukewarm water. Hot water warps plastics.
- ✓ **Adjust:** Do not alter unfamiliar custom splints; refer back to maker.

Peripheral Nerve Injury & NMJ Recovery

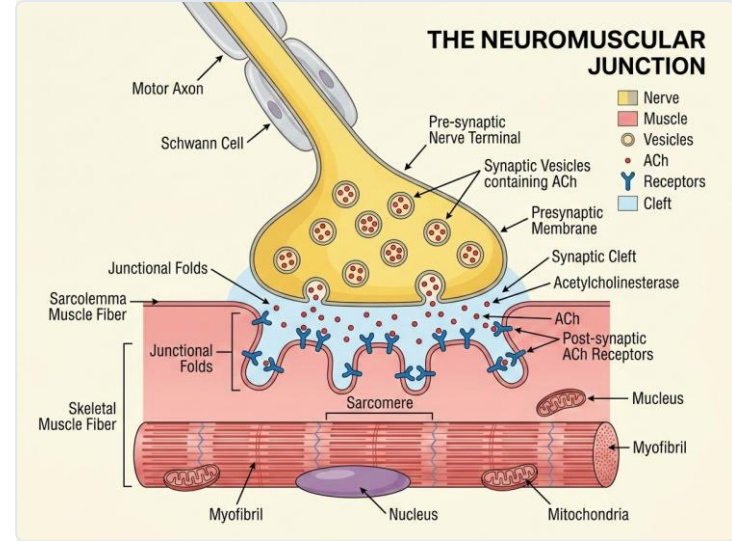
72h

Atrophy Onset Post-Injury

NMJ Degradation Timeline

Major peripheral nerve injuries lead to severe muscle weakness and rapid atrophy, which initiates within **72 hours** of the trauma.

The neuromuscular junction (NMJ) undergoes significant pathological changes and serves as the **most critical point** for establishing long-term functional recovery.



TENS Intervention Protocol

Transcutaneous Electrical Nerve Stimulation (TENS) helps maintain NMJ health. Evidence indicates optimal benefits when treatment is **delayed 1 week post-injury** and applied at **100Hz**.

Phase 2 Rehabilitation: Restoring Motor Function (ICF Activities)

Step 1: Isometric

Often the only safe exercise in acute complex fractures.



Step 2: Gravity-eliminated

Utilize slings/support to assist movement.



Step 3: Resisted & Open/Closed Chain



Weight-Bearing: Essential once fractures/nerves are stabilized to stimulate neural pathways.

Progression Staircase



THE LIMIT LINE

If pins and needles, numbness, or increased pain occur, the exercise is too hard. Back down immediately to prevent damage to healing nervous tissue.

Graded Exercise & Clinical Boundaries

✓ Graded Exercise Protocol

Individualized Graded Program

Progression improves muscle strength while safeguarding vulnerable healing tissues:

- **Isometric Stage:** The only permissible activity in the acute phase of complex injuries.
- **Anti-Gravity:** Support slings can assist movement and unweight the limb.
- **Resisted-Range:** Advanced phase targeting functional load and full strength recovery.

***Pacing:** Pacing is vital throughout all phases to minimize fatigue and protect nerve healing.*

⚠ Safety & Red Flags

Protect Healing Nervous Tissue

If any of these neurovascular warning boundaries are crossed, exercise intensity is too high:

- **Pins & Needles (Paresthesia)**
Indicates transient ischemia or mechanical nerve irritation.
- **Numbness / Sensory Deficit**
Signifies complete or partial block of neural sensory pathways.
- **Increased Local/Radiating Pain**
A clear signal of tissue distress and excessive physical load.

Sensory Re-Education & Recovery

Sensory re-education is essential for the recovery of sensibility and functional task relearning. The brain is utilized to regain visio-tactile and audio-tactile interactions, particularly focusing on fine motor skills in the upper limb and coordination in the lower limb.

Sensory Stimulation Techniques

- **Pinching & Tapping:** For localized tactile re-education and sensory mapping.
- **Brushing:** Desensitization protocols and controlled cutaneous stimulation.
- **Icing:** Thermal stimulation to enhance neural receptor response thresholds.

Addressing Proprioceptive Deficits

- **Exercise Balls:** Targeted training to promote core stability and balance.
- **Balance Pads:** Therapeutic weight-bearing activities to rebuild joint awareness.



Weight-Bearing & Mobility Retraining



CRITICAL WEIGHT-BEARING RESTRICTIONS

Surgical & Medical Compliance

Adherence to weight-bearing restrictions prescribed by the medical or surgical team is vital for patient recovery.

Premature Weight-Bearing Warning:

Loading joints or limbs too early can significantly delay bone and soft-tissue healing, leading to potential fixation failures or anatomical instability.



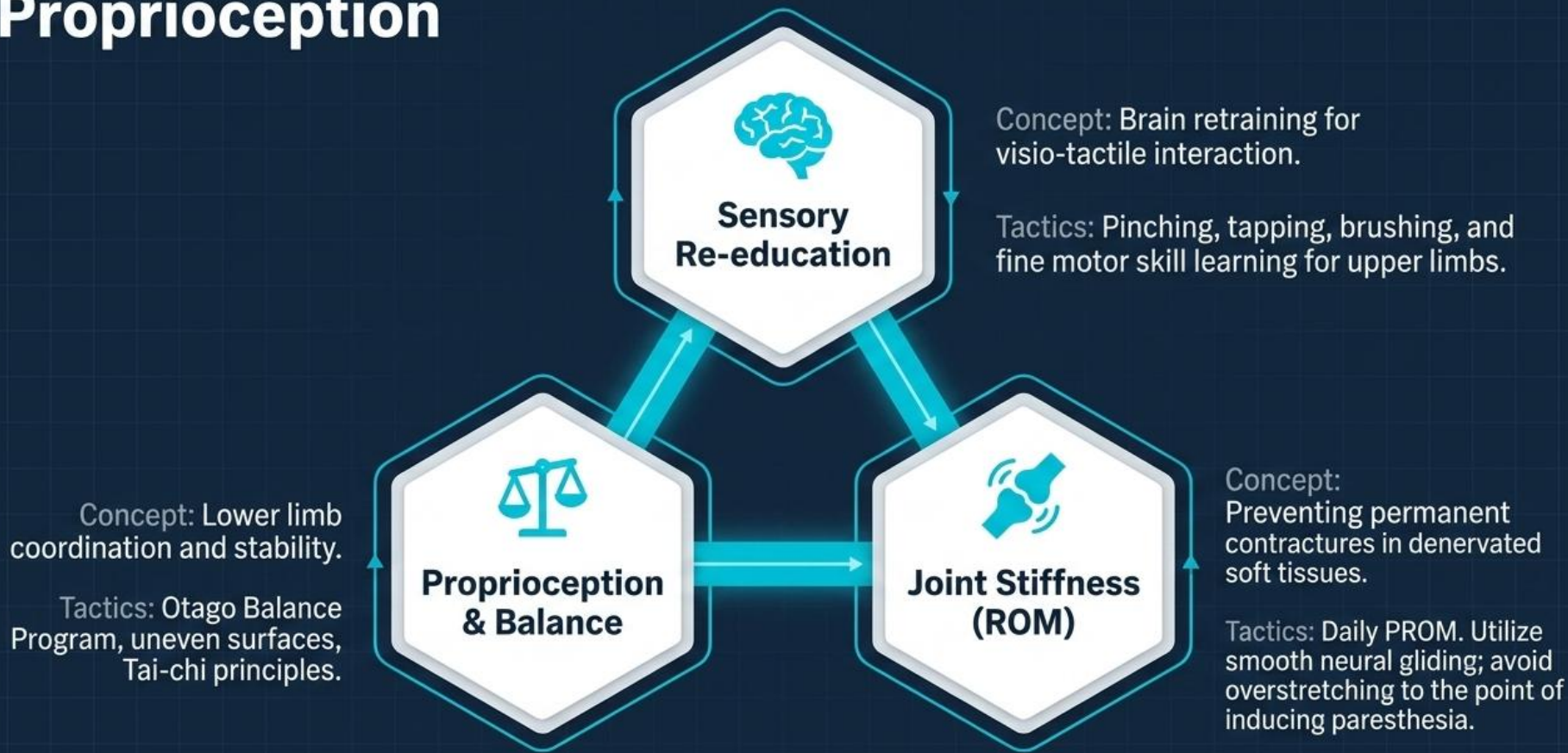
STABLE WEIGHT-BEARING & REHAB

Early Mobilization & Neural Benefits

Once fractures and nerve injuries are stable, fixed, or immobilized, initiate weight-bearing ASAP for both upper and lower extremities.

- **Neural Activation:** Early and safe loading stimulates motor control and reinforces crucial sensory pathways.
- **Functional Retraining:** Direct therapeutic focus toward safe transfer mechanics and structured mobility practice.

Phase 3 Rehabilitation: Sensory Re-education & Proprioception



Psychological Impact of Nerve Injuries

Addressing Mental Health and Clinical Quality of Life (QOL) in PNI Rehabilitation

The Psychological Toll of PNI

Peripheral nerve injuries profoundly impact mental health due to muscle weakness, visible deformities, and severe pain.

Chronic neuropathic pain has a life-debilitating effect, causing significant emotional stress and a reduced quality of life.

Clinical Priorities for QOL

- Post-traumatic stress disorder
- Depression and anxiety
- Associated psychological considerations
- Sleep disorders
- **Improved overall quality of life**



Patient & Caregiver Education

Crucial home care and protective protocols for successful long-term management

Daily Safety Protocols

- **Meticulous Skin Inspection:** Teach patients systematic daily inspections using a hand mirror to spot early pressure spots or redness.
- **Moisture & Temperature Safety:** Avoid extreme water temperatures to prevent thermal injuries; thoroughly dry skin folds to maintain skin integrity.
- **Continuous Protective Habit:** Always wear protective shoes to prevent silent mechanical cuts and injuries to insensate zones.
- **Contracture Prevention:** Consistently maintain passive range of motion to prevent joint and tendon contractures during long healing timeframes.



Contraindication Alert: Never apply heat or cold packs to insensate areas. Potential sensory loss significantly increases the risk of rapid, undetected burns and severe tissue damage.



THANK YOU