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Proximal Median Nerve Compression Around the Elbow: A Narrative Review

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Abstract

Background: Median nerve compression at the elbow exists on a biomechanical spectrum. Static compression involves persistent constriction within rigid tunnels, while dynamic compression occurs during specific movements and muscle activation. This fundamental distinction creates unique diagnostic and therapeutic challenges. The clinical presentation is a constellation of sensory, motor, and provocative symptoms that localize the pathology to the proximal forearm.

Objective: This review aims to summarize the anatomy, pathophysiology, clinical presentation, diagnostic evaluation, and treatment of proximal median nerve compression around the elbow.

Conclusion: Diagnosis requires dynamic provocative testing rather than standard nerve conduction studies. Treatment must address motion-induced constrictors through targeted surgical releases, emphasizing the need for tailored approaches distinct from those used for static compression. When appropriate patient selection and complete decompression are achieved, proximal median nerve release generally provides excellent symptom relief and high patient satisfaction.

Keywords: Median nerve compression; Lacertus syndrome; Pronator syndrome; Dynamic ultrasound; Clinical triad; Surgical release.

Introduction

The median nerve, formed by the union of the lateral and medial cords of the brachial plexus (C5–T1), travels alongside the brachial artery through the arm and forearm, passing between the pronator teres heads and beneath the flexor digitorum superficialis arch. It supplies motor fibers to most forearm flexors and thenar muscles, and sensory fibers to the palmar thumb, index, middle, and radial half of the ring finger. Its long course through multiple fibro-osseous tunnels makes it highly susceptible to compression at several anatomical sites. (1)

The elbow region represents a complex anatomical tunnel where the median nerve is particularly vulnerable to compression at several specific sites as it transitions from the arm to the forearm. Its precise anatomical relationships are critical to understanding various entrapment neuropathies. (2)

Far more common are the compressions of the median nerve (MN) in the proximal forearm, collectively called proximal median nerve entrapments (PMNE). Recent publications have shown that one of the primary compression points is at the level of the lacertus fibrosus (LF), in what is known as the lacertus syndrome

Nerve compression syndromes, characterized by the mechanical and often dynamic compression of peripheral nerves, are a significant yet frequently under appreciated cause of pain, muscle weakness, and functional impairment. These conditions often disrupt daily activities and diminish quality of life. Traditional diagnostic methods, including electromyography (EMG) and imaging techniques, have limitations, particularly in detecting nerve compressions in their early stages. Electrodiagnostic studies (EDX) are commonly employed to evaluate suspected nerve entrapments, particularly in the upper extremities. However, their sensitivity and specificity, especially for conditions beyond carpal and cubital tunnel syndromes, are limited, typically ranging between 30

and 65% . EDX may also struggle to detect mixed nerve injuries and is unable to provide comprehensive muscle function assessments or identify early-stage compressions. Therefore, clinical examination techniques should be integrated with or prioritized over sole reliance on EDX. (3)

In the early stages of nerve compression, dynamic pressure on the nerve can disrupt saltatory conduction and lead to dynamic nerve ischaemia. Mackinnon and colleagues coined the term "Sunderland Zero" to describe this level of nerve impairment, characterized by dynamic nerve compression without axonal loss .

These compressions typically affect superficial nerve fascicles at the site of pressure, resulting in sensory or motor deficits—or both—depending on the nerve segment affected.

To diagnose nerve compressions effectively, clinical evaluations must assess both motor and sensory function while identifying pain at the compression site. A clinical triad for a structured assessment of median nerve compressions was first described in 2013 , and has since been described in upper extremity nerve compression syndromes . (4).

Anatomy of Median Nerve

The median nerve, formed by the union of the lateral and medial cords of the brachial plexus (C5–T1), travels alongside the brachial artery through the arm and forearm, passing between the pronator teres heads and beneath the flexor digitorum superficialis arch. It supplies motor fibers to most forearm flexors and thenar muscles, and sensory fibers to the palmar thumb, index, middle, and radial half of the ring finger. Its long course through multiple fibro-osseous tunnels makes it highly susceptible to compression at several anatomical sites. (1)

Course of the Median Nerve

The median nerve embarks on a lengthy journey from the axilla down to the hand, maintaining close relationships with various muscles, arteries, and bones along its path. These anatomical relationships are crucial as they define potential sites of compression.

In the Elbow Region

The elbow region is a critical area for median nerve anatomy due to the numerous structures that can potentially compress the nerve. As the median nerve approaches the elbow, it lies medial to the brachial artery and anterior to the medial epicondyle of the humerus. It then passes through the cubital fossa.(5)

Relations of the Median Nerve

The elbow region represents a complex anatomical tunnel where the median nerve is particularly vulnerable to compression at several specific sites as it transitions from the arm to the forearm. Its precise anatomical relationships are critical to understanding various entrapment neuropathies. (2)

Lacertus Fibrosus (Bicipital Aponeurosis):

Situated at a critical anatomical junction in the proximal forearm, the lacertus fibrosus—or bicipital aponeurosis—is a resilient fibrous expansion that originates from the biceps tendon and seamlessly blends into the deep antebrachial fascia. As the median nerve courses distally into the forearm, it is obligated to pass directly beneath this fascial layer.

this anatomical relationship poses a significant risk of entrapment, as the lacertus fibrosus can act as an unyielding fascial tether. During biomechanical actions that require repetitive or forceful forearm pronation, this aponeurosis dynamically tightens. This tension effectively transforms the fibrous band into a constricting guillotine, exerting focal, transient pressure on the underlying median nerve and triggering the distinct compressive neuropathy known as Lacertus Syndrome (6).

Pronator Teres Muscle: This is the most cited site of median nerve entrapment in the forearm, leading to Pronator Syndrome. The nerve passes between the humeral (superficial) and ulnar (deep) heads of the pronator teres muscle . Compression here can be caused by hypertrophy of the muscle (e.g., from repetitive use), fibrous bands between the two heads, or an abnormally tight tendinous arch of the humeral head (7)

Flexor Digitorum Superficialis (FDS) Arch: Distal to the pronator teres, the median nerve travels underneath a fibrous arch formed by the proximal edge of the flexor digitorum superficialis muscle. A thick, unyielding, or hypertrophied arch can compress the nerve, often presenting with pain in the proximal forearm and paresthesia that worsens during repetitive gripping activities (6) .

Ligament of Struthers: This is an anomalous structure present in approximately 1-3% of the population. When a supracondylar process (a bony spur on the distal humerus) exists, the ligament of Struthers can connect it to the medial epicondyle. The median nerve (and often the brachial artery) passes under this ligament, creating a potential site for compression high above the elbow, termed Supracondylar Process Syndrome (8)

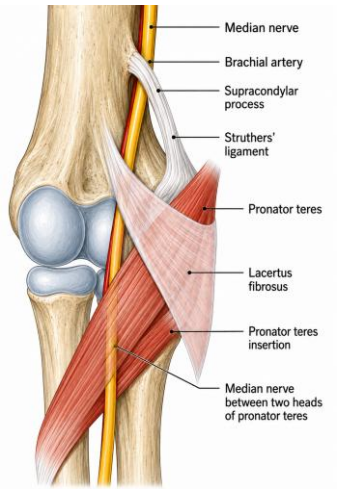

Figure 1: A detailed view of the median nerve's anatomical relations at the elbow, highlighting potential compression points.

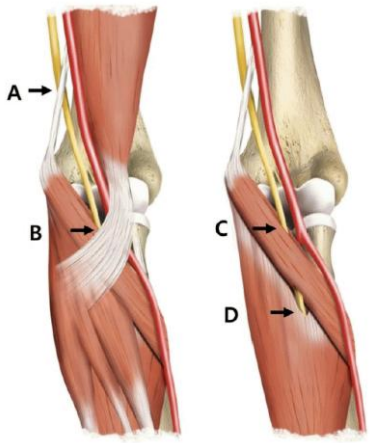

Figure 2: Potential sites of proximal median nerve compression around the elbow. A) Ligament of Struthers B) Lacertus fibrosus (bicipital aponeurosis) (C) Pronator teres muscle, (D) Fibrous arch of the flexor digitorum superficialis (FDS)

3. Anterior Interosseous Nerve (AIN):

The AIN is a major purely motor branch that arises from the median nerve just distal to the pronator teres. It descends down the forearm along the anterior aspect of the interosseous membrane, between the FDP and FPL, to reach the pronator quadratus .

It innervates three key muscles:

Flexor pollicis longus (FPL): Flexes the thumb tip (IP joint).

Lateral (radial) half of Flexor Digitorum Profundus (FDP): Flexes the tips of the index and middle fingers (DIP joints).

Pronator quadratus (PQ): Provides the primary pronation force of the forearm.

Anterior Interosseous Nerve Syndrome (AINS) is a compression neuropathy of this branch, resulting in a characteristic inability to form the "OK" sign (due to FPL and FDP weakness). Crucially, it involves no sensory loss. (9)

4. Palmar Cutaneous Branch:

This sensory branch arises from the radial side of the median nerve in the distal forearm, approximately 5 cm proximal to the wrist crease. It pierces the antebrachial fascia and travels superficial to the flexor retinaculum . This is a critical anatomical distinction. It innervates the skin over the lateral (thenar) aspect of the palm. Because it does not pass through the carpal tunnel, sensation in this area is typically preserved in Carpal Tunnel Syndrome (CTS), helping to differentiate it from more proximal compression or a brachial plexus lesion.(10)

Pathophysiology of Median Nerve Compression

(1) From Mechanical Stress to Neural Dysfunction

Compression above capillary pressure (20–30 mmHg) reduces neural blood flow, causing ischemia, inflammation, and endoneurial edema. Rising intraneural pressure then creates a cycle of further ischemia and nerve dysfunction (11).

(2) Progression of Injury

Early compression causes reversible demyelination and intermittent paresthesia. Prolonged compression leads to fibrosis, axonal degeneration, permanent sensory loss, weakness, and muscle atrophy (12).

Site-Specific Pathology at the Elbow

The lacertus fibrosus causes dynamic compression during elbow flexion and forearm pronation. The pronator teres produces a scissor-like effect, while the FDS arch acts as a fixed constrictor (6).

(3) Molecular Mechanisms

Ischemia–reperfusion produces oxidative stress, while Schwann cells and inflammatory cytokines such as TNF-α and IL-1β promote axonal injury and neuropathic pain (13).

Nature of Median Nerve Compression at the Elbow: Dynamic vs. Static

Median nerve compression at the elbow exists on a biomechanical spectrum. Static compression involves persistent constriction within rigid tunnels, while dynamic compression occurs during specific movements and muscle activation. This fundamental distinction creates unique diagnostic and therapeutic challenges (6)

Mechanisms of Dynamic Compression Dynamic compression involves transient, recurrent pressure during functional movements. The lacertus fibrosus tightens during elbow flexion and pronation, creating a pincer-like effect on the nerve. Simultaneously, the pronator teres contributes through a scissor-like action between its two heads. This mechanism combines direct compression with impaired nerve gliding and traction forces, producing a unique pattern of intermittent injury (14)

Lacertus Syndrome: A Paradigm Lacertus Syndrome exemplifies dynamic compression with characteristic activity-induced symptoms triggered by resisted elbow flexion and pronation. Diagnosis requires dynamic ultrasound, which shows real-time nerve deformation during muscle contraction—contrasting sharply with the static nerve swelling seen in conditions like carpal tunnel syndrome (15)

Pathophysiological Differences The injury patterns differ fundamentally: dynamic compression creates cyclical stress with ischemia-reperfusion injury and oxidative damage, while static compression causes chronic hypoxia

and progressive demyelination. This explains why dynamic compression causes intermittent symptoms relieved by rest, unlike persistent deficits in static compression (16)

Clinical Implications Diagnosis requires dynamic provocative testing rather than standard nerve conduction studies. Treatment must address motion-induced constrictors through targeted surgical releases, emphasizing the need for tailored approaches distinct from those used for static compression (14)

Sunderland "Zero" injury Theory

The diagnosis of Lacertus Syndrome—a dynamic compression of the median nerve by the bicipital aponeurosis (lacertus fibrosus) at the elbow—is clinically and pathophysiologically aligned with a Sunderland "Zero" injury, a concept refined by Mackinnon et al. (2019) to describe a state of physiological nerve dysfunction without structural axonal damage.

This syndrome presents with a specific pattern of motor weakness (e.g., in the Flexor Carpi Radialis and Abductor Pollicis Brevis) and sensory abnormalities in the median nerve territory, as detailed in diagnostic algorithms. Its dynamic nature means symptoms are often provoked by specific arm positions or repetitive activities. Crucially, the pathophysiology involves no axonal injury but changes in axonal transport and focal demyelination, fitting the classic neuropraxic model where nerve conduction is blocked while the axon's architecture remains intact. (17)

This explains the diagnostic challenge: standard electrodiagnostic studies (EMG/NCS) have low sensitivity (~30%) as they often fail to capture the transient conduction block, especially if performed with the limb at rest. Consequently, diagnosis relies heavily on targeted muscle testing algorithms, which have demonstrated high diagnostic accuracy (88-93% sensitivity/specificity in blinded studies) by dynamically provoking the compression, thereby identifying the functional deficit consistent with a Sunderland Grade 0 lesion (18).

Clinical Picture of Proximal Median Nerve Compression

The clinical presentation is a constellation of sensory, motor, and provocative symptoms that localize the pathology to the proximal forearm.

Sensory Symptoms

Sensory disturbances are often the presenting complaint, but their pattern and provocation are key differentiators.

Paresthesia and Numbness: Patients report tingling, "pins and needles," or reduced sensation in the classic median nerve distribution: the palmar aspects of the thumb, index, middle, and radial half of the ring finger. A critical distinction from CTS is that the palmar cutaneous branch, which arises proximal to the carpal tunnel, may be involved. Therefore, patients may report numbness on the thenar eminence of the palm, a finding that definitively points to a compression site proximal to the wrist(19).

Pain: The quality and location of pain are highly indicative. Instead of localized wrist pain, patients describe a deep, aching, or tiredness-like pain in the proximal volar forearm, just distal to the elbow crease. This pain is often described as a "soreness" or "heavy fatigue" in the flexor muscle mass, exacerbated by sustained activity.(20).

Nocturnal Symptoms: Like CTS, nocturnal awakening with numbness can occur. However, a pivotal differentiating feature is the patient's response. In Lacertus Syndrome, shaking the hand (the "flick sign") typically provides little to no relief, as the compression is not at the wrist. Relief may instead come from extending the elbow and supinating the forearm, which relaxes the lacertus fibrosus (21).

Motor Symptoms and Signs

Motor deficits are a hallmark of proximal compressions and are the strongest clinical indicator that distinguishes them from isolated CTS. The involvement of branches to the forearm musculature is pathognomonic.

Weakness and Clumsiness: Patients may report a general loss of grip strength and dexterity. However, more specific is weakness in pinch grip between the thumb and index finger. This is due to the involvement of the Flexor Pollicis Longus (FPL) and the index finger Flexor Digitorum Profundus (FDP), muscles innervated by the anterior interosseous nerve (AIN) branch, which can be affected under the lacertus.

Pronator and Flexor Weakness: In more pronounced cases, weakness in forearm pronation and elbow flexion may be detectable, indicating compression of the main nerve trunk before it gives off its terminal branches.

Objective Signs: On examination, the "Pinch Sign" or "OK Sign" weakness is a key finding. The patient is unable to maintain a tight "OK" gesture (tip of thumb to tip of index finger) as the examiner pulls it apart, due to FPL and index FDP weakness. In chronic, severe cases, thenar muscle atrophy may be present, but its co-occurrence with proximal weakness is the critical clue (22).

Provocative Maneuvers

The diagnosis of Lacertus Syndrome is heavily reliant on specific physical exam tests that reproduce symptoms by dynamically increasing pressure at the suspected site.

Resisted Elbow Flexion and Pronation: This is the cardinal provocative test for Lacertus Syndrome. With the patient's elbow at 90-110 degrees of flexion and the forearm pronated or in a neutral position, the examiner resists active elbow flexion. This maneuver tenses the biceps tendon and the attached lacertus fibrosus, compressing the underlying median nerve against its sharp medial edge. A positive test reproduces or significantly worsens the patient's pain and/or paresthesia in the proximal volar forearm, often radiating into the median nerve distribution of the hand(21).

Direct Palpation (Tinel's Sign): Percussion or direct pressure over the lacertus fibrosus, located just medial to the distal biceps tendon in the antecubital fossa, may elicit a Tinel-like sign, producing paresthesia radiating into the median-innervated digits.(22).

Lacertus Syndrome as a Distinct Clinical Entity

Lacertus Syndrome has emerged from being a rarely diagnosed condition to a recognized cause of activity-related forearm pain, particularly in athletes and manual laborers. Its pathophysiology involves a Dynamic, mechanical compression where the tense lacertus fibrosus acts as a "band" constricting the median nerve during muscle contraction (23).

This explains why symptoms are often task-specific and not constant.

The clinical profile of Lacertus Syndrome can be summarized by a Diagnostic Triad:

1.Proximal Forearm Pain: Aching and fatigue localized to the volar proximal forearm.

2.Provocative Weakness: Documentable weakness in pinch grip and/or other median-innervated forearm functions.

3.Dynamic Provocation: Reproduction of symptoms with resisted elbow flexion/supination, without relief from the flick sign.

Failure to recognize this entity often leads to misdiagnosis as refractory CTS, resulting in unsuccessful carpal tunnel release surgery.

Clinical Provocative Tests for Median Nerve Compression

1. Sensoy Collapse Test (SCT)

The Sensory Collapse Test (SCT) is a relatively novel, dynamic clinical examination developed by Dr. Susan Mackinnon's group to localize sites of peripheral nerve compression. The test is based on the principle of a transient inhibition of motor function (a brief "collapse" of postural tone) following light tactile stimulation (scratching) of an irritated cutaneous nerve or its compression site. How it Works ?

It is performed with the patient's arms in a neutral position while the examiner applies resisted internal rotation; after lightly "scratching" the skin over the suspected compression site, the resistance test is immediately repeated. A positive test is defined as a brief loss of external rotation resistance ("collapse") on the affected side, indicating nerve compression.

The proposed mechanism is most strongly related to the cutaneous silent period, where a noxious stimulus transiently inhibits motor activity, although other theories such as substance P release have been suggested. Clinically, the SCT is valued for its ability to localize single or multiple compression sites along a nerve, making it useful in conditions such as lacertus syndrome and pronator syndrome.

However, its diagnostic accuracy is variable, with studies showing moderate specificity but low and inconsistent sensitivity, and only moderate interobserver reliability. The test is highly operator-dependent, requiring precise anatomical knowledge, correct force application, and patient cooperation, which contributes to variability in results. Therefore, SCT should be used as an adjunct rather than a standalone diagnostic tool, in combination with clinical examination and electrodiagnostic studies. (24).

Procedure:

1.The patient sits with arms adducted, elbows flexed to 90°, and forearms pronated.

2.The examiner applies isometric external rotation resistance to both forearms, asking the patient to maintain position against moderate, equal pressure.

3.Once a stable baseline resistance is established, the examiner lightly scratches the skin over the suspected nerve compression site (e.g., carpal tunnel, pronator teres, lacertus fibrosus) for 1-2 seconds.

4.The examiner immediately re-tests the resistance to external rotation on the affected side.

5.A positive test is defined as a brief, transient loss of resistance ("collapse") on the affected side only, immediately following the scratch stimulus.

Mechanism:

The SCT is believed to be a cutaneous-motor reflex. The scratch stimulus over the cutaneous field of an irritated sensory nerve generates an inhibitory spinal reflex loop, leading to a momentary inhibition of the corresponding motor neurons. This results in a transient weakness of the muscles responsible for maintaining external rotation (primarily infraspinatus, teres minor, and posterior deltoid), mediated via the spinal cord and supraspinal pathways. It is not a test of voluntary strength. (24)

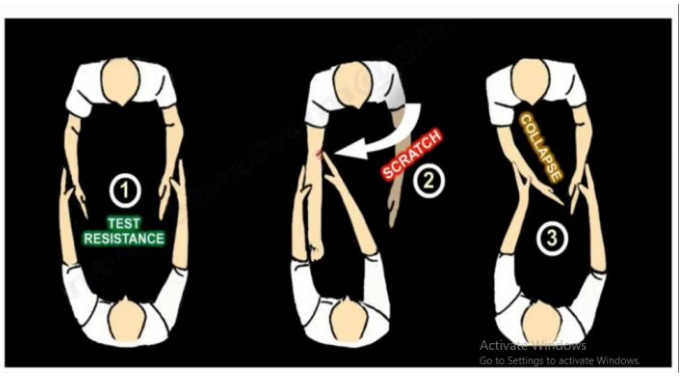

Figure 3: procedure for sensory collapse test

2. Tinel Test (Tinnel's Sign)

The Tinel test detects irritation or regeneration of a peripheral nerve. The examiner gently taps along the nerve from distal to proximal toward the suspected compression site. A positive result produces tingling or an electric shock-like sensation in the nerve's distal sensory distribution.

For median nerve assessment, the test is performed over the carpal tunnel for distal compression and over the pronator teres or lacertus fibrosus for proximal compression. It stimulates hypersensitive, demyelinated, or regenerating axons. In carpal tunnel syndrome, its sensitivity is 23–60% and specificity is 64–87%. Although less sensitive for proximal compression, a positive forearm test with a negative wrist test may help localize the lesion. Its main limitations are subjectivity and possible false-positive results following previous nerve injury or in polyneuropathy (25).

3. Phalen Test (Phalen's Maneuver)

The Phalen test provokes median nerve symptoms by increasing pressure within the carpal tunnel. The patient maximally flexes both wrists and holds the backs of the hands together for 30–60 seconds. Reproduction of tingling, numbness, or pain in the thumb, index, middle, or radial half of the ring finger indicates a positive test.

Wrist flexion reduces carpal tunnel volume, increases pressure beneath the flexor retinaculum, and temporarily compromises neural blood flow. Therefore, the test mainly identifies distal median nerve compression and is usually negative in isolated proximal neuropathy. Its reported sensitivity is 68–85%, with a specificity of 73–89%. A reverse Phalen test using wrist extension may also be performed. Limitations include discomfort and false results in severe or chronic cases (25).

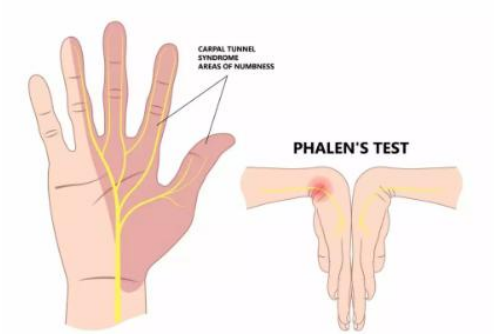

Figure 4: Phalen test for clinical evaluation of median nerve compression

Investigations

1. Nerve Conduction Studies (NCS) and Electromyography (EMG)

Nerve conduction studies (NCS) and electromyography (EMG) are cornerstone tests for nerve integrity, but they face a critical challenge in diagnosing Lacertus Syndrome. This condition involves dynamic, posture-dependent compression of the median nerve under the bicipital aponeurosis during elbow flexion and pronation. Standard NCS/EMG protocols are performed with the limb at rest, failing to replicate the transient mechanical stress that causes symptoms. Consequently, results are often normal, leading to a very low sensitivity (estimated below 50%). (26)

Why the Tests Fall Short ?

The issue is one of physiology versus mechanics. NCS/EMG excel at detecting persistent demyelination or axonal loss but are poorly suited for intermittent, functional compression that causes ischemia without permanent nerve damage. While specialized "dynamic NCS" attempts to record signals during provocative positioning, these methods lack standardization and are not widely adopted. EMG may reveal subtle changes in muscles like the pronator teres, but these findings are inconsistent and non-specific, as they can also occur in other proximal neuropathies. (26,25)

A normal NCS/EMG cannot rule out Lacertus Syndrome, and abnormal findings are not definitive proof. This low diagnostic yield underscores that NCS/EMG provides a static snapshot of nerve physiology, not an assessment of dynamic mechanical compromise. . NCS/EMG remain essential for ruling out conditions like carpal tunnel syndrome or radiculopathy, their role in confirming Lacertus Syndrome is minimal.(27)

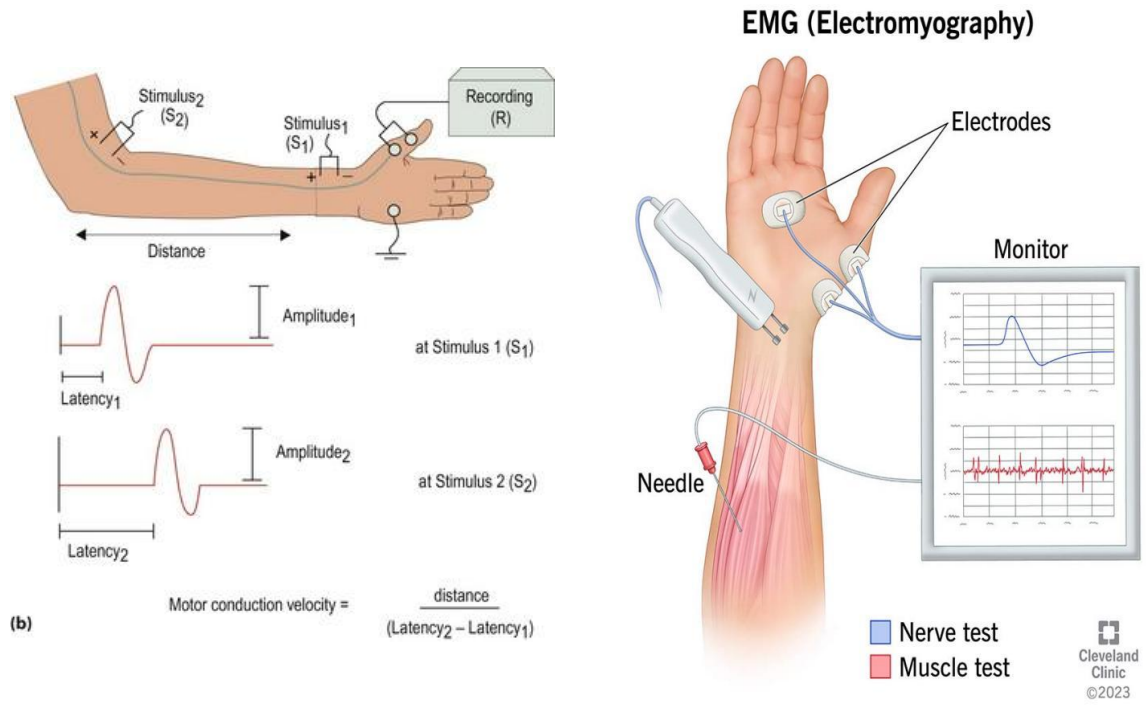

Figure 5: Electromyography (EMG) and Nerve Conduction Studies (NCS) in Median Nerve Compression

2.Dynamic Ultrasound: The Diagnostic Key for Proximal Median Nerve Compression

Technical Core & Application

Dynamic ultrasound utilizes high-frequency transducers (12-18 MHz) to provide real-time visualization of the median nerve during active, symptom-provoking movements like pronation, supination, and resisted elbow flexion. This allows direct assessment of nerve mobility, focal compression, and mechanical interactions with adjacent structures—capturing the intermittent pathology that resting studies miss. Key diagnostic measurements include increased Cross-Sectional Area (CSA), abnormal flattening ratio, and reduced nerve displacement. (28).

Definitive Findings for Specific Syndromes

For Lacertus Syndrome, the pathognomonic sign is the sudden flattening or "snapping" of the median nerve under the taut bicipital aponeurosis during resisted elbow flexion and supination. In Pronator Teres Syndrome, compression is visualized between the pronator heads during resisted pronation. Diagnostic criteria often include a CSA >12-15 mm² at the compression site and a flattening ratio >3:1 during the provocative maneuver. (29)

Superiority and Limitations

Dynamic ultrasound's primary advantage is its functional, real-time assessment, making it the most sensitive tool for diagnosing dynamic entrapments. It surpasses electrodiagnostic studies for these conditions by providing anatomical visualization of the compression event. However, its accuracy is highly operator-dependent, lacks universal standardized criteria, and can be technically limited in larger patients. (28,29)

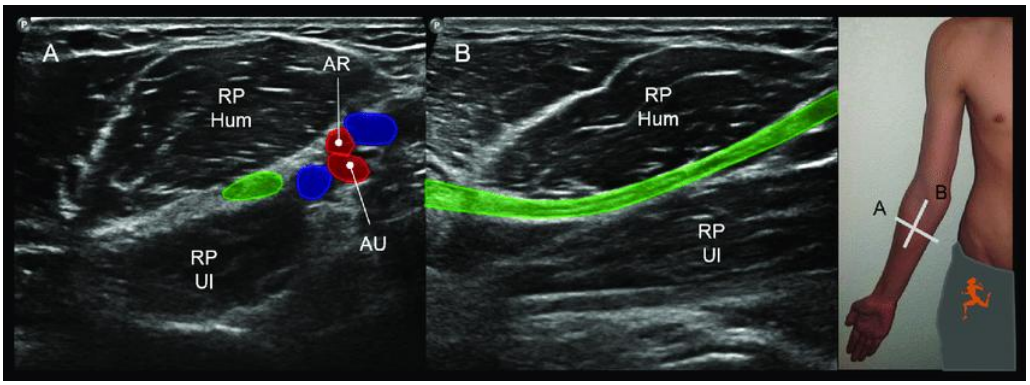

Figure 6: US depicting the median nerve at the level of the pronator teres muscle: The median nerve (in green) courses between the humeral (RP Hum) and the ulnar (RP Ul) heads of the pronator teres muscle, beneath the division of the brachial artery into its ulnar (AU) and radial (AR) branches.

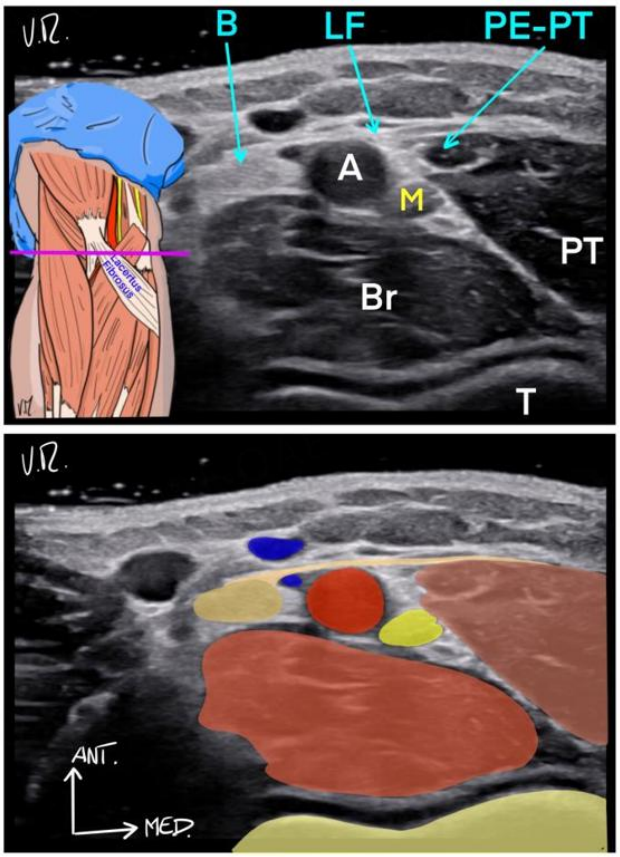

Figure 7: . Transverse Ultrasonographic view of the anterior aspect of the Elbow. A: Humeral Artery; B: Biceps tendon; Br: Brachialis; LF: Lacertus Fibrosus; M: Median Nerve; PT: Pronator Teres; PE-PT: Proximal Edge of Pronator Teres; T: humeral Trochlea.

3.Magnetic Resonance Imaging (MRI)

Magnetic Resonance Imaging (MRI) provides critical visualization of the median nerve's relationship to the bicipital aponeurosis (lacertus fibrosus) at the elbow. Unlike dynamic ultrasound, MRI offers a static but highly detailed view of the nerve and surrounding structures, clearly identifying focal nerve flattening, swelling, or signal changes (increased T2 signal) directly beneath the thickened or taut lacertus fibrosus. It is particularly valuable for detecting secondary signs of chronic compression, such as denervation edema or fatty infiltration in the pronator teres and flexor carpi radialis muscles, which helps confirm the diagnosis and assess severity. MRI

excels at ruling out structural mimics like soft-tissue masses, anomalous muscles, or concurrent pathology in the same region. While it cannot capture the dynamic compression during movement like ultrasound can, MRI provides essential anatomical confirmation and is crucial for preoperative planning when surgical release of the lacertus fibrosus is considered.(30)

The Clinical Triad: A Structured Approach to Diagnosing Peripheral Nerve Compressions

Nerve compression syndromes, characterized by the mechanical and often dynamic compression of peripheral nerves, are a significant yet frequently under appreciated cause of pain, muscle weakness, and functional impairment. These conditions often disrupt daily activities and diminish quality of life. Traditional diagnostic methods, including electromyography (EMG) and imaging techniques, have limitations, particularly in detecting nerve compressions in their early stages. Electrodiagnostic studies (EDX) are commonly employed to evaluate suspected nerve entrapments, particularly in the upper extremities. However, their sensitivity and specificity, especially for conditions beyond carpal and cubital tunnel syndromes, are limited, typically ranging between 30 and 65% . EDX may also struggle to detect mixed nerve injuries and is unable to provide comprehensive muscle function assessments or identify early-stage compressions. Therefore, clinical examination techniques should be integrated with or prioritized over sole reliance on EDX. (3)

The clinical triad of nerve compressions

In the early stages of nerve compression, dynamic pressure on the nerve can disrupt saltatory conduction and lead to dynamic nerve ischaemia. Mackinnon and colleagues coined the term "Sunderland Zero" to describe this level of nerve impairment, characterized by dynamic nerve compression without axonal loss .

These compressions typically affect superficial nerve fascicles at the site of pressure, resulting in sensory or motor deficits—or both—depending on the nerve segment affected.

To diagnose nerve compressions effectively, clinical evaluations must assess both motor and sensory function while identifying pain at the compression site. A clinical triad for a structured assessment of median nerve compressions was first described in 2013 , and has since been described in upper extremity nerve compression syndromes . (4).

The Triad includes:

- Manual muscle testing: From proximal to distal, to identify the level of compression.
- Sensory provocative testing: Using methods such as the sensory-collapse test (SCT) to confirm compression levels.
- Pain testing: Evaluating for allodynia at the site of compression to further verify the diagnosis.

Muscle testing algorithm

The principles of manual muscle testing (MMT) were first described by Sir Herbert Seddon in 1954. MMT enables clinicians to identify specific patterns of weakness, aiding in localizing the level of nerve compression. Muscle strength is graded on a scale from M0 (no muscle power) to M5 (full strength against maximum resistance), with particular attention to the M4 grade:

• M4-: Power against slight resistance.

• M4: Power against moderate resistance.

• M4+: Strong power but not at maximum resistance.

Clinicians must actively evaluate for M4 weaknesses, which can provide critical diagnostic information about nerve compressions.

Strengths and limitations of MMT

The muscle testing algorithm has several strengths, includ- ing proven validity, high reliability, and sensitivity, as well as optimal accessibility, requiring only the clinician and Tinel's and Phalen’s flexion tests.

In a systematic review of the sensitivity and specificity for diagnosis of nerve compressions syndromes, the general sensitivity is rela- tively low (38%), whereas the specificity is very high (94%). The conclusion of this is that SCT is a useful confirmatory tool that is most effective when used along- side other diagnostic methods, such as MMT, but is not recommended to be used as a stand-alone tool for nerve compression diagnosis (31).

The correlation between SCT and EDX was recently investigated in patients with carpal tunnel syndrome (CTS), where a positive SCT was correlated with positive EDX findings, in particular in cases if decreased median nerve conduction velocity and amplitudes, further supporting it's use in diagnosis of nerve compressions.

The SCT has been proposed to be renamed the "sensory" collapse test instead of the "scratch" collapse test, as any sensory irritant can be used to provoke areas of allodynia. The physiology underlying the SCT is characterized by dis- tinct reflex pathways that purposefully facilitate a protective response to external noxious stimuli

. In a pilot study investigating the utility of an "SCT-EMG," a temporary loss of muscle tone, as measured by EMG, was seen fol- lowing cutaneous stimulation of a compressed nerve . Notably, this effect was absent after successful surgical decompression.(32)

The SCT is performed by having the patient resist applied force before and after the examiner scratches/ touches the skin over the suspected nerve compression site. A focal nerve compression results in a temporary loss of muscle strength and is called a "positive SCT" . Cold-spray (ethyl chloride) may also be used to confirm the site of compression by temporarily resolv- ing the allodynia, resulting in a negative SCT (32)

Clinical triad – median nerve compressions

Table 1 Median Nerve Compression Syndromes and the Clinical Triad

MEDIAN NECRVE TRIADS	Motor Weakness	SCT	Pain (compression site)
Lacertus Syndrome	FCR FPL FDP to index	Over the lacertus fibrosus	At the proximal edge of the lacertus fibrosus
Superficialis Syndrome	FDS to middle ring fingers	5–7 cm distal of the elbow crease, over the superficialis arcade	At the superficialis arcade
Anterior Interosseous Syndrome	FPL FDP to index PQ	Mid-forearm level, 2–3 cm distal of the superficialis arcade	Volar mid-forearm
Carpal Tunnel Syndrome	APB	At the level of the carpal tunnel	Wrist flexion crease (pos Tinels test)

Abbreviatons: FCR flexor carpi radialis, *FPL* flexor pollicis longus, *FDP* flexor digitorum profundus, *PQ* pronator quadratus, *APB* abductor pollicis longus

Figure 8: Table for median nerve compression syndromes and the clinical triad

Lacertus syndrome

Far more common are the compressions of the median nerve (MN) in the proximal forearm, collectively called proximal median nerve entrapments (PMNE). Recent publications have shown that one of the primary compression points is at the level of the lacertus fibrosus (LF), in what is known as the lacertus syndrome

Muscle testing: Dynamic or static compression of the nerve at this level results in weakness in the FCR, FPL, FDP2.

Sensory testing: Testing is directed to the area from the ulnovolar antecubital fossa to the LF

Pain: Pain will be present at the level of the LF. (33,34)

Anterior interosseous nerve syndrome

Rarely seen, the anterior interosseous nerve syndrome entails a compression of the AIN after the bifurcation from the median nerve .

Muscle testing: As with the lacertus syndrome, weak- ness will be seen in the FPL, FDP II but not in the FCR. Pronator quadratus weakness may be present, but is often difficult to isolate and test due to strength in the pronator teres.

SCT: Testing is directed to the mid-forearm level

Pain: Positive about 2–3 cm distal of the superficialis arcade, mid-forearm (6)

Carpal tunnel syndrome (CTS)

CTS is by far the most commonly diagnosed and described nerve compression. As such, there are a myriad of clinical examination tests for this diagnosis, in addition to nerve conduction tests .

For this article, only the findings in line with the clinical triad are described.

Muscle testing: Thenar atrophy may be seen in advanced CTS. As most patients present with earlier stages of nerve compression, a subtle weakness in the APB is most commonly found.

SCT: A positive test will be elicited at the proximal edge of the carpal tunnel.

Pain: CTS is only sometimes associated with pain on compression. Rather, the triad is supported by perform- ing Tinel's test, where percussion of the median nerve at the carpal tunnel will result in paresthesias into the median innervated area of the hand. (35)

Double-crush and multiple-crush syndromes

The examination of any patient presenting with pain, numbness, and/or weakness must include consideration of more than one concurrent nerve compression. The double-crush syndrome refers to the presence of two or more compressive lesions along the same nerve pathway. One of the most frequent combinations is concomitant carpal tunnel syndrome and lacertus syndrome at the elbow. (36)

The related concept of multiple-crush syndromes is commonly observed in patients with underlying systemic conditions such as diabetes mellitus or following trauma. A recent study underscored this by investigating the co-occurrence of carpal tunnel syndrome (CTS) and fall injuries linked to common peroneal nerve (CPN) compression. This suggests that patients diagnosed with CTS should also be screened for possible CPN entrapment, as early identification provides a straightforward, treatable means to prevent future falls.(37)

Therefore, clinicians must be systematic, employing a consistent clinical triad and conducting examinations from proximal to distal in all patients with neurological complaints. This approach is critical because compressive neuropathies can exist at multiple levels, potentially affecting both the upper and lower extremities simultaneously.(36,37)

Treatment Proximal Median Nerve Compression

1. Conservative (Non-Surgical) Management

Conservative treatment is the first-line approach for recent or mild-to-moderate median nerve compression (14,38).

1.Rest and Activity Modification

Patients should avoid repetitive pronation, forceful gripping, and sustained elbow flexion. Maintaining a neutral forearm and improving workplace ergonomics may relieve symptoms in 30–40% of cases within 4–6 weeks (14,39).

2.Splinting and Immobilization

Forearm-based splints limit elbow and forearm movements that provoke proximal compression. Night splinting is particularly useful, while neutral wrist splints are preferred for CTS. Combined with activity modification, splinting may achieve 50–60% clinical improvement (38,40).

3.Physical Therapy

A structured rehabilitation program may include:

Manual therapy: Soft-tissue mobilization of the pronator teres, lacertus fibrosus, and FDS arch (41).

Median nerve gliding: Controlled exercises performed for 6–8 weeks may improve symptoms in 45–55% of cases (42).

Strengthening: Gradual strengthening of the scapular and shoulder stabilizers helps reduce abnormal neural tension (41,42)

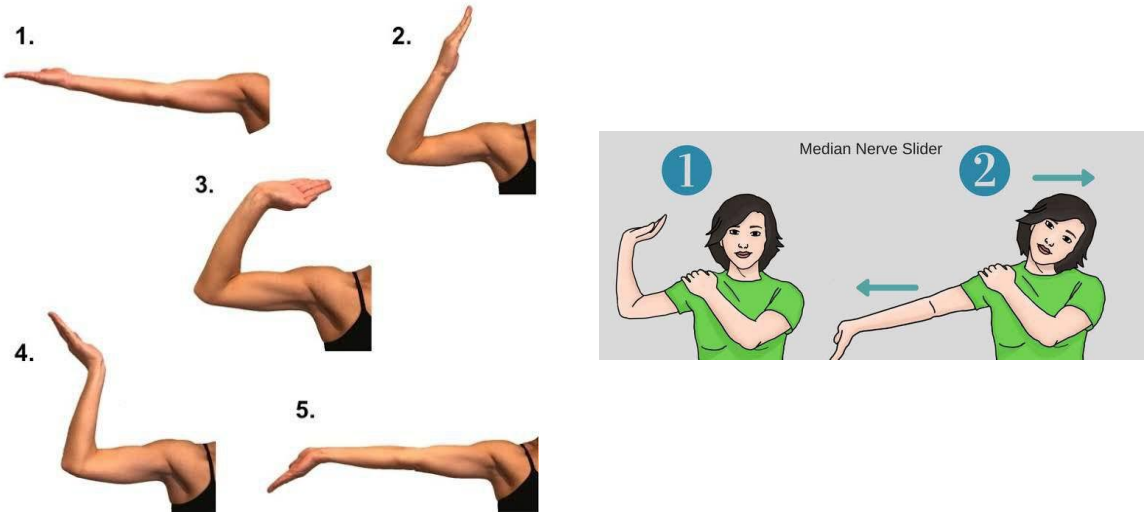

Figure 9: median nerve gliding exercise as conservative trial for decompression

Pharmacological Management

Medication is selected according to the predominant symptoms:

NSAIDs: Short courses of 2–4 weeks may reduce inflammation and pain (43).

Corticosteroid injections: Ultrasound-guided injection at the compression site may provide 70–80% short-term symptom relief (44).

Neuropathic medications: Gabapentin or pregabalin may be considered for predominant neuropathic pain (45).

Other Conservative Modalities

Kinesio Taping in Median Nerve Compression

Kinesio taping aims to reduce pressure, improve nerve gliding, regulate muscle tone, and decrease edema. Its proposed effects include fascial lifting, proprioceptive stimulation, improved lymphatic drainage, and sensory modulation (46).

Application varies according to the compression site:

Pronator teres syndrome: A strip with 15–25% tension is applied along the pronator teres to reduce muscle hypertonicity.

Lacertus syndrome: A Y-strip with 50–75% tension is applied over the lacertus fibrosus to lift the superficial fascia (46).

Carpal tunnel syndrome: An I-strip with 10–15% tension is applied along the median nerve pathway to facilitate nerve excursion (46).

Figure 10: example for a strip of kineso taping stretch for decmprssing median nerve under lacertus fibrosus band

Summary of Conservative Treatment Efficacy

When applied consistently over a minimum of 3-6 months, a comprehensive conservative regimen can yield symptomatic improvement in 29–70% of patients with proximal median nerve compression. (14,38,41)

2.Surgical Interventions & Management

2.1 Preoperative Considerations

Surgery is considered after 3–6 months of structured conservative treatment or earlier in patients with severe symptoms or progressive neurological deficits. Diagnosis and lesion localization should be supported by clinical examination, EMG/NCS, ultrasound, or MRI when appropriate. Surgical planning must also assess multiple compression sites, including double-crush syndrome, reported in 15–20% of cases (36,16).

2.2 Lacertus Syndrome Release

1. Indications

· Clinical evidence of proximal median nerve compression at the elbow.

· Weakness of the FPL, FDP II, and FCR.

· Positive lacertus compression test.

· Failure of conservative treatment for 6–12 weeks.

· Persistent symptoms following distal decompression, such as carpal tunnel release (47,48).

Anatomy & Goal: Release of the constricting bicipital aponeurosis (lacertus fibrosus) compressing the median nerve.

Technique: A 3-4 cm longitudinal incision is made distal to the elbow crease. The medial antebrachial cutaneous nerve is protected. The lacertus fibrosus is identified and completely divided longitudinally, with potential additional release of the deep flexor-pronator fascia if thickened. (47)

Outcomes: High success rate of 85-90%, with rapid recovery of strength typically within 4-6 weeks. Complications are rare but include nerve branch injury (2-3%) and hematoma.(47,49)

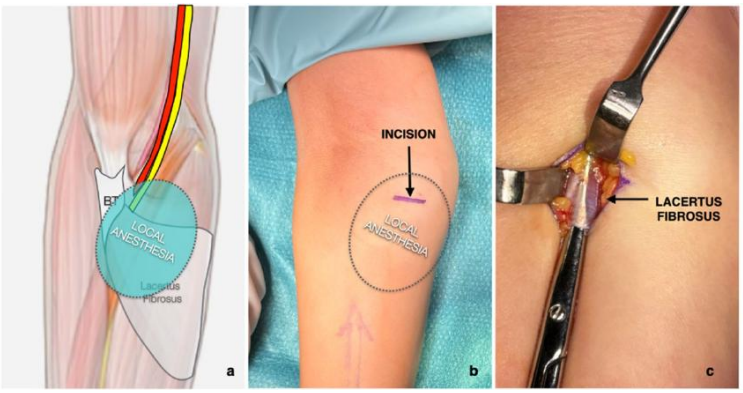

Figure 11: Illustration of the minimally invasive surgical technique for lacertus release.

2.3 Pronator Teres Syndrome Release

Anatomy & Goal: Decompression of the median nerve at its four potential proximal entrapment sites: the ligament of Struthers, bicipital aponeurosis, between the heads of the pronator teres, and beneath the flexor digitorum superficialis arch.

Technique: An extended Henry approach (Lazy S)via a 6-8 cm curvilinear incision allows systematic exploration. Releases typically include division of the lacertus fibrosus, partial or complete release of the superficial pronator teres head, and division of the flexor digitorum superficialis arch. (50,51)

Outcomes: 80-85% of patients achieve good-to-excellent results. Pronator function is preserved with partial releases. Return to light duties occurs at 6-8 weeks, with heavy labor resuming around 12 weeks. (50)

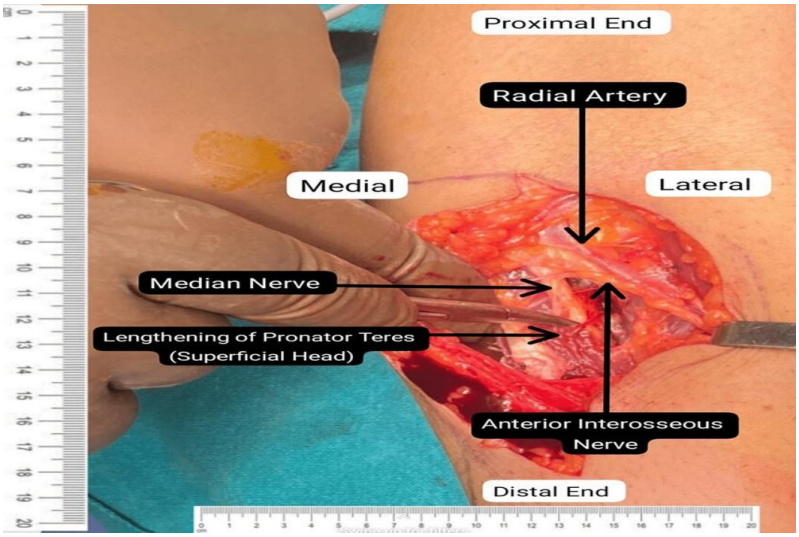

Figure 12: Exploration after lazy 'S' shaped incision over the ventral aspect of the left forearm followed by lengthening of the superficial head of the PT muscle. PT: pronator teres.

2.4 Anterior Interosseous Nerve (AIN) Syndrome Release

Considerations: This pure motor syndrome often requires meticulous exploration due to variable anatomy.

Technique: Dissection proceeds deep through the pronator teres to identify and decompress the AIN. Common compression sources released include accessory heads of the flexor pollicis longus (Gantzer's muscle), thrombosed vessels, and fibrous bands. (9)

Outcomes: Recovery is highly time-dependent. Surgery within 6 months yields ~90% motor recovery, while delayed intervention beyond 12 months reduces recovery rates to 50-60%. (9)

2.5 Combined Procedures with Carpal Tunnel Release

In the 20-30% of patients with coexisting proximal and distal compression, a combined approach may be indicated. Strategy (single-stage vs. staged) is based on symptom dominance. Endoscopic carpal tunnel release can often be effectively combined with an open proximal decompression. (36,16)

3. Postoperative Rehabilitation

Immediate Phase (0-2 weeks): Focus on edema control, wound care, and initiating finger motion.

Intermediate Phase (2-6 weeks): Incorporates gradual range-of-motion exercises, scar management, and gentle neural gliding.

Advanced Phase (6-12 weeks): Progressive strengthening and work-specific conditioning to facilitate a full return to activity. (52)

4. Evidence-Based Outcomes and Prognostic Factors

Success Rates: Conservative management succeeds in 50-70% of mild-to-moderate cases. Surgical success ranges from 80-95%, varying by specific syndrome.

Prognostic Factors: Poorer outcomes are associated with prolonged symptom duration, advanced age, comorbidities (e.g., diabetes), and smoking. (53)

Complications (3-8%): Include neurological injury (2-3%), hematoma (1-2%), infection (<1%), and symptom recurrence (5-10%). Management involves early recognition and possible revision surgery for persistent symptoms after 6 months. (47,50)

5. Emerging Techniques and Future Directions

Innovations focus on minimizing invasiveness and improving outcomes. These include the development of endoscopic and ultrasound-guided percutaneous release techniques, intraoperative nerve mapping, and biological strategies like anti-adhesive nerve wraps and adjunctive platelet-rich plasma (PRP) applications to reduce perineural fibrosis. (54,55)

Complications of Longstanding Untreated Proximal Median Nerve Compression

1.Progressive Neuropathy Untreated proximal median nerve compression may progress from intermittent pain and paresthesia to persistent neuropathic symptoms associated with functional limitation. (56,57)

2.Motor Dysfunction Chronic compression can lead to weakness of the forearm flexors, reduced grip and pinch strength, and impaired hand dexterity. (56,57)

3.Sensory Deficits Persistent nerve entrapment may result in diminished sensation and impaired proprioception, negatively affecting fine motor performance. (56)

4.Nerve Degeneration Prolonged compression causes ischemia, demyelination, disruption of axonal transport, and eventual axonal loss, reducing the potential for neurological recovery. (57,58)

5.Muscle Atrophy Advanced cases may develop irreversible atrophy of median nerve-innervated muscles due to prolonged denervation. (56,58)

6.Double Crush Syndrome Proximal compression may increase susceptibility to distal median nerve entrapment, particularly carpal tunnel syndrome, contributing to persistent symptoms after isolated distal decompression. (56,59)

7.Poor Prognosis Delayed diagnosis and treatment are associated with poorer functional outcomes and reduced recovery potential. (56,57)

Complications of Lacertus and Proximal Median Nerve Release

1.Iatrogenic Nerve Injury Surgical release may occasionally injure adjacent sensory nerves, resulting in dysesthesia, numbness, or neuroma formation. (60,61)

2.Incomplete Decompression Failure to identify and release all compression sites may lead to persistent or recurrent symptoms following surgery. (60,62)

3.Perineural Fibrosis Scar tissue formation around the nerve may cause recurrent entrapment and ongoing discomfort. (61,62)

4.Postoperative Complications Minor complications such as pain, hematoma, infection, and wound-related problems may occur after surgery. (61,62)

5.Diagnostic Error Failure to recognize alternative diagnoses or associated double crush syndrome may result in unsatisfactory surgical outcomes. (60)

6.Residual Weakness Patients with longstanding compression and preoperative axonal damage may continue to experience weakness despite successful decompression. (61,62)

Overall Outcomes When appropriate patient selection and complete decompression are achieved, proximal median nerve release generally provides excellent symptom relief and high patient satisfaction. (63)

Conclusion

Clinical Implications Diagnosis requires dynamic provocative testing rather than standard nerve conduction studies. Treatment must address motion-induced constrictors through targeted surgical releases, emphasizing the need for tailored approaches distinct from those used for static compression . Overall Outcomes When appropriate patient selection and complete decompression are achieved, proximal median nerve release generally provides excellent symptom relief and high patient satisfaction.

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