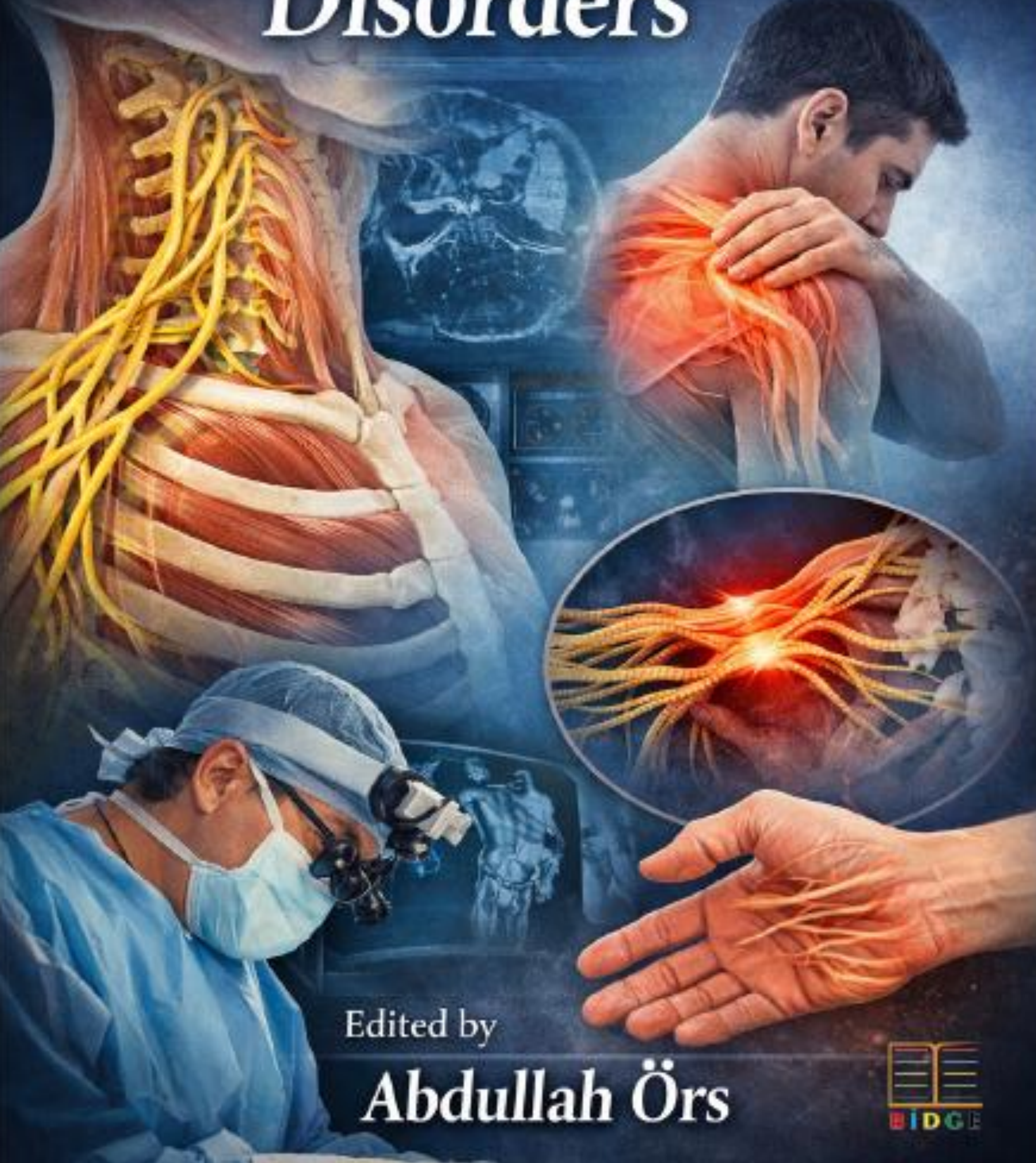
Anatomical and Clinical Perspectives on

Plexus Brachialis Disorders



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Anatomical and Clinical Perspectives on Plexus Brachialis Disorders

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CHAPTER 1

BRACHIAL PLEXUS: ANATOMICAL ORGANIZATION AND CLINICAL CORRELATION

TUNCAY ÇOLAK[1]

1. Introduction

The brachial plexus represents the most complex peripheral nerve network responsible for the motor and sensory innervation of the upper extremity, extending from the cervical spine to the axilla and encompassing a broad anatomical and functional territory. This structure is not merely an anatomical convergence of nerve fibers; rather, it constitutes a dynamic organization shaped by embryological development, topographic relationships, and biomechanical factors. Consequently, disorders of the brachial

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plexus cannot be confined to a single anatomical level and must be considered within a wide clinical spectrum.

In clinical practice, pathologies related to the brachial plexus comprise a heterogeneous group ranging from congenital lesions and traumatic injuries to compression syndromes and distal entrapment neuropathies. This diversity necessitates a thorough understanding of the plexus's segmental organization and anatomical transition zones. The difference in clinical outcomes between a proximal root lesion and a distal terminal nerve entrapment arises not only from the specific nerve fibers involved but also from the functional architecture of the plexus itself.

The aim of this chapter is not to reiterate a detailed anatomical description of the brachial plexus, but rather to elucidate its segmental organization and clinical significance. In doing so, conditions discussed in subsequent sections, such as Erb–Duchenne palsy, thoracic outlet syndrome, and terminal nerve entrapments, can be evaluated within a unified anatomical–clinical framework.

Segmental Organization of the Brachial Plexus

The brachial plexus is classically organized in a hierarchical manner consisting of roots (radices), trunks, divisions, cords (fasciculi), and terminal branches. This segmental structure represents not merely an anatomical classification, but a fundamental principle that enables localization of lesions and prediction of functional deficits during clinical evaluation.

Roots and Trunks: Supraclavicular Organization

The brachial plexus is formed by the anterior rami of the C5–T1 spinal nerve roots. After a short cervical course, these roots unite to form the upper, middle, and lower trunks. This stage represents the supraclavicular portion of the plexus and constitutes the most proximal region in which clinically significant lesions may occur.

Lesions at this level typically affect multiple muscle groups and extensive sensory territories. Because segmental differentiation has not yet occurred, injuries in this region produce clinical patterns characterized by functional chain disruption rather than isolated single-nerve deficits. This phenomenon is particularly evident in congenital lesions and high-energy traumatic injuries.

1.2 Divisions and Cords: Infraclavicular Transition

As the trunks divide into anterior and posterior divisions, the brachial plexus enters the infraclavicular region. At this stage, nerve fibers undergo not only segmental but also functional reorganization. Anterior divisions convey fibers destined for flexor compartments, whereas posterior divisions supply extensor compartments.

The divisions recombine to form the lateral, medial, and posterior cords, which are named according to their topographic relationship with the axillary artery. This relationship highlights the close anatomical association between the brachial plexus and vascular structures, underscoring the clinical relevance of vascular proximity. Lesions at this level are often position-dependent, variable, and at times reversible.

Terminal Branches: Distal Functional Differentiation

The terminal branches of the brachial plexus, musculocutaneous, median, ulnar, radial, and axillary nerves, represent the most distal and functionally specific level of organization. At this stage, nerve fibers are directed toward well-defined motor and sensory territories. Accordingly, terminal nerve lesions present with more localized, predictable, and clinically distinct patterns.

Unlike proximal plexus lesions, pathologies at the terminal nerve level produce symptoms confined to specific muscle groups and limited sensory distributions, allowing distal entrapment neuropathies to be more clearly delineated clinically.

Clinical Significance of Segmental Organization

The segmental organization of the brachial plexus forms the conceptual basis of lesion-level assessment in clinical practice. As one progresses from proximal to distal levels, clinical presentations evolve from widespread and complex patterns to more localized and specific deficits. This principle serves as a cornerstone in the classification and differential diagnosis of brachial plexus disorders.

Accordingly, the brachial plexus should be regarded not merely as an anatomical nerve network, but as a functional system that generates distinct clinical patterns directly related to the level of injury.

2. Topographic Anatomy: Proximal-to-Distal Transition

The clinical behavior of the brachial plexus depends not only on its segmental organization but also on its topographic course from the cervical region to the axilla. Along this trajectory, nerve fibers maintain close relationships with various osseous, muscular, and vascular structures, which play a decisive role in determining lesion localization and clinical characteristics. Thus, the brachial plexus should be considered a dynamic system exposed to different risks at distinct anatomical transition zones.

Cervical Region

In the cervical region, the brachial plexus courses lateral to the cervical vertebrae, between deep neck muscles and fascial planes. At this level, the nerve roots lie in close proximity to the transverse processes and adjacent muscular structures. Movements of the cervical spine, particularly lateral flexion and rotation, may generate traction forces on the nerve roots.

Lesions in the cervical region typically produce widespread motor and sensory deficits. Because functional differentiation of fibers has not yet occurred, clinical presentations at this level often suggest central or plexal involvement rather than peripheral nerve entrapment.

Thoracic Outlet Region

As the brachial plexus proceeds distally, it enters the thoracic outlet, a critical anatomical transition zone between the neck and upper extremity. In this region, nerves and vessels traverse narrow, confined, and position-sensitive spaces, bordered by osseous structures, muscles, and fibrous bands.

The defining feature of the thoracic outlet region is the susceptibility of the brachial plexus to dynamic compression. Upper extremity positioning, shoulder girdle configuration, and postural factors directly influence mechanical stress on neural structures. Consequently, lesions in this region often manifest with position-dependent, intermittent, and clinically variable symptoms.

This zone functions as an intermediate station between proximal root lesions and distal terminal nerve entrapments, explaining the coexistence of both widespread and partially localized clinical findings.

Axillary Region

Beyond the thoracic outlet, the brachial plexus enters the axilla, a relatively protected yet anatomically dense region. Here, the nerves maintain close proximity to the axillary artery and begin their final differentiation into terminal branches. This level represents a stage of increasing functional specificity.

Lesions in the axillary region typically affect specific cords or terminal branches, producing more definable motor and sensory deficits. Nevertheless, the axilla remains vulnerable to traumatic injuries and iatrogenic damage during surgical interventions.

Distal Course and Terminal Nerves

Distal to the axilla, the brachial plexus gives rise to terminal nerves that supply distinct compartments of the upper extremity. At this stage, nerve fibers are associated with well-defined motor functions and limited sensory territories, resulting in clinically discrete presentations.

Distal entrapment neuropathies generally involve more static and localized compression mechanisms compared with proximal plexus lesions. However, accurate assessment requires exclusion of proximal plexal pathology, as distal symptoms may represent secondary manifestations of more proximal involvement.

Clinical Relevance of Topographic Anatomy

The proximal-to-distal topographic course of the brachial plexus underpins localization-based clinical reasoning. As evaluation progresses distally, clinical patterns shift from diffuse and complex to localized and predictable. This transition is critical for differential diagnosis and treatment planning.

Accordingly, the brachial plexus should be regarded not only as an anatomical nerve network but as a topography-sensitive system with distinct clinical vulnerabilities at each anatomical level.

3. Clinical–Anatomical Correlation

Accurate clinical assessment of brachial plexus disorders relies on establishing a precise correlation between anatomical organization and clinical manifestations. The segmental and topographic distribution of nerve fibers within the plexus gives rise to distinct motor and sensory patterns depending on lesion location. Therefore, clinical presentations should be interpreted not solely by the name of the affected nerve, but by the anatomical level of the lesion.

Proximal Lesions: Root and Trunk Level

Lesions at the root and trunk levels typically present with extensive and complex clinical patterns. Because nerve fibers have not yet differentiated into terminal branches, multiple muscle groups and broad sensory territories are affected simultaneously. Clinical findings rarely conform to the distribution of a single peripheral nerve.

Upper trunk involvement predominantly affects shoulder abduction, external rotation, and elbow flexion, whereas lower trunk lesions preferentially involve intrinsic hand muscles and ulnar-distributed sensory territories. These patterns are characterized by widespread motor chain dysfunction and may mimic cervical spine pathology.

Transitional Plexus Lesions

The supraclavicular and infraclavicular transition zones of the brachial plexus are anatomically narrow and dynamic, giving rise to unique clinical presentations. Lesions at these levels often produce variable and position-dependent symptoms.

Compression in these regions may result in the coexistence of both widespread and partially localized findings. Motor and sensory symptoms are frequently intermittent and fluctuate with upper extremity positioning, contributing to diagnostic complexity.

Distal Plexus and Terminal Nerve Lesions

Distal lesions involving the cords or terminal nerves generate more predictable and clearly defined clinical patterns. Because nerve fibers at this level are directed toward specific motor and sensory territories, deficits typically correspond to a single nerve distribution.

Terminal nerve entrapments are characterized by localized weakness and well-demarcated sensory loss, making them more amenable to confirmation through clinical examination and ancillary diagnostic studies. Nevertheless, potential coexisting proximal plexus pathology must always be considered.

Importance of Differential Diagnosis in Clinical Correlation

Establishing accurate clinical–anatomical correlation is essential for differential diagnosis in brachial plexus disorders. Cervical radiculopathies, plexus lesions, and distal entrapment neuropathies may present with overlapping symptoms. However, combined evaluation of motor patterns, sensory distribution, and reflex changes facilitates localization of the lesion.

Clinical correlation thus depends not only on anatomical knowledge but also on functional assessment and systematic examination. The clinical behavior of the brachial plexus should be viewed as a direct reflection of its anatomical organization.

General Principles of the Clinical–Anatomical Approach

Determining whether a lesion affects a single nerve or an entire neural chain is critical in approaching brachial plexus disorders. Widespread motor and sensory deficits suggest proximal involvement, whereas localized and sharply defined deficits indicate distal nerve pathology. This principle guides both diagnostic reasoning and therapeutic planning.

4. Clinical Clues in Proximal versus Distal Lesions

Correct diagnosis of brachial plexus pathology hinges on distinguishing between proximal and distal lesions. Differences in the distribution, severity, and progression of clinical findings provide essential diagnostic clues.

Motor Patterns

Proximal lesions produce widespread motor deficits involving multiple muscle groups and disrupting coordinated functional movements of the shoulder, elbow, and hand. In contrast, distal lesions cause selective weakness confined to muscles innervated by a specific terminal nerve.

Sensory Distribution

Proximal plexus lesions typically result in broad, poorly demarcated sensory disturbances involving dermatomal or segmental regions. Distal entrapment neuropathies, by contrast, produce sharply defined sensory deficits corresponding to a single peripheral nerve territory.

Reflex Changes

Reflex examination is particularly valuable in assessing proximal lesions. Involvement of proximal plexus segments may lead to diminished or absent reflexes associated with specific spinal levels, a finding rarely seen in distal nerve entrapments.

Positional and Mechanical Factors

Proximal and plexus-level lesions frequently exhibit position-sensitive symptoms influenced by neck movements and shoulder girdle configuration, suggesting dynamic compression mechanisms. Distal lesions tend to be more static, with symptoms less affected by cervical or shoulder positioning.

Clinical Course and Progression

Proximal lesions often follow a fluctuating and variable course with intermittent symptoms and functional adaptation. Distal entrapment neuropathies usually demonstrate a more predictable and progressive pattern. These distinctions are relevant for both diagnosis and treatment planning.

Integrated Clinical Assessment

Reliance on a single clinical sign may be misleading. Accurate differentiation between proximal and distal lesions requires integrated evaluation of motor, sensory, reflex, positional, and temporal features.

5. General Principles in the Management of Brachial Plexus Disorders

Evaluation of brachial plexus disorders must extend beyond identification of the affected nerve to include analysis of lesion level, mechanism, and clinical behavior.

Determining the Level of Lesion

The initial step is establishing whether the lesion is proximal or distal. Proximal lesions produce widespread deficits, whereas distal lesions generate localized, nerve-specific findings.

Mechanism of Injury: Static versus Dynamic Factors

Static mechanisms are typically associated with anatomical narrowing or structural pathology, whereas dynamic mechanisms arise from positional changes, repetitive movements, or biomechanical stress. Positional symptom variability suggests a dynamic component.

Integrated Analysis of Clinical Findings

Motor weakness, sensory disturbances, and reflex changes should be evaluated collectively. Concordance between clinical patterns and anatomical organization provides more reliable localization.

Systematic Differential Diagnosis

Differentiation among cervical radiculopathy, plexus lesions, and distal entrapment neuropathies represents a common diagnostic challenge. A systematic proximal-to-distal assessment strategy minimizes diagnostic error and unnecessary interventions.

Treatment Strategy Development

Treatment planning should consider not only diagnosis but also clinical course, symptom duration, and functional requirements. Conservative approaches are appropriate for dynamic and reversible mechanisms, whereas structural compression or progressive deficits may necessitate interventional strategies.

Multidisciplinary Evaluation and Follow-up

Brachial plexus disorders frequently require collaboration among neurology, orthopedics, physical medicine and rehabilitation, and vascular surgery. Long-term follow-up is essential for evaluating treatment outcomes and optimizing functional recovery.

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CHAPTER 2

ERB–DUCHENNE PALSY

ESRA BABAOĞLU[1]

1. Introduction

Definition

Erb–Duchenne palsy (upper trunk palsy) is a type of brachial plexus injury that results from damage to the C5 and C6 spinal nerve roots, corresponding to the upper trunk of the plexus. It most commonly occurs in the obstetric setting, particularly in situations involving excessive traction on the shoulder during delivery or shoulder dystocia.

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When the injury extends to involve the upper and middle trunks (C5–C7), the condition is referred to as extended Erb–Duchenne palsy.

Erb–Duchenne palsy may resolve spontaneously, or it may require rehabilitative treatment and/or surgical intervention, depending on the severity of the injury and the clinical course.

Historical Background

Wilhelm Heinrich Erb (1840–1921) was a German physician often described as the *father of neurology*. He advocated for neurology as a distinct medical specialty separate from psychiatry and made significant contributions to the understanding of peripheral nerve physiology, deep tendon reflexes, and muscular dystrophies.

Guillaume Benjamin Amand Duchenne (1806–1875) was a French physician widely regarded as the *father of electrotherapy and electrodiagnosis*. He provided accurate descriptions of numerous neuromuscular disorders, including pseudohypertrophic muscular dystrophy, now known as Duchenne muscular dystrophy.

Duchenne investigated muscle and nerve physiology using electrical stimulation techniques and, in 1861, was the first to clinically describe obstetric brachial plexus paralysis. In 1874, Erb investigated the specific anatomical source of upper brachial plexus injury and presented four adult cases demonstrating that the paralysis was related to involvement of the C5–C6 nerve roots.

The classic clinical posture of Erb–Duchenne palsy, known as the “waiter’s tip” position, was first introduced as a term in Graham E. E.’s pediatric textbook published in 1916.

In summary, Duchenne defined the clinical syndrome, whereas Erb identified its anatomical localization at the C5–C6 level.

Epidemiology

Reported incidence rates of obstetric brachial plexus palsy vary across geographic regions. According to a study reviewing data from 63 publications, the incidence in the United States was reported as 1.51 per 1,000 live births, while the average incidence in other countries was approximately 1.3 per 1,000 live births. A notable decline in incidence has been observed since the year 2000.

A study conducted in Turkey in 2015 reported an incidence of 0.72 per 1,000 live births. Overall, the global incidence of obstetric brachial plexus palsy has been estimated at 1.74 per 1,000 live births.

Variations in reported incidence have been attributed to differences in study settings, population-based data, availability of maternal–fetal care, and racial or ethnic factors.

It has been reported that approximately 76% of obstetric brachial plexus palsy cases occur without accompanying shoulder dystocia.

Although most cases improve over time, a subset of infants may require surgical intervention. Permanent disability is uncommon; however, complete recovery is not achieved in approximately 20–30% of cases.

2. Anatomical Basis

Because Erb–Duchenne palsy results from injury to the C5–C6 nerve roots of the brachial plexus, it is essential to first examine the structures innervated by these segments.

Branches arising from the roots of the brachial plexus include muscular branches (rr. musculares) that contribute to the innervation of the longus colli muscle and the scalene muscles (mm. scaleni), primarily from C5–C8.

The longus colli muscle functions to flex the neck when acting bilaterally and to rotate the neck contralaterally when acting unilaterally.
The scalene muscles elevate the ribs to assist inspiration and, when their insertions are fixed, laterally flex the neck toward the ipsilateral side (with the middle scalene contributing partially to contralateral flexion). Bilateral contraction results in cervical flexion.

In addition to these muscles, numerous other muscles—listed in the table below—receive innervation from C5 and C6. Consequently, in Erb–Duchenne palsy, muscles supplied by nerve fibers originating from these roots are at risk of functional impairment.

Table 1. Muscles Commonly Affected in Erb–Duchenne Palsy (C5–C6)

Muscle	Nerve	Root Levels	Primary Function
Musculus deltoideus	Axillary nerve	C5–C6	Shoulder abduction (15°–90°)
Musculus teres minor	Axillary nerve	C5–C6	Shoulder adduction, external rotation
Musculus teres major	Lower subscapular nerve	C5–C7	Shoulder adduction, extension, internal rotation
Musculus supraspinatus	Suprascapular nerve	C5–C6	Initiation of shoulder abduction (first 15°)
Musculus infraspinatus	Suprascapular nerve	C5–C6	External rotation of the arm
Musculus subscapularis	Upper and lower subscapular nerves	C5–C6	Shoulder adduction, internal rotation
Musculus pectoralis major (clavicular head)	Lateral pectoral nerve	C5–C6	Shoulder flexion and adduction
Musculus pectoralis minor	Lateral pectoral nerve	C5–C6	Scapular protraction and depression
Musculus biceps brachii	Musculocutaneous nerve	C5–C6	Elbow flexion, forearm supination, shoulder flexion

Muscle	Nerve	Root Levels	Primary Function
Musculus brachialis	Musculocutaneous nerve	C5–C6	Elbow flexion
Musculus coracobrachialis	Musculocutaneous nerve	C5–C7	Shoulder flexion and adduction
Musculus brachioradialis	Radial nerve	C5–C6	Elbow flexion; assists pronation/supination to neutral
Musculus serratus anterior	Long thoracic nerve	C5–C7	Scapular protraction and upward rotation; arm elevation above 90°
Musculi rhomboidei (major & minor)	Dorsal scapular nerve	C4–C5	Scapular retraction, elevation, downward rotation
Musculus levator scapulae	Dorsal scapular nerve	C4–C5	Scapular elevation
Musculus subclavius	Nerve to subclavius	C5–C6	Stabilization of the clavicle
Diaphragma (partial innervation)	Phrenic nerve	C3–C5	Respiration
Musculus latissimus dorsi	Thoracodorsal nerve	C6–C8	Shoulder extension, adduction, internal rotation

In classic Erb–Duchenne palsy, involvement is limited primarily to the C5–C6 nerve roots. As a result, hand, wrist, and finger movements are generally preserved. In contrast, extended Erb–Duchenne palsy, which involves the C5–C7 nerve roots, is associated with functional impairment of wrist and finger extension, reflecting additional involvement of more distal motor pathways. Accordingly, the prognosis of extended Erb–Duchenne palsy is generally less favorable than that of the classic form.

Because these conditions represent nerve injuries, it is appropriate to consider the two principal classification systems used in the evaluation of peripheral nerve damage: the Seddon and Sunderland classifications.
The Seddon classification employs a simple three-stage framework

consisting of neurapraxia, axonotmesis, and neurotmesis. The Sunderland classification provides a more detailed grading system, describing five degrees of nerve injury, with some sources recognizing a sixth degree. This classification is particularly valuable in surgical decision-making and prognostic assessment.

Table 2. Seddon and Sunderland Classification of Peripheral Nerve Injuries

Seddon (1943)	Sunderland (1951)	Description	Recovery / Prognosis
Neurapraxia	Grade I	Isolated myelin injury; axon intact; conduction block present	Complete recovery within days to weeks
Axonotmesis	Grade II	Axonal injury with intact endoneurium, perineurium, and epineurium	Complete regeneration possible; recovery is prolonged
—	Grade III	Injury to axon and endoneurium; perineurium and epineurium intact	Regeneration occurs but is often disorganized; functional recovery may be incomplete
—	Grade IV	Injury to axon, endoneurium, and perineurium; epineurium intact	Spontaneous recovery unlikely; surgical intervention usually required
Neurotmesis	Grade V	Complete transection of the nerve including all connective tissue layers	No spontaneous recovery; surgery required
—	Grade VI*	Mixed injury with different degrees of damage within the same nerve segment	Variable prognosis; surgical treatment is often indicated

3. Etiology and Risk Factors

In general, injury results from excessive separation of the shoulder and neck, leading to traction-related damage to the brachial plexus. Although Erb–Duchenne palsy most commonly develops as a consequence of birth trauma, similar mechanisms may also occur following traumatic events such as motorcycle accidents, falls onto the shoulder, or falls from height (e.g., horseback riding).

In such scenarios, the shoulder is suddenly arrested by impact while the head and trunk continue to move, resulting in traction, stretching, or tearing of the upper components of the brachial plexus. In addition to traction injuries, space-occupying lesions such as tumors, as well as conditions that impair the arterial blood supply of these nerves, may also lead to neural damage.

When the arterial supply of a neuron is compromised, adenosine triphosphate (ATP) production decreases, leading to failure of ion pumps. This cascade results in hypoxia, energy depletion, and glutamate-mediated excitotoxicity, which may ultimately cause neuronal cell death through either regulated apoptosis or uncontrolled necrosis.

Erb–Duchenne palsy is most frequently attributed to obstetric factors, particularly techniques applied during delivery. The condition has been most commonly associated with shoulder dystocia and fetal macrosomia.

Major risk factors include shoulder dystocia, macrosomia (birth weight $\geq$ 4,500 g), maternal diabetes, instrument-assisted vaginal delivery, breech presentation, difficult or prolonged labor, prolonged second stage of labor, cephalopelvic disproportion, excessive fetal manipulation, clavicular or humeral fractures, multiple gestations, preeclampsia, maternal obesity, forceps or traction-based obstetric interventions, and a history of a previous child with Erb–Duchenne palsy.

Although cesarean delivery is generally considered to have a protective effect, rare cases of Erb–Duchenne palsy following cesarean section have also been reported in the literature.

4. Clinical Classification

Several clinical classification systems are used for different purposes in obstetric brachial plexus palsy.

Narakas Classification

The Narakas classification, first described by Ladislaus Narakas, is the most commonly used clinical classification in obstetric brachial plexus palsy. It categorizes patients according to the extent of plexus involvement and associated clinical findings. Type I (Erb type) represents the mildest and most frequent form, in which the shoulder and elbow are affected while the hand remains normal; the lesion predominantly involves C5–C6.
Type II (extended Erb type) includes the features of Type I with additional involvement consistent with radial nerve dysfunction, classically described as loss of the triceps reflex, and typically corresponds to C5–C7 involvement.
Type III (total plexus palsy) denotes involvement of the entire upper extremity, with paralysis affecting both shoulder/elbow and hand/finger function, generally corresponding to C5–T1.
Type IV includes Type III findings plus Horner syndrome (ptosis, miosis, anhidrosis), indicating total plexus involvement with additional disruption of the sympathetic chain; this pattern is associated with a worse prognosis.

Mallet Scoring System

The Mallet score is a functional clinical assessment method, generally applied in children older than three years, to evaluate upper extremity function, particularly shoulder function, through five key movements. These include shoulder abduction, external rotation, and functional tasks such as bringing the hand to the neck, to the mouth, and to the back. Each movement is typically scored on a five-point scale (1 = absent, 5 = normal). The total score ranges from 5 to 25 and is used to support assessment of clinical outcome and to help determine whether surgical intervention may be warranted.

Gilbert–Raimondi Classification

The Gilbert–Raimondi classification is used primarily to evaluate shoulder function, particularly in the postoperative setting. It is based mainly on the degree of shoulder abduction: Grade I indicates no shoulder movement, Grade II corresponds to <30° of abduction, Grade III to 30–90° of abduction, and Grade IV to >90° of abduction.

5. Clinical Manifestations

Duchenne described obstetric brachial plexus palsy as a clinical picture in which the upper extremity appears elongated, with the forearm and hand in pronation due to the position of the humerus; the proximal part of the limb is relatively smaller, and paralysis involves muscles such as m. biceps brachii, m. deltoideus, m. supraspinatus, and m. brachialis, while cutaneous sensation is often preserved and the hand is typically normal in classic Erb involvement.

In the medical literature, the classic posture of Erb palsy, arm adduction and internal rotation, elbow extension, forearm pronation, and wrist flexion,nis widely known as the "waiter's tip" position. Beyond this characteristic posture, patients may also demonstrate sensory disturbance (typically on the lateral aspect of the arm), decreased or absent reflexes, weakness and muscle atrophy, and loss of the Moro reflex on the affected side.

In infants with suspected Erb palsy, associated findings such as Horner syndrome, evidence of other nerve injuries, and torticollis should also be assessed. The presence of Horner syndrome suggests more severe injury, including possible lower trunk involvement and/or root avulsion, and is generally associated with a worse prognosis.

Key clinical signs in narrative form

Clinically, the "waiter's tip" posture is characterized by a flaccid upper limb held in internal rotation with the elbow extended and the forearm pronated, often accompanied by reflex loss and, in more persistent cases, muscle atrophy.
Loss or depression of the biceps reflex (related primarily to the musculocutaneous nerve, predominantly C5–C6) and the brachioradialis reflex (via the radial nerve, with strong C5–C6 contribution) is a diagnostically and prognostically important indicator when C5–C6 segments are involved.
Weakness and atrophy are most evident in muscles responsible for shoulder abduction and elbow flexion, including m. deltoideus, m. biceps brachii, and m. brachialis, resulting in functional limitation of these movements.
Sensory impairment, when present, is commonly detected along the lateral arm, particularly from the shoulder toward the elbow, consistent with C5–C6 dermatomal predominance.
Loss of the Moro reflex on the affected side may be observed and supports the presence of nerve root-level injury.

Most commonly involved muscles (C5–C6 dominant pattern)

In classic Erb palsy, impairment most often involves muscles innervated predominantly by C5–C6. Weakness of m. deltoideus (axillary nerve, C5–C6) leads to loss of shoulder abduction and contributes to the arm hanging at the side. Dysfunction of m. biceps brachii and m. brachialis (musculocutaneous nerve, C5–C7 with C5–C6 dominance) causes loss or marked reduction of elbow flexion and forearm supination, often accompanied by a diminished biceps reflex. Weakness of m. brachioradialis (radial nerve, C5–T1 with strong C5–C6 contribution) further reduces elbow flexion and the ability to return the forearm toward neutral. Involvement of the suprascapular nerve (C5–C6) affects m. supraspinatus and m. infraspinatus, resulting in weak initiation of abduction and impaired external rotation. Weakness of m. teres minor (axillary nerve, C5–C6) and m. infraspinatus further contributes to loss of external rotation, while relative preservation or imbalance of internal rotators promotes the characteristic internally rotated posture. Early assessment of reflexes and muscle function is therefore central to guiding management and predicting outcome.

6. Diagnostic Methods

Diagnosis is primarily established through history and physical examination. When fractures are suspected, particularly clavicular or humeral fractures, plain radiography should be obtained.

For functional evaluation, several structured scoring systems may be used. The Active Movement Scale (AMS) evaluates active range of motion across multiple movements of the shoulder, elbow, forearm, and hand; it is especially useful in infants and young children, although separating gravity effects can be challenging in very small infants. The Toronto Test Score (TTS) is used mainly to support surgical decision-making in infants with obstetric brachial plexus palsy, with particular emphasis on biceps function and elbow flexion; it is useful in the 3–9-month decision window but does not fully represent the entire upper extremity. The Modified Mallet Scale is typically used in older children (commonly >3 years) to assess shoulder function and functional positioning tasks; it requires cooperation and is therefore not appropriate for small infants.

Electrodiagnostic studies (EMG/NCS) may support surgical planning and prognostic assessment. In clinical practice, EMG is often performed around 2–4 weeks to 1 month after injury, and may be repeated every 2–3 months if needed to track denervation and reinnervation patterns.

High-resolution MRI, including MR neurography (e.g., T2-STIR, 3D sequences such as FIESTA/3D-T2 depending on protocol), is valuable for evaluating nerve continuity, preoperative mapping, and distinguishing preganglionic versus postganglionic injuries. MR myelography has been reported to show high diagnostic accuracy for detecting root avulsion, and MRI findings can contribute to surgical planning. However, because imaging sensitivity and specificity vary by technique and timing, surgical exploration remains the reference standard in many settings for definitive assessment of avulsion and severe rupture.

In recent years, ultrasonography (US) has been increasingly utilized due to its accessibility, real-time evaluation, and the lack of need for sedation or contrast. US may be particularly helpful in preoperative mapping of the supraclavicular brachial plexus and can complement MRI. It may also detect nerve injury patterns such as neuroma formation and scar tissue.

7. Differential Diagnosis

Conditions to consider in the differential diagnosis include fractures, septic shoulder, cerebral palsy, isolated radial nerve palsy, Klumpke palsy (C8–T1), pseudoparalysis, congenital myopathies, cervical spinal cord lesions, congenital varicella infection, spinal muscular atrophy (SMA), Duchenne muscular dystrophy (DMD), and Becker muscular dystrophy (BMD).

Clinically, the grasp reflex is usually preserved in Erb–Duchenne palsy, which helps distinguish it from Klumpke palsy. A systematic approach integrates (i) clinical posture and proximal weakness with reflex loss, (ii) EMG/NCS demonstrating denervation in the C5–C6 distribution, (iii) imaging (MRI/US) for avulsion or rupture versus alternative structural pathology, (iv) laboratory markers such as creatine kinase (CK) where muscular disease is suspected, (v) genetic testing when dystrophinopathies are in question, and (vi) history consistent with birth trauma versus congenital/genetic onset.

8. Treatment Methods

Obstetric brachial plexus palsy requires longitudinal follow-up from early infancy until musculoskeletal maturation and achievement of optimal function. Although many cases show spontaneous recovery, delay in appropriate assessment and rehabilitation may contribute to avoidable long-term sequelae, and recent reports have noted a meaningful proportion of persistent deficits.

The first-line approach is early physiotherapy, initiated as soon as possible, with goals including prevention of contractures, strengthening, sensory stimulation, passive range of motion exercises, and support of normal developmental milestones. Splinting has been reported to reduce deformity, prevent contractures, and improve motor development. Use of a supination and external rotation orthosis (Sup-ER) worn for prolonged daily periods (e.g., up to 22 hours/day in some protocols) aims to preserve shoulder development until reinnervation improves function; early initiation (around 6 weeks) has been reported to support functional gains in selected patients.

Botulinum toxin type A (BTX-A) injections may help manage muscle imbalance and, in selected cases, may delay, modify, or potentially reduce the need for surgery. Ultrasound-guided application can improve accuracy and allow lower dosing with fewer adverse effects.

Surgical treatment is considered for preganglionic avulsions, for postganglionic injuries without adequate motor recovery within 3–6 months, for severe rupture or transection, or when nonoperative management fails to achieve meaningful improvement. Lack of reinnervation signs on EMG at around 3 months, together with persistent denervation findings, may support surgical referral. Because many newborn cases show partial or complete recovery within the first 3 months, close monitoring and conservative treatment are the standard early approach; surgical decisions are commonly guided by 3–6-month evaluations.

Treatment by developmental stage (narrative)

In the early period (0–3 months), management emphasizes passive range of motion exercises, gentle mobilization to support shoulder abduction and external rotation, scapular stabilization, and splinting when appropriate to prevent contracture (including Sup-ER–typeprotocols).

During the intermediate period (3–6 months), active-assisted exercises, age-appropriate isometric strengthening (particularly targeting deltoid, biceps, supraspinatus), and play-based functional training are introduced; surgical evaluation is considered if recovery isinsufficient.

In the later period (>6 months), progressive strengthening, proprioceptive training, and, in selected contexts, electrostimulation may be used; if persistent deficits or secondary deformities develop, orthopedic surgical assessment (e.g., tendon transfer or osteotomy) may be indicated.

Surgical timing and strategy (narrative)

Surgery performed within 3–6 months is often described as early surgery and tends to yield better outcomes, particularly for avulsion and severe rupture. If biceps contraction does not emerge within this time window, primary nerve surgery, including nerve grafting, nerve transfers, or neurolysis, may be considered. Intraoperative assessment of conduction across a neuroma may influence strategy: cases with relatively preserved conduction may benefit from neurolysis, whereas poor conduction and more severe pathology may favor nerve transfer procedures.

If primary nerve surgery is performed after 6 months (commonly between 6 and 9–12 months), it is often termed delayed surgery; although grafts/transfers may still be performed, denervation atrophy and motor end-plate degeneration increasingly limit recovery. After 12–18 months, primary nerve surgery becomes less effective, and secondary reconstructions such as muscle transfer, tendon transfer, or osteotomies are typically considered.

From a practical standpoint, avulsion injuries usually mandate surgical reconstruction (most commonly nerve transfers and plexus reconstruction) within the early window. Rupture injuries may require grafting or transfer if spontaneous recovery is unlikely. Neuroma-in-continuity may be treated with neurolysis or transfer depending on functional status and conduction. Suprascapular nerve paralysis may be treated with nerve transfer (e.g., from the spinal accessory nerve), aiming to improve shoulder function.

9. Prognosis and Follow-up

Prognosis depends on lesion severity and the timing/quality of rehabilitation and surgical decision-making. Follow-up is typically structured by age: early infancy focuses on range of motion and early motor signs; 3–6 months represents a critical reassessment period; 6–12 months includes postoperative monitoring when surgery is performed and continued rehabilitation; and long-term follow-up (1–3 years and beyond) focuses on functional development and daily activity performance, with revision procedures considered when needed.

A key prognostic marker is acquisition of antigravity biceps activity, reflecting the ability to flex the elbow against gravity. Failure to gain this capacity suggests delayed motor recovery or more severe nerve injury. Muscle strength is commonly quantified using the Medical Research Council (MRC) scale, ranging from 0 (no activity) to 5 (normal strength). If m. biceps brachii does not achieve antigravity activity by 3–6 months, nerve repair or transfer is considered; in later periods, tendon transfer and other secondary options may be required.

In routine follow-up, combinations of MRC, Mallet, and AMS scoring are often used. Serial AMS scores are particularly helpful for tracking recovery and supporting clinical decisions between continued conservative management and surgical referral. Low early Toronto scores and poor Mallet performance over time may similarly indicate the need for escalation of treatment.

10. Conclusion

Although Erb–Duchenne palsy (obstetric brachial plexus palsy) demonstrates spontaneous improvement in many patients, a clinically meaningful proportion develop persistent functional limitations and sequelae. Accordingly, a multidisciplinary strategy incorporating early standardized assessment, structured follow-up, and timely surgical decision-making is essential. Current evidence supports the use of regular objective evaluation tools (e.g., AMS, TTS, Modified Mallet, and serial MRC assessments), with particular attention to key milestones at 3 and 6 months, especially the presence or absence of antigravity biceps recovery. Early conservative measures, physiotherapy, splinting (including Sup-ER protocols), and selected use of botulinum toxin, constitute the foundation of management; however, preganglionic avulsion, open rupture, or lack of meaningful motor recovery by 3–6 months may warrant primary nerve surgery (nerve transfer and/or grafting). Long-term outcomes are optimized through integration of rehabilitation, appropriate orthoses, surgical interventions when indicated, and sustained functional follow-up, while standardization of multidisciplinary protocols and systematic data collection remain important for improving population-level incidence and prognosis data.

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CHAPTER 3

THORACIC OUTLET SYNDROME (TOS)

ABDULLAH ÖRS[1]

1. Introduction

Definition

Thoracic outlet syndrome (TOS) is a clinical syndrome that arises as a result of chronic or intermittent compression of the brachial plexus and/or the subclavian vessels within anatomically predisposed narrow spaces of the cervicothoracic transition zone. This compression is characterized by neurological and vascular manifestations in the upper extremity, including pain, paresthesia, weakness, sensory deficits, coldness, color changes, and circulatory disturbances. Rather than representing a single pathological process,

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TOS refers to a heterogeneous clinical entity resulting from the combination of anatomical variations, mechanical factors, and environmental influences.

The thoracic outlet region is a complex anatomical transition zone encompassing the neck, thorax, and axilla, and contains three narrow anatomical spaces through which the brachial plexus, subclavian artery, and subclavian vein course together: the interscalene triangle, the costoclavicular space, and the retropectoralis minor space. Congenital anomalies of the osseous, muscular, and fibrous structures within these regions, such as cervical ribs, variations of the first rib, scalene muscle anomalies, and fibrous bands, increase the susceptibility of the neurovascular structures to compression. However, it is widely accepted that anatomical predispositions alone are insufficient to produce symptoms; in most cases, the clinical picture emerges in conjunction with triggering factors such as trauma, inflammation, postural abnormalities, or repetitive use of the upper extremity.

From a clinical perspective, thoracic outlet syndrome is classified into three main subtypes: neurogenic, venous, and arterial. Neurogenic TOS constitutes the vast majority of cases and is primarily associated with compression of the brachial plexus, particularly the lower trunk. In venous TOS, compression of the subclavian vein leads to predominant findings such as upper extremity swelling, cyanosis, and venous distension. In arterial TOS, involvement of the subclavian artery results in symptoms including coldness, pallor, exertional pain, and, rarely, distal embolic manifestations. In this regard, TOS differs from isolated peripheral nerve entrapment syndromes in that both neural and vascular structures may be affected simultaneously.

Thoracic outlet syndrome has also been described under various terminological and nosological designations throughout history. Terms such as cervical rib syndrome, scalenus anticus syndrome, and costoclavicular syndrome have been used in the literature to describe clinical conditions that are now encompassed within the concept of TOS. Contemporary approaches emphasize the shared pathophysiological basis of these entities, considering all neurovascular compressions occurring within the thoracic outlet region under the unified concept of thoracic outlet syndrome.

Historical Background

The historical development of thoracic outlet syndrome has progressed in parallel with an improved understanding of the relationship between anatomical observations and clinical symptoms. The concept that neurovascular structures in the cervicothoracic region could be subjected to compression first emerged in the mid-19th century through descriptions of anatomical variations. In particular, the association between the presence of a cervical rib and neurological symptoms of the upper extremity was among the earliest clinical observations to attract attention. In 1861, Holmes Coote described a case of brachial plexus compression caused by a cervical rib, thereby laying the foundation for pathologies that would later be classified under the umbrella of thoracic outlet syndrome.

In the early 20th century, recognition that anatomical structures other than the cervical rib could produce similar clinical presentations led to an expansion of the concept. During the first half of the 1900s, Adson and Coffey emphasized the importance of the relationship between the anterior scalene muscle and the subclavian artery and introduced the term “scalenus anticus syndrome” into the literature. The description of the Adson maneuver during this period contributed significantly to the development of diagnostic approaches and reinforced the concept of the thoracic outlet as a dynamic compression region.

The 1940s and 1950s marked a period during which thoracic outlet syndrome began to be addressed more systematically from a clinical standpoint. In 1956, Peet and colleagues used the term "thoracic outlet syndrome" in a comprehensive framework for the first time, proposing that compressions originating from the cervical rib, costoclavicular space, and scalene triangle be unified under a single clinical heading. This approach represented a turning point by demonstrating that previously independent clinical entities shared a common pathophysiological basis.

In the 1970s, the work of Roos played a decisive role in shaping the surgical anatomy and treatment strategies of thoracic outlet syndrome. With the description of the transaxillary first rib resection technique, TOS surgery became more standardized, and attention was drawn to the clinical significance of congenital bands, fibrous structures, and muscular anomalies. During this period, it became evident that thoracic outlet syndrome was too complex to be attributed solely to osseous abnormalities.

In the 1980s and 1990s, advances in electrophysiological methods and imaging techniques enabled more detailed investigation of thoracic outlet syndrome, particularly its neurogenic form. However, heterogeneity in diagnostic criteria and the limited availability of objective tests led to the perception of TOS as a controversial clinical entity. At times, concerns regarding overdiagnosis were raised, further underscoring the importance of precise definitions and classification efforts.

Since the 2000s, the approach to thoracic outlet syndrome has evolved toward a multidisciplinary perspective. Clearer delineation of neurogenic, venous, and arterial subtypes; recognition of previously underappreciated mechanisms such as pectoralis minor compression; and emphasis on the dynamic and functional aspects of the syndrome have refined current understanding. Today, thoracic outlet syndrome is regarded not as a static anatomical abnormality but as a complex clinical syndrome resulting from the interaction between individual anatomical predispositions and environmental and mechanical factors.

Epidemiology

The epidemiology of thoracic outlet syndrome remains a subject of debate due to the heterogeneous nature of the syndrome and variability in diagnostic criteria. Reported prevalence and incidence rates vary considerably depending on the diagnostic standards employed, the population studied, and the TOS subtype evaluated. Consequently, it is difficult to define precise and universally accepted epidemiological data for thoracic outlet syndrome.

In the general population, the prevalence of thoracic outlet syndrome has been reported to range between 0.3% and 8%. This wide range is largely attributable to the subjective nature of diagnosing neurogenic TOS. Neurogenic TOS accounts for approximately 70–90% of all cases and represents the most common subtype. Venous TOS is reported in approximately 3–5% of cases, whereas arterial TOS is the rarest form, accounting for less than 1% of all cases. Although vascular forms are less common, their diagnoses are generally more straightforward due to the presence of objective clinical and imaging findings.

With regard to sex distribution, thoracic outlet syndrome is reported to occur more frequently in women than in men. Neurogenic TOS, in particular, has been reported to be approximately two to four times more common in women. This difference has been attributed to a higher prevalence of cervical ribs and fibromuscular variations in women, differences in shoulder girdle anatomy, and postural factors. In contrast, venous TOS has been reported more frequently in young men and is often associated with intensive upper extremity use and repetitive strenuous activities, as seen in Paget–Schrötter syndrome.

In terms of age distribution, thoracic outlet syndrome is most commonly diagnosed in young and middle-aged adults. Neurogenic TOS typically presents between the ages of 20 and 50 years, whereas venous TOS often manifests as an acute condition in young adult males. Arterial TOS is frequently associated with underlying osseous anomalies and may be diagnosed either at younger ages or later in life as a result of long-standing subclavian artery compression.

Occupational and environmental factors play a significant role in the epidemiology of thoracic outlet syndrome. An increased incidence of TOS has been reported in occupational groups involving repetitive and strenuous use of the upper extremities. Athletes engaged in overhead activities (such as swimmers, volleyball players, and baseball pitchers), individuals performing heavy lifting, those working long hours at computers, and individuals with poor postural habits are considered to be at increased risk. These findings support the concept that TOS is not merely a static anatomical disorder but also a dynamic and functional syndrome.

Many epidemiological studies have demonstrated that anatomical variations within the thoracic outlet region are relatively common in the general population, yet the majority of affected individuals remain asymptomatic. Structural features such as cervical ribs, fibrous bands, and muscular anomalies are not sufficient on their own to cause disease; rather, the clinical syndrome typically emerges in conjunction with additional factors such as trauma, postural abnormalities, or repetitive mechanical stress. This observation highlights the limitations of interpreting epidemiological data based solely on anatomical findings.

In conclusion, the epidemiology of thoracic outlet syndrome cannot be defined with precise boundaries due to diagnostic challenges and clinical heterogeneity. Contemporary approaches emphasize that epidemiological data should be interpreted by considering TOS subtypes, risk factors, and functional components, and underscore the need for studies based on more standardized diagnostic criteria, particularly for neurogenic thoracic outlet syndrome.

2. Anatomy

The thoracic outlet region is an anatomically narrow and functionally dynamic transitional zone through which the neurovascular structures extending from the cervical spine to the axilla pass. Within this region, the brachial plexus, subclavian artery, and subclavian vein traverse confined anatomical spaces surrounded by osseous, muscular, and fibrous structures to reach the upper extremity. The anatomical basis of thoracic outlet syndrome consists not only of the normal anatomical relationships of these structures but also of variations that are frequently encountered among individuals.

General Anatomical Organization of the Thoracic Outlet Region

From both anatomical and clinical perspectives, the thoracic outlet is evaluated within three principal transitional spaces: the interscalene space (spatium interscalenum), the costoclavicular space (spatium costoclaviculare), and the subcoracoid or retropectoralis minor space (spatium subcoracoideum). These regions are considered potential sites of narrowing along the physiological course of the neurovascular structures supplying the upper extremity.

Among these, the interscalene space holds central importance in the anatomical basis of thoracic outlet syndrome, as the roots and trunks of the brachial plexus and the subclavian artery pass together through this region.

Interscalene Space (Spatium Interscalenum)

The interscalene space is a triangular anatomical interval bounded anteriorly by the anterior scalene muscle (musculus scalenus anterior), posteriorly by the middle scalene muscle (musculus scalenus medius), and inferiorly by the first rib (costa prima). The subclavian artery and the roots and trunks of the brachial plexus pass through this space.

In contrast, the subclavian vein lies anterior to this space, on the anterior surface of the anterior scalene muscle, and is not considered a component of the interscalene space.

Anterior Scalene Muscle (Musculus Scalenus Anterior)

The anterior scalene muscle originates from the anterior tubercles of the transverse processes of the third to sixth cervical vertebrae (C3–C6) and inserts onto the scalene tubercle (tuberculum musculi scaleni anterioris) of the first rib. Its muscle fibers course in an inferolateral direction.

The medial surface of the muscle is in close relationship with the brachial plexus and the subclavian artery, while its anterior surface is associated with the subclavian vein. Due to this anatomical positioning, hypertrophy, fibrotic changes, or insertional anomalies of the anterior scalene muscle are of critical importance in the development of thoracic outlet syndrome.

The presence of accessory muscle slips, a broad insertion, or a more posterior-than-normal position of the anterior scalene muscle may significantly reduce the volume of the interscalene space.

Middle Scalene Muscle (Musculus Scalenus Medius)

The middle scalene muscle originates from the posterior tubercles of the transverse processes of the second to seventh cervical vertebrae (C2–C7) and inserts onto the posterior aspect of the first rib. It is generally larger and longer than the anterior scalene muscle.

The roots and trunks of the brachial plexus are located between the anterior and middle scalene muscles. An abnormal anterior displacement of the middle scalene muscle or a reduction in the distance between it and the anterior scalene muscle may result in chronic compression of the neural structures.

In some individuals, fibers of the brachial plexus pass through or between the fibers of the middle scalene muscle, rendering the nerves more susceptible to mechanical irritation and traction.

Scalenus Minimus Muscle (Musculus Scalenus Minimus)

The scalenus minimus muscle is not defined as a standard anatomical structure in Terminologia Anatomica; however, it represents a frequently encountered muscular variation in the thoracic outlet region. It typically originates from the transverse processes of the sixth and seventh cervical vertebrae (C6–C7) and inserts onto the first rib or the cupula pleurae.

This muscle is often located between the subclavian artery and the inferior trunk of the brachial plexus and may directly cause neurovascular compression. Clinically, it is particularly associated with neurogenic thoracic outlet syndrome.

Anatomical Variations of the Scalene Muscles

Variations of the scalene muscle group constitute one of the most important anatomical factors underlying thoracic outlet syndrome. These variations may manifest as abnormal origins or insertions, partial or complete fusion of muscle fibers, accessory muscular bands, or fibrous structures.

Partial or complete fusion of the anterior and middle scalene muscles may lead to obliteration or marked narrowing of the interscalene space. In addition, fibrous bands extending between muscle fibers may cause oblique compression of elements of the brachial plexus.

Although many of these variations may also be present in asymptomatic individuals, the clinical syndrome often emerges when they coexist with factors such as trauma, postural abnormalities, or repetitive upper extremity movements.

Clinical and Surgical Anatomical Considerations

Detailed anatomical knowledge of the scalene muscles and their variations is of critical importance for the diagnostic evaluation and surgical treatment of thoracic outlet syndrome. During supraclavicular and infraclavicular surgical approaches, particular attention must be paid to the relationships between the scalene muscles and the phrenic nerve, long thoracic nerve, and dorsal scapular nerve.

In conclusion, the anatomy of the thoracic outlet region demonstrates significant individual variability, particularly with respect to the scalene muscles and their variations. This anatomical diversity accounts for the clinical heterogeneity of thoracic outlet syndrome and underscores the necessity for individualized assessment in each patient.

3. Etiology and Risk Factors

The etiology of thoracic outlet syndrome is multifactorial and cannot be attributed to a single cause. It represents a complex process resulting from the combined effects of congenital anatomical features, acquired structural changes, and functional–mechanical factors. Contemporary perspectives regard thoracic outlet syndrome not as a static anatomical anomaly but as a dynamic syndrome arising from the interaction between individual anatomical predispositions and environmental and biomechanical stresses.

Congenital Anatomical Factors

Congenital factors constitute the fundamental predisposing elements for thoracic outlet syndrome. These factors rarely produce symptoms in isolation but create a substrate upon which additional mechanical or traumatic influences may precipitate the clinical condition.

The most frequently reported congenital causes include cervical ribs, anomalies of the first rib, scalene muscle variations, and fibrous bands. Cervical ribs typically arise from the transverse process of the seventh cervical vertebra (C7) and may develop in complete or incomplete forms. Incomplete cervical ribs are often connected to the first rib by fibrous bands, leading to compression particularly of the inferior trunk of the brachial plexus.

Anomalies of the first rib may present as enlargement, hypoplasia, abnormal articulation, or oblique orientation. These variations may reduce the volume of the interscalene and costoclavicular spaces, thereby impeding the passage of neurovascular structures.

Congenital variations of the scalene muscles represent one of the most important anatomical substrates for thoracic outlet syndrome. Partial or complete fusion of the anterior and middle scalene muscles, the presence of accessory muscular bands, or the existence of a scalenus minimus muscle may result in direct compression of the brachial plexus and subclavian artery.

Acquired Structural Factors

Acquired factors play a significant role in rendering thoracic outlet syndrome clinically apparent. These include trauma-related changes, degenerative processes, and inflammatory conditions.

Trauma affecting the cervical region and shoulder girdle, particularly whiplash-type injuries, may lead to fibrosis and thickening of the scalene muscles, resulting in narrowing of the interscalene space. Callus formation or scar tissue following fractures of the clavicle or first rib may create a substantial mechanical obstacle to neurovascular structures within the costoclavicular space.

Repetitive microtrauma and chronic inflammation may reduce muscle elasticity and promote fibrotic transformation, thereby diminishing the adaptive capacity of the thoracic outlet region. This process particularly exposes elements of the brachial plexus to chronic traction and friction.

Functional and Mechanical Risk Factors

Functional factors often act as triggering mechanisms in the development of thoracic outlet syndrome. Repetitive and forceful use of the upper extremity, overhead activities, and prolonged static postures may dynamically compress neurovascular structures within the thoracic outlet region.

Postural abnormalities characterized by depression, protraction, and anterior tilt of the shoulder girdle reduce the distance between the clavicle and the first rib, thereby narrowing the costoclavicular space. Simultaneously, inferior and anterior rotation of the scapula increases the risk of compression within the subcoracoid space due to the pectoralis minor muscle.

Athletes, particularly swimmers, volleyball players, and individuals engaged in throwing sports, are considered at high risk for thoracic outlet syndrome due to repetitive upper extremity abduction and external rotation. Similarly, prolonged computer use may contribute to postural deterioration of the shoulder girdle, facilitating the development of the syndrome.

Etiological Characteristics According to Subtypes

Neurogenic thoracic outlet syndrome is most commonly associated with chronic compression of the brachial plexus resulting from anatomical variations and functional stressors, with muscular and fibrous structures playing a predominant role.

Venous thoracic outlet syndrome typically develops as a result of repetitive mechanical stress on the subclavian vein and may present with acute thrombosis. Arterial thoracic outlet syndrome is most often associated with prominent osseous factors, such as cervical ribs or first rib anomalies, and represents the rarest subtype.

Multifactorial Approach to Etiopathogenesis

Current literature indicates that no single factor is solely responsible for the etiopathogenesis of thoracic outlet syndrome. The interaction between anatomical variations, environmental influences, and functional loading plays a decisive role in the emergence of the syndrome. Therefore, thoracic outlet syndrome should be regarded not merely as a structural abnormality but as a complex clinical entity shaped by an individual risk profile.

4. Clinical Manifestations

The clinical manifestations of thoracic outlet syndrome vary according to the neurovascular structure subjected to compression. Therefore, clinical assessment should be performed based on three main subtypes: neurogenic, venous, and arterial. Each subtype is characterized by distinct symptom patterns and clinical course features.

Neurogenic Thoracic Outlet Syndrome

Neurogenic thoracic outlet syndrome constitutes the largest proportion of thoracic outlet syndrome cases. The clinical presentation is primarily related to compression of the brachial plexus, most commonly affecting fibers of the lower trunk (C8–T1).

The most frequent symptoms are pain radiating to the neck, shoulder, scapular region, and upper extremity, accompanied by paresthesia. Pain is typically dull, burning, or aching in character and tends to worsen with prolonged use of the upper extremity, overhead activities, or sustained static postures. Paresthesia is most prominent along the ulnar aspect of the hand, particularly involving the fourth and fifth digits.

Patients commonly report early fatigability of the upper extremity, a sense of weakness, and difficulty with fine motor activities. In advanced cases, weakness of the intrinsic hand muscles and, rarely, atrophy may develop. However, the frequent absence of clear objective neurological deficits represents one of the principal reasons for the diagnostic challenge of neurogenic TOS.

Symptoms are generally position-dependent and may be exacerbated by shoulder abduction, external rotation, or cervical lateral flexion. Because the clinical picture may overlap with cervical radiculopathy or distal entrapment neuropathies, differential diagnosis is of major importance.

Venous Thoracic Outlet Syndrome

Venous thoracic outlet syndrome results from compression of the subclavian vein within the thoracic outlet region. Although less common than the neurogenic subtype, it may present with a more dramatic and acute clinical course.

Typical findings include sudden-onset swelling of the upper extremity, a sensation of fullness, and cyanosis. The arm is often described as tense and heavy. Prominence of superficial veins and asymmetry in arm circumference are common. Pain is usually diffuse and may increase with elevation of the arm.

In some cases, particularly in young and physically active individuals, venous thoracic outlet syndrome may present with acute subclavian vein thrombosis. This entity is described in the literature as effort thrombosis or Paget–Schrötter syndrome, in which marked edema, pain, and discoloration develop abruptly.

Symptoms in venous TOS typically progress rapidly; in chronic cases, the development of collateral venous circulation may partially mask clinical findings.

Arterial Thoracic Outlet Syndrome

Arterial thoracic outlet syndrome is the rarest subtype and develops as a result of compression of the subclavian artery. In most cases, there is an underlying prominent osseous anomaly, particularly a cervical rib or an anomalous first rib.

Clinical manifestations include coldness, pallor, early fatigability, and exertional pain in the upper extremity. Patients often complain of a sensation of impaired circulation in the hand and forearm. In advanced cases, diminished pulses or position-dependent loss of pulses may be detected.

Chronic arterial compression may lead to intimal injury of the subclavian artery, post-stenotic dilatation, or aneurysm formation. This increases the risk of distal embolization and may result in serious clinical conditions such as ischemic digital pain, ulceration, or, rarely, gangrene.

Because of its potential for serious complications, arterial thoracic outlet syndrome requires early diagnosis and treatment.

Overlap Between Clinical Subtypes

Although thoracic outlet syndrome is clinically categorized into three main subtypes, it should be recognized that some patients may present with both neurogenic and vascular findings. Particularly in cases of advanced anatomical narrowing or long-standing compression, symptoms may reflect involvement of more than one structure, resulting in a complex clinical picture.

Accordingly, during clinical evaluation, the type, duration, positional dependence, and progression of symptoms should be analyzed carefully; while attempting to distinguish subtypes, the overall clinical coherence should not be overlooked.

5. Diagnostic Methods

The diagnosis of thoracic outlet syndrome cannot be established by a single test; rather, it requires integrated interpretation of a detailed clinical assessment, selected provocative tests, imaging modalities, and ancillary diagnostic investigations. The diagnostic approach varies according to symptom characteristics and the suspected compressed structure (neurogenic, venous, or arterial).

Clinical Assessment and Physical Examination

A detailed history constitutes the cornerstone of the diagnostic process. The location and duration of symptoms, their relationship with position, exacerbation with activity, and whether they improve with rest should be assessed carefully. Symptom provocation with overhead activities, shoulder abduction, and prolonged static postures is diagnostically important.

On physical examination, cervical range of motion, shoulder girdle posture, scapular position, and muscular imbalances are evaluated. In neurogenic TOS, overt objective neurological deficits are often absent, which contributes to diagnostic difficulty.

Provocative tests may be helpful but have limited sensitivity and specificity and should be interpreted in conjunction with the clinical picture:

Adson test: Decrease in radial pulse or symptom provocation with cervical rotation and deep inspiration.

Roos (EAST) test: Symptom reproduction during repeated hand opening and closing with the shoulders at 90° abduction and external rotation.

Wright test: Assessment of symptoms and pulse changes during shoulder hyperabduction.

A positive test is not diagnostic on its own and must be interpreted in the clinical context.

Imaging Modalities

Imaging plays an important role in identifying structural causes and mechanisms of compression in thoracic outlet syndrome.

Plain radiography: First-line evaluation for detecting a cervical rib, first rib anomalies, and osseous variations.

Ultrasonography (US): Particularly valuable in vascular TOS due to the ability to perform dynamic assessment; changes in vessel caliber and flow with arm positioning can be evaluated.

Computed tomography (CT) and CT angiography: Provide detailed assessment of osseous structures and vascular compression.

Magnetic resonance imaging (MRI) and MR angiography: Useful for evaluating soft tissues, the brachial plexus, and vascular relationships; often preferred in neurogenic TOS to demonstrate anatomical variations.

For many imaging modalities, evaluation in dynamic positions increases diagnostic yield.

Electrophysiological Studies

Electromyography and nerve conduction studies (EMG/NCS) are adjunctive diagnostic tools in thoracic outlet syndrome. However, particularly in neurogenic TOS, electrophysiological findings may be within normal limits in many patients.

EMG/NCS is mainly used to differentiate: distal entrapment neuropathies (e.g., cubital tunnel syndrome, carpal tunnel syndrome), and cervical radiculopathies.

Although findings consistent with lower trunk involvement may be detected in advanced and chronic neurogenic TOS, normal EMG/NCS results do not exclude the diagnosis.

Diagnostic Injections

In selected cases, particularly when neurogenic TOS is suspected, diagnostic injections of the scalene muscles or the pectoralis minor muscle using a local anesthetic may be utilized. Marked and temporary symptom relief following injection suggests that the targeted anatomical region is responsible for symptoms and may contribute to treatment planning.

Diagnostic Approach According to Subtypes

Neurogenic TOS: Clinical assessment is fundamental; imaging and EMG/NCS are mainly used for differential diagnosis.

Venous TOS: Doppler ultrasonography, CT/MR venography, and, when necessary, conventional venography are central to diagnosis.

Arterial TOS: CT/MR angiography and conventional angiography are important for assessing arterial wall pathology and distal embolic risk.

General Principles of the Diagnostic Approach

In thoracic outlet syndrome, diagnosis is established not by isolated positive tests but by the consistency of clinical findings and the integrated evaluation of structural and functional data. Individualization of the diagnostic process is crucial to avoid unnecessary interventions.

6. Treatment Approaches

Treatment of thoracic outlet syndrome should be planned according to symptom severity and duration, the affected neurovascular structure, and the patient's functional requirements. Contemporary management begins with conservative methods, progressing to interventional and, when necessary, surgical options in cases with persistent or progressive symptoms. Treatment, particularly for neurogenic TOS, requires a stepwise and individualized approach.

Conservative Treatment

Conservative treatment is the first-line approach, especially in the neurogenic form of thoracic outlet syndrome. The primary goal is to reduce mechanical load on neurovascular structures within the thoracic outlet and to control symptoms.

Physiotherapy and exercise programs form the basis of conservative treatment. These programs are designed to correct shoulder girdle posture, enhance scapular stability, and address cervicothoracic muscular imbalances. Stretching exercises targeting the scalene muscles (musculi scaleni), pectoralis minor muscle (musculus pectoralis minor), and levator scapulae muscle (musculus levator scapulae), as well as strengthening of the lower trapezius (musculus trapezius pars descendens/inferior portion) and serratus anterior (musculus serratus anterior), are important components of management.

Postural education and ergonomic modifications are effective in symptom control, particularly in individuals working in prolonged static positions. Limitation of overhead movements and repetitive strenuous upper extremity use in daily activities is recommended.

Pharmacological treatment is used for symptomatic relief. Nonsteroidal anti-inflammatory drugs and analgesics may help control pain; however, pharmacological therapy alone does not provide a permanent solution and is supportive in nature.

Interventional Treatment Methods

Interventional methods may be considered when symptoms persist despite conservative therapy or when diagnostic uncertainty remains. These approaches may serve both diagnostic and therapeutic purposes.

Scalene muscle injections are applied in selected patients, particularly in neurogenic TOS. Local anesthetic or botulinum toxin injections aim to reduce muscle spasm. Temporary symptom improvement following injection may suggest that the targeted anatomical region is responsible and may aid surgical planning.

In vascular thoracic outlet syndrome, particularly the venous form, catheter-directed thrombolysis and anticoagulation therapy may be used in the acute setting. This approach is important for restoring vessel patency and preventing chronic venous obstruction.

Interventional methods generally provide temporary benefit and may be insufficient in the presence of persistent anatomical compression; therefore, patient selection is critical.

Surgical Treatment

Surgical treatment should be considered in cases with persistent symptoms despite conservative and interventional approaches, significant functional impairment, or risk of vascular complications. The primary objective of surgery is elimination of the anatomical causes of compression within the thoracic outlet.

In neurogenic TOS, surgery typically involves first rib resection, scalenectomy, and, when necessary, excision of fibrous bands. Surgical approaches may be supraclavicular, infraclavicular, or transaxillary, depending on the site of compression and surgeon experience.

In venous TOS, surgical treatment is often planned after thrombolysis in the acute phase or in cases with chronic venous obstruction. The goal is to relieve extrinsic compression of the subclavian vein and maintain venous patency.

In arterial TOS, surgical treatment is generally unavoidable. Removal of osseous compressive causes such as a cervical rib or anomalous first rib may be combined with arterial reconstruction or aneurysm repair.

General Principles in Treatment Selection

Successful treatment of thoracic outlet syndrome depends on appropriate patient selection and timing. In neurogenic TOS, adequate time should be allowed for conservative therapy; in vascular forms, a more early and aggressive approach is often required due to the risk of complications. Multidisciplinary evaluation is particularly important before surgical decision-making.

7. Prognosis

The prognosis of thoracic outlet syndrome varies depending on subtype, symptom duration and severity, underlying anatomical causes, and the treatment modality applied. Overall, prognosis is favorable in cases diagnosed early and treated appropriately. Nevertheless, due to the heterogeneous nature of the syndrome, long-term outcomes may differ among individuals.

Neurogenic Thoracic Outlet Syndrome

In neurogenic TOS, prognosis is more variable than in other subtypes. A substantial proportion of patients who respond well to conservative treatment may achieve marked symptom reduction or complete resolution. Long-term outcomes are generally favorable in cases with short symptom duration and without significant muscle atrophy or permanent neurological deficits.

Reported surgical success rates for neurogenic TOS vary widely in the literature. With appropriate patient selection and adequate removal of the anatomical causes of compression, functional improvement and enhanced quality of life may be achieved. Conversely, outcomes may be more limited in patients with long-standing symptoms, central sensitization, or concomitant psychosocial factors.

Venous Thoracic Outlet Syndrome

In venous TOS, prognosis largely depends on timing of diagnosis and treatment. Long-term venous patency and functional outcomes are generally good in patients treated during the acute phase with appropriate thrombolytic therapy combined with surgical decompression.

Delayed treatment increases the risk of chronic venous obstruction, development of collateral circulation, and post-thrombotic syndrome, which may lead to persistent swelling, pain, and functional limitation of the upper extremity. Therefore, venous TOS is regarded as a clinical entity requiring early diagnosis and intervention.

Arterial Thoracic Outlet Syndrome

Although rare, arterial TOS has particular prognostic significance due to potentially serious complications. Surgical removal of prominent osseous compressive causes such as a cervical rib or first rib anomalies typically results in symptom improvement.

However, delayed diagnosis may lead to intimal injury of the subclavian artery, aneurysm formation, or distal embolization. Because these complications may cause permanent ischemic damage and functional loss in the upper extremity, prognosis in arterial TOS is closely linked to early surgical intervention.

Factors Influencing Prognosis

Key factors influencing prognosis in thoracic outlet syndrome include symptom duration, the affected neurovascular structure, underlying anatomical variations, and appropriateness of the selected treatment modality. Patient adherence, continuity of physiotherapy, and correction of postural habits may also affect long-term outcomes.

Current literature suggests that prognosis can be optimized not only through anatomical decompression but also through functional rehabilitation and a multidisciplinary approach. Accordingly, post-treatment follow-up plays an important role in improving outcomes.

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CHAPTER 4

CLINICAL ANATOMY AND PATHOLOGIES OF THE RADIAL NERVE

EMRE KAYGIN[1]

1. Introduction

The radial nerve (*nervus radialis*) is a mixed (motor and sensory) peripheral nerve arising from the posterior cord of the brachial plexus, typically containing fibers from the C5–T1 spinal nerve roots. It is the principal motor nerve of the extensor compartment of the upper limb and also provides cutaneous sensation to specific regions of the arm, forearm, and dorsum of the hand.

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The radial nerve accompanies the profunda brachii artery (*arteria profunda brachii*) and passes through the medial axillary space (*spatium axillare mediale*) to reach the posterior aspect of the arm. It courses deep to the triceps brachii muscle, spirals laterally around the humerus, and then pierces the lateral intermuscular septum (*septum intermusculare laterale*) to enter the anterior compartment of the arm.

At the level of the lateral epicondyle, the radial nerve divides into its two terminal branches: the superficial branch (*ramus superficialis*) and the deep branch (*ramus profundus*). Before this division, it supplies motor branches to the triceps brachii, anconeus, brachioradialis, brachialis, and extensor carpi radialis longus muscles.

The superficial branch of the radial nerve accompanies the radial artery (*arteria radialis*), courses distally along the forearm, and passes to the dorsal aspect of the wrist, where it runs superficially to the extensor retinaculum (*retinaculum extensorum*). It gives rise to the dorsal digital nerves (*nn. digitales dorsales*), supplying cutaneous sensation to the second through fifth digits and to the adjacent dorsal surfaces of the first and second digits.

The deep branch of the radial nerve, which continues as the posterior interosseous nerve, innervates the muscles of the posterior forearm, including the extensor digitorum, extensor carpi radialis brevis, extensor carpi ulnaris, extensor digiti minimi, extensor indicis, extensor pollicis longus, extensor pollicis brevis, and abductor pollicis longus.

The cutaneous branches of the radial nerve include:

- Posterior cutaneous nerve of the arm (*nervus cutaneus brachii posterior*), supplying the posterior aspect of the arm;

- Inferior lateral cutaneous nerve of the arm (*nervus cutaneus brachii lateralis inferior*), supplying the lateral aspect of the distal arm;
- Posterior cutaneous nerve of the forearm (*nervus cutaneus antebrachii posterior*), supplying the posterior surface of the forearm.

Table 1. Motor and sensory branches of the radial nerve and their innervation areas

Branch / Nerve	Type	Innervation / Sensory Distribution
Radial nerve (before division)	Motor	*Triceps brachii*, *anconeus*, *brachioradialis*, lateral part of *brachialis*, *extensor carpi radialis longus*
Deep branch (posterior interosseous nerve)	Motor	*Extensor carpi radialis brevis*, *extensor digitorum*, *extensor carpi ulnaris*, *extensor digiti minimi*, *extensor indicis*, *extensor pollicis longus*, *extensor pollicis brevis*, *abductor pollicis longus*
Posterior cutaneous nerve of the arm (n. cutaneus brachii posterior)	Sensory	Posterior surface of the arm
Inferior lateral cutaneous nerve of the arm (n. cutaneus brachii lateralis inferior)	Sensory	Lateral aspect of the distal half of the arm
Posterior cutaneous nerve of the forearm (n. cutaneus antebrachii posterior)	Sensory	Posterior surface of the forearm
Superficial branch of the radial nerve (ramus superficialis)	Sensory	Dorsum of the hand; dorsal surfaces of digits 2–5 and adjacent dorsal surfaces of digits 1 and 2

2. Axillary and Proximal Arm Level

In the axilla, the radial nerve (nervus radialis) lies posterior to the axillary artery (arteria axillaris) and courses between the humerus and the long head of the triceps brachii muscle. At this level, the nerve remains as a single trunk and has not yet divided into its terminal branches. The radial nerve begins to give off motor branches to the triceps brachii, particularly to the long head (caput longum) and medial head (caput mediale).

This anatomical configuration forms the basis of high-level radial nerve lesions, such as those observed in crutch palsy or prolonged axillary compression. Lesions at this level may result in significant impairment of elbow extension, reflecting involvement of the triceps brachii muscle.

3. Humeral Spiral Groove (Sulcus Nervi Radialis)

The radial nerve continues its course along the posterior surface of the humerus within the radial groove (sulcus nervi radialis), accompanied by the profunda brachii artery (arteria profunda brachii), and passes laterally around the humeral shaft. This segment represents one of the most clinically vulnerable portions of the radial nerve.

Fractures of the humeral diaphysis, particularly at the junction of the middle and distal thirds, may result in neurapraxia or more severe nerve injuries due to the close proximity of the nerve to the bone. At the level of the spiral groove, the radial nerve completes its motor innervation to the lateral head of the triceps brachii. Consequently, triceps function is often preserved in lesions at this level, whereas wrist and finger extension weakness is a prominent clinical finding.

4. Lateral Intermuscular Septum and Elbow Region

After leaving the spiral groove, the radial nerve pierces the lateral intermuscular septum and enters the anterior compartment of the arm. This transition zone represents a relatively fixed anatomical point, rendering the nerve susceptible to traction and compression injuries.

As the nerve approaches the elbow, it courses between the brachialis and brachioradialis muscles and divides at the level of the lateral epicondyle into two terminal branches: the predominantly motor deep branch (ramus profundus) and the purely sensory superficial branch (ramus superficialis). The exact level of bifurcation may vary among individuals, which is of particular importance during surgical procedures in this region.

5. Motor Branches and Innervation

The motor innervation of the radial nerve can be summarized in a proximal-to-distal sequence:

- Proximal arm: Innervation of all heads of the triceps brachii and the anconeus muscle.
- Spiral groove level: Innervation of the brachioradialis, extensor carpi radialis longus, and partial innervation of the extensor carpi radialis brevis.
- Forearm – deep branch (ramus profundus / posterior interosseous nerve): After passing through the supinator muscle, the nerve supplies the deep extensor muscles of the posterior compartment of the forearm, including the extensor digitorum, extensor digiti minimi, extensor carpi ulnaris, abductor pollicis longus, extensor pollicis longus, extensor pollicis brevis, and extensor indicis.

The clinical significance of this motor innervation is closely related to the dependence of grip strength on wrist extension. Paralysis of the wrist extensors results in a marked reduction in grasping ability and constitutes the basis of the classic “wrist drop” deformity.

6. Sensory Branches and Cutaneous Distribution

The superficial branch of the radial nerve (ramus superficialis nervi radialis) accompanies the radial artery in the distal half of the forearm. Near the wrist, it becomes superficial and passes dorsally to supply the radial aspect of the dorsum of the hand.

Its sensory distribution includes the dorsal surface of the thumb (excluding the nail bed), the dorsal surface of the index finger, and the radial half of the dorsal surface of the middle finger. During clinical examination, the first dorsal web space between the thumb and index finger is considered the most reliable area for assessing radial nerve sensory function.

7. Entrapment and Compression Sites

Due to its long and complex anatomical course, close relationship with osseous structures, and superficial passage at certain levels, the radial nerve is vulnerable to multiple sites of entrapment and compression. Mechanical pressure at these locations may disrupt nerve conduction and result in variable degrees of motor and/or sensory deficits.

Axilla

At the axillary level, the radial nerve courses between the humerus and the long head of the triceps brachii. Prolonged compression in this region predominantly affects motor fibers. Classic clinical examples include crutch palsy and Saturday night palsy, which may occur following improper use of crutches or prolonged arm abduction and extension associated with alcohol or sedative intoxication. Lesions at this level are typically characterized by extensive motor and sensory involvement.

Humeral Diaphysis – Radial Groove (Spiral Groove)

The close relationship of the radial nerve to the humerus within the spiral groove renders this region particularly susceptible to traumatic injuries. Humeral shaft fractures, tight casts or bandages, and repetitive microtrauma may lead to nerve injury at this level. Clinically, this represents one of the most common sites of radial nerve injury.

Lateral Epicondyle – Radial Tunnel

At the elbow, the radial nerve divides into the superficial and deep branches. Compression of the deep branch as it passes beneath the fibrous arch of the supinator muscle (Arcade of Frohse) constitutes the pathophysiological basis of posterior interosseous nerve syndrome. Compression in this region is commonly associated with repetitive pronation–supination movements, inflammatory processes, or anatomical variations.

Distal Forearm

In the distal forearm, the segment where the superficial branch of the radial nerve becomes subcutaneous is particularly vulnerable to external compression. Sensory symptoms caused by tight watches, bracelets, bandages, or casts are referred to as Wartenberg syndrome. As this branch is purely sensory, motor deficits are absent in this condition.

Table 2. Entrapment sites of the radial nerve and their anatomical relationships

Level of Entrapment	Affected Branch	Mechanism of Compression / Anatomical Structures	Clinical Significance
Axilla	Main trunk of the radial nerve	Compression between the humerus and the long head of the triceps brachii; proximity to the axillary artery (*arteria axillaris*)	Crutch palsy, Saturday night palsy
Radial groove (sulcus nervi radialis)	Main trunk of the radial nerve	Close contact with the humeral diaphysis; accompanied by the profunda brachii artery (*arteria profunda brachii*)	Humeral shaft fractures, tight casts or splints
Lateral intermuscular septum	Main trunk of the radial nerve	Transition from the posterior to the anterior compartment of the arm	Traction and compression injuries
Radial tunnel	Deep branch of the radial nerve / posterior interosseous nerve (PIN)	Fibrous arch of the supinator muscle (Arcade of Frohse), fibrous bands	Radial tunnel syndrome, posterior interosseous nerve syndrome
Distal forearm	Superficial branch of the radial nerve	Compression between the radius and fascia; external pressure from watches, bracelets, or tight bandages	Wartenberg syndrome (cheiralgia paresthetica)

8. Clinical Presentations and Findings According to the Level of Lesion

The clinical presentation of radial nerve lesions varies markedly depending on the anatomical level of injury and the type of nerve fibers involved (motor versus sensory). Therefore, in clinical evaluation, the pattern of motor dysfunction and the distribution of sensory deficits are of major diagnostic importance.

In axillary-level lesions, the radial nerve has not yet divided into its terminal branches; consequently, all extensor muscle groups, including the *m. triceps brachii*, are affected. Elbow extension is markedly weakened or completely lost. This is accompanied by extensive sensory loss, which may extend from the posterior aspect of the arm to the dorsum of the hand.

In lesions at the level of the radial groove (sulcus nervi radialis), the motor branches to the *m. triceps brachii* usually arise proximally and are therefore often spared. As a result, elbow extension is frequently preserved. In contrast, there is pronounced weakness or loss of wrist and finger extension. Clinically, the most common finding at this level is the characteristic appearance of wrist drop.

In posterior interosseous nerve lesions, no sensory deficit is observed because the nerve is purely motor. Severe weakness of finger and thumb extension predominates. Wrist extension is generally preserved but is often accompanied by radial deviation, which represents an important distinguishing clinical sign.

Lesions of the superficial branch (ramus superficialis) of the radial nerve present with complete preservation of motor function, while sensory symptoms predominate. Patients typically report paresthesia, burning sensations, and hypoesthesia over the dorsum of the hand. These sensory disturbances are usually localized to a limited area.

Crutch Palsy and Saturday Night Palsy

Anatomical basis:

In the axilla, the radial nerve courses posterior to the arteria axillaris, between the humerus and the long head of the m. triceps brachii. In this region, the nerve is relatively superficial and has limited protection from surrounding soft tissues.

Pathophysiology:

Crutch palsy develops as a result of prolonged compression of the radial nerve in the axilla, most commonly due to improperly sized or incorrectly used crutches.

Saturday night palsy occurs when the arm remains compressed against a hard surface (such as the edge of a chair or sofa) for an extended period during deep sleep, often following alcohol consumption.

These conditions are typically characterized by neuropraxia or mild axonotmesis.

Clinicalfindings:

- • Weakness of elbow extension, including involvement of the m. triceps brachii
- • Marked loss of wrist and finger extension (wrist drop)
- • Widespread sensory loss involving the posterior arm, posterior forearm, and dorsum of the hand

Prognosis:

Depending on the duration and severity of compression, most cases show spontaneous recovery within weeks to months with conservative management.

Lesions Associated with Humeral Shaft Fractures and Tight Casts

Anatomical basis:

The radial nerve lies in direct contact with bone within the sulcus nervi radialis along the humeral shaft. In this region, the nerve is highly vulnerable to injury from the periosteum and fracture fragments.

Pathophysiology:

In humeral diaphyseal fractures, the nerve may be subjected to direct laceration, traction, or entrapment between bone fragments.

Excessively tight casts, bandages, or tourniquets may produce secondary compression of the nerve.

Clinical findings:

- • Triceps function is usually preserved due to proximal branching
- • Loss of wrist and finger extension
- • Sensory impairment over the dorsum of the hand, particularly in the first dorsal web space

Clinical relevance:

In patients with humeral fractures, neurological examination must be performed both before and after fracture reduction. Early recognition of iatrogenic nerve injury is critical for timely surgical decision-making.

Traction and Compression Injuries

Anatomical basis:

Throughout its long course from the shoulder to the forearm, the radial nerve passes through several relatively fixed points, including the axilla, the radial groove, and the region around the lateral epicondyle. These fixation points render the nerve particularly susceptible to traction forces.

Pathophysiology:

- • Shoulder dislocations
- • High-energy trauma

- • Prolonged surgical positioning, especially in the lateral decubitus position
- These mechanisms may lead to axonal injury due to excessive nerve elongation.

Clinical spectrum:

- • Transient weakness in mild cases
- • Permanent motor and sensory deficits in severe cases

Radial Tunnel Syndrome

Anatomical basis:

The radial tunnel is a narrow anatomical passage where the deep branch of the radial nerve, the posterior interosseous nerve, passes beneath the fibrous arch of the m. supinator (Arcade of Frohse).

Pathophysiology:

Repetitive pronation–supination movements, muscle hypertrophy, or thickening of the fibrous arch may lead to chronic compression of the nerve.

Clinical findings:

- • Deep, dull pain in the proximal forearm distal to the lateral epicondyle
- • Motor weakness is usually mild or appears in later stages
- • No sensory deficit, as the posterior interosseous nerve is purely motor

Differential diagnosis:

Radial tunnel syndrome is frequently confused with lateral epicondylitis (tennis elbow); however, pain in radial tunnel syndrome is typically deeper and more diffuse.

Wartenberg Syndrome (Cheiralgia Paresthetica)

Anatomical basis:

The superficial branch of the radial nerve passes beneath the tendon of the m. brachioradialis in the distal forearm and then becomes superficial in the subcutaneous tissue.

Pathophysiology:

- • External compression from watches, bracelets, tight bandages, or handcuffs
- • Repetitive pronation–supination movements

Clinical findings:

- • Paresthesia, burning, and numbness over the dorsoradial aspect of the hand
- • No motor deficit
- • Symptoms may be exacerbated by wrist movements

Clinical significance:

The purely sensory nature of this entrapment neuropathy, with complete preservation of motor function, is a key distinguishing feature.

Wrist Drop

Wrist drop is a clinical condition characterized by the inability to extend the wrist, resulting in the hand hanging in flexion under the influence of gravity. This occurs due to dysfunction of the wrist extensor muscles innervated by the radial nerve, including the

m. extensor carpi radialis longus, m. extensor carpi radialis brevis, and m. extensor carpi ulnaris.

Pathophysiology:

Radial nerve injury may produce different pathophysiological mechanisms depending on the severity of structural damage to the nerve fibers:

- • Neuropraxia: transient conduction block with preserved axonal integrity
- • Axonotmesis: axonal damage with intact connective tissue sheaths
- • Neurotmesis: complete transection or severe destruction of the nerve

These injury types are the principal determinants of clinical severity and the duration of functional impairment.

Etiology and etiopathogenesis:

The most common causes of wrist drop are traumatic and compressive factors. Humeral shaft fractures, prolonged axillary compression, surgical interventions, and repetitive mechanical stress may disrupt nerve conduction, leading to loss of extensor muscle function. The pathogenesis involves ischemia due to mechanical compression, axonal degeneration, and myelin damage.

From a functional standpoint, loss of wrist extension also compromises the effective action of the finger flexors. This results in a marked reduction in grip strength and severe impairment of fine motor skills, significantly affecting daily activities and tasks requiring manual dexterity.

Table 3. Radial nerve pathologies and associated clinical presentations

Pathology / Level of Lesion	Motor Findings	Sensory Findings	Typical Clinical Presentation
Axillary-level lesion	Loss of elbow, wrist, and finger extension	Widespread dorsal sensory loss	High radial nerve palsy
Radial groove (sulcus nervi radialis) lesion	Loss of wrist and finger extension	Sensory loss over the radial aspect of the dorsum of the hand	Wrist drop
Radial tunnel / posterior interosseous nerve (PIN) lesion	Loss of finger and thumb extension	None	Pure motor palsy
Superficial branch lesion	None	Paresthesia over the dorsoradial hand	Wartenberg syndrome
Neuropraxia	Transient weakness	Transient sensory disturbance	Favorable prognosis
Axonotmesis / Neurotmesis	Persistent motor deficit	Variable	May require surgical intervention

9. Diagnostic Approach and Electrophysiological Evaluation (EMG–NCS)

In radial nerve lesions, clinical examination is often sufficient to establish the diagnosis; however, electrophysiological studies play a fundamental role in determining the level, severity, and type of nerve injury. Electromyography (EMG) and nerve conduction studies (NCS) are indispensable tools, particularly for prognostic assessment.

Nerve Conduction Studies (NCS)

Motor and sensory nerve conduction velocities and amplitude measurements are evaluated.

- Motor conduction: Reduced compound muscle action potential (CMAP) amplitudes suggest axonal damage, whereas prolonged distal latencies and slowed conduction velocities are indicative of demyelination.

- Sensory conduction: In cases involving the superficial branch of the radial nerve, decreased sensory nerve action potential (SNAP) amplitudes may be detected.
- Posterior interosseous nerve lesions: Sensory conduction studies are typically normal, as this nerve is purely motor.

Electromyography (EMG)

In the affected muscles, EMG assessment focuses on the presence of:

- Fibrillation potentials
- Positive sharp waves
- Changes in motor unit potential morphology

EMG is particularly critical for differentiating axonotmesis from neuropraxia. Normal EMG findings in the early phase favor a diagnosis of neuropraxia, whereas denervation findings appearing approximately 2–3 weeks after injury indicate axonal damage.

10. Treatment Approach

The management of radial nerve lesions is determined by the level of injury, etiology, type of nerve damage, and clinical severity. Treatment strategies are generally divided into conservative and surgical approaches.

Conservative Treatment

Conservative management is the first-line approach, especially in cases of neuropraxia and mild to moderate axonotmesis.

- Splinting to maintain the wrist and fingers in a functional position
- Physiotherapy aimed at preserving joint range of motion
- Exercise programs designed to prevent muscle atrophy

This approach aims to prevent functional deterioration in cases with a high potential for spontaneous recovery.

Surgical Treatment

Surgical intervention is considered under the following circumstances:

- Suspicion of neurotmesis
- Penetrating injuries
- Absence of clinical and electrophysiological recovery within 3–6 months
- Presence of tumors or persistent mechanical compression

Surgical options include nerve exploration, neurolysis, nerve grafting, and tendon transfer procedures. The choice of technique depends on the location and duration of the injury.

11. Prognosis and Recovery Patterns

Among peripheral nerve injuries, radial nerve lesions are generally associated with one of the most favorable prognoses. This is attributed to the nerve's broad motor reserve, functional synergies within the upper extremity, and a regenerative capacity conducive to recovery.

Prognostic Factors

Factors influencing prognosis include:

- Type of injury (neuropraxia > axonotmesis > neurotmesis)
- Level of the lesion
- Severity of trauma
- Patient age
- Associated vascular or skeletal injuries

Recovery Process

- Neuropraxia: Complete recovery within days to weeks
- Axonotmesis: Recovery may take months, depending on the rate of axonal regeneration
- Neurotmesis: Recovery is not expected without surgical intervention

Axonal regeneration proceeds from proximal to distal at an average rate of approximately 1–3 mm per day. Clinically, recovery typically begins in proximal muscle groups and progresses distally.

12. Clinical and Functional Outcomes

Functional recovery is not limited to the restoration of muscle strength alone. Reestablishment of wrist extension enables effective use of finger flexors, significantly improving grip strength and hand coordination.

In long-standing or untreated cases, the following complications may develop:

- Muscle atrophy
- Joint contractures
- Permanent functional impairment

Therefore, early diagnosis, appropriate treatment, and regular follow-up are crucial determinants of favorable functional outcomes.

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CHAPTER 5

CUBITAL TUNNEL SYNDROME

İSMAİL SİVRİ[1]

1. Introduction

Definition

Cubital tunnel syndrome (CuTS) is an entrapment neuropathy of the ulnar nerve at the elbow and represents the second most common peripheral nerve compression syndrome of the upper extremity, following carpal tunnel syndrome. The condition results from compression of the ulnar nerve within the fibro-osseous cubital tunnel located posterior to the medial epicondyle of the humerus.

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Clinically, CuTS is characterized by pain along the ulnar aspect of the hand and forearm, paresthesia or hypoesthesia in the fourth and fifth digits, and, in advanced stages, weakness and atrophy of the intrinsic hand muscles. Early symptoms are typically sensory, manifesting as intermittent numbness and tingling in the ulnar nerve distribution. As the condition progresses, patients may develop persistent sensory loss, impaired fine motor skills, and intrinsic muscle weakness.

Although the term *cubital tunnel syndrome* traditionally refers to compression of the ulnar nerve beneath the Osborne ligament, in contemporary clinical and radiological practice it is frequently used in a broader sense. In this context, CuTS encompasses all potential compression sites along the ulnar nerve from the arcade of Struthers proximally, through the cubital tunnel behind the medial epicondyle, and distally to the region between the humeral and ulnar heads of the flexor carpi ulnaris muscle.

Historical Background

Cubital tunnel syndrome was first described in 1958 by Feindel and Stratford as a mononeuropathy caused by compression of the ulnar nerve at the elbow. It is widely recognized as the second most common peripheral nerve entrapment syndrome of the upper limb.

The Osborne ligament, also referred to in the literature as the *Osborne fascia*, *Osborne band*, *humeroulnar aponeurotic arch*, or *cubital tunnel retinaculum*, represents the most frequent site of ulnar nerve compression at the elbow. In 1959, George Osborne described this ligament as forming the roof of the cubital tunnel, extending between the medial epicondyle and the olecranon.

Anatomical studies have reported the Osborne ligament to be approximately 2.2 cm in length and 4 mm in width. The ligament becomes progressively taut with elbow flexion, beginning at approximately 135 degrees and reaching maximal tension around 90 degrees, thereby reducing the volume of the cubital tunnel. This dynamic narrowing explains why CuTS symptoms, particularly numbness and paresthesia in the ulnar digits, are often exacerbated during elbow flexion and at night.

Epidemiology

Cubital tunnel syndrome is the second most common nerve compression disorder of the upper extremity after carpal tunnel syndrome. The reported prevalence in the general population ranges from approximately 1.9% to 5.8%, with an estimated annual incidence of 20 cases per 100,000 individuals.

The condition predominantly affects adults, with a typical age of onset between 40 and 50 years, and is rare in children and adolescents. The incidence is generally similar between sexes, although some studies report a slight predominance in females. Patients often present with multiple comorbidities, and CuTS frequently coexists with carpal tunnel syndrome, as well as cervical spine or shoulder disorders.

Recognized risk factors include obesity, smoking, diabetes mellitus, and occupational exposure to repetitive elbow flexion or vibration, particularly in manual labor. Dyslipidemia has also been suggested as a contributing factor to peripheral nerve compression disorders.

2. Anatomy

The ulnar nerve is a terminal continuation of the medial cord of the brachial plexus, receiving fibers primarily from C8 and T1, with occasional contribution from C7. It courses distally through the axilla medial to the axillary artery, then along the medial aspect of the arm without giving off major branches.

Approximately 10 cm proximal to the medial epicondyle, the ulnar nerve pierces the medial intermuscular septum and enters the posterior compartment of the arm. It then passes through the arcade of Struthers, a fibrous canal formed by the medial intermuscular septum and the fascial sheath of the medial head of the triceps brachii.

After traversing the arcade, the nerve passes posterior to the medial epicondyle, where it is subcutaneous and easily palpable, making it particularly vulnerable to trauma and compression. It then enters the cubital tunnel, whose boundaries are formed by the humeroulnar joint capsule and the aponeurotic arch connecting the humeral and ulnar heads of the flexor carpi ulnaris (FCU).

During full elbow flexion, the cross-sectional area of the cubital tunnel may decrease by up to 55%, a change associated with reduced ulnar nerve conduction velocity. Upon entering the forearm, the nerve passes between the two heads of the FCU and travels alongside the ulnar artery beneath the muscle.

In the forearm, the ulnar nerve innervates the flexor carpi ulnaris and the medial half of the flexor digitorum profundus, supplying the fourth and fifth digits. Approximately 5 cm proximal to the wrist, it gives off a dorsal cutaneous branch. At the wrist, the nerve passes through the Guyon canal, where it divides into superficial sensory and deep motor branches.

3. Etiology and Risk Factors

Cubital tunnel syndrome most commonly results from chronic compression of the ulnar nerve at the elbow, frequently considered idiopathic in origin. The most vulnerable site is the region beneath the Osborne ligament, where elbow flexion significantly increases intratunnel pressure and nerve strain.

Contributing factors include elbow trauma, post-traumatic deformities, heterotopic ossification, inflammatory joint diseases such as rheumatoid arthritis or osteoarthritis, and anatomical variations. Systemic risk factors include obesity, smoking, diabetes, and repetitive occupational activities involving prolonged elbow flexion.

4. Clinical Manifestations

Ulnar nerve dysfunction leads to characteristic intrinsic hand muscle paralysis, resulting in a claw hand deformity affecting the fourth and fifth digits. Weakness of the adductor pollicis produces a positive Froment sign, while impaired interossei function contributes to fine motor dysfunction and finger abduction/adduction weakness.

Sensory symptoms, numbness, paresthesia, and pain in the fourth and fifth digits, often precede motor deficits and are typically worse at night or during prolonged elbow flexion. As compression progresses, intrinsic muscle atrophy becomes evident, particularly in the first dorsal interosseous muscle. Additional clinical signs include Tinel's sign at the cubital tunnel and Wartenberg's sign, reflecting ulnar motor imbalance.

5. Diagnostic Evaluation

Diagnosis is primarily clinical, supported by nerve conduction studies (NCS) and electromyography (EMG). A conduction velocity below 50 m/s across the elbow or a reduction in compound muscle action potential amplitude greater than 20% supports the diagnosis.

High-resolution ultrasonography allows dynamic evaluation of the ulnar nerve during elbow movement, while magnetic resonance imaging (MRI) provides superior soft-tissue resolution, identifying nerve enlargement, signal changes, space-occupying lesions, and anatomical variants.

6. Treatment Strategies

Management depends on symptom severity. Conservative treatment, including activity modification, night splinting to limit elbow flexion, and ergonomic adjustments, is appropriate for mild cases. Corticosteroid injections may provide temporary symptom relief but do not address the underlying mechanical compression.

Surgical intervention is indicated for persistent symptoms, muscle weakness, or significant electrodiagnostic abnormalities. Surgical options include simple decompression and anterior transposition of the ulnar nerve. Simple decompression generally yields excellent results with fewer complications, while transposition is reserved for cases with nerve instability or recurrent symptoms.

7. Prognosis

Surgical outcomes are generally favorable in mild to moderate CuTS, with 60–85% of patients experiencing symptom improvement or resolution. Recovery may be gradual, extending up to 12 months postoperatively. Poor prognostic factors include long-standing compression, severe motor involvement, absent sensory nerve action potentials, and advanced age. Delayed diagnosis and inadequate decompression are common causes of surgical failure.

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CHAPTER 6

CARPAL TUNNEL SYNDROME (CTS)

FURKAN MEHMET ÖZDEN[1]

1. Introduction

Definition

Carpal Tunnel Syndrome (CTS) is a symptomatic compression neuropathy caused by entrapment of the median nerve (nervus medianus) within the carpal canal (carpal tunnel) at the level of the wrist. It is the most common entrapment neuropathy and accounts for approximately 90% of all entrapment neuropathies. The syndrome is characterized by pain, numbness, tingling in the hand, and, at advanced stages, decline in hand function.

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Historical background

The condition was first described in 1854 by Sir James Paget. However, its modern clinical definition and significance became clearer through Phalen's studies in the 1950s.

Epidemiology

CTS affects approximately 3.8% to 5% of the general population. Its incidence may reach 276 cases per 100,000 person-years. It is more common in women than men (approximately 2:1 or 3:1) and typically peaks between 40 and 60 years of age. CTS also represents a substantial proportion of work-related musculoskeletal disorders.

2. Hand Anatomy

Overview and skeletal structure

The hand (manus) is a complex structure located distal to the forearm (antebrachium) and enables functions such as grasping, holding, and pointing. Its skeletal framework consists of three main parts: the wrist (carpus), the metacarpus, and the digits (digiti/phalanges).

Bones (osteology)

Carpal bones (ossa carpi)

The wrist (carpus) is composed of eight bones, arranged in two rows (proximal and distal).

Proximal row (lateral to medial):

- Scaphoid (os scaphoideum): boat-shaped; articulates with the radius; palpable in the anatomical snuffbox (fovea radialis).
- Lunate (os lunatum): crescent-shaped.

- Triquetrum (os triquetrum): pyramid-shaped.
- Pisiform (os pisiforme): pea-shaped; located on the palmar surface of the triquetrum.

Distal row (lateral to medial):

- Trapezium (os trapezium): articulates with the first metacarpal (os metacarpale I).
- Trapezoid (os trapezoideum): wedge-shaped.
- Capitate (os capitatum): largest carpal bone; has a head-shaped proximal end.
- Hamate (os hamatum): has a hook-like projection (hamulus ossis hamati).

Metacarpals and phalanges

The palm contains five metacarpal bones (ossa metacarpalia). The first metacarpal is the shortest and thickest. The thumb (pollex) has two phalanges (proximal and distal), whereas the other fingers have three (proximal, middle, distal).

3. Fascia and compartments of the hand

Palmar aponeurosis (aponeurosis palmaris)

A thick, strong, triangular fascia covering the palm. Its apex continues with the flexor retinaculum (retinaculum musculorum flexorum) and the palmaris longus tendon.

Compartments

Intrinsic muscles of the hand are arranged in five compartments:

- Thenar compartment: forms the thenar eminence at the base of the thumb.

- Hypothenar compartment: forms the hypothenar eminence at the base of the little finger.
- Central compartment: contains flexor tendons and lumbricals.
- Adductor compartment: deep; contains the adductor pollicis.
- Interosseous compartment: between metacarpals.

4. Hand muscles and movements

Hand movements are produced by long forearm tendons (extrinsic) and small intrinsic hand muscles.

Intrinsic muscles

Thenar muscles (thumb movements):

- Abductor pollicis brevis (abduction)
- Flexor pollicis brevis (flexion)
- Opponens pollicis (opposition—the most complex and functionally important thumb movement)

Hypothenar muscles (little finger movements):

- Abductor digiti minimi (abduction)
- Flexor digiti minimi brevis (flexion)
- Opponens digiti minimi (opposition)

Short muscles:

- Lumbricals: four worm-like muscles; flex the metacarpophalangeal joints and extend the interphalangeal joints.

- Interossei: dorsal interossei abduct the fingers; palmar interossei adduct the fingers.

5. Neurovascular structures

Nerves

- Median nerve (nervus medianus): passes through the carpal tunnel; innervates most thenar muscles (except adductor pollicis and deep head of flexor pollicis brevis) and the first two lumbricals; provides sensation to the lateral 3.5 digits.
- Ulnar nerve (nervus ulnaris): passes through Guyon's canal (canalis ulnaris); responsible for fine motor control; innervates most intrinsic hand muscles.
- Radial nerve (nervus radialis): has no motor function in the hand; provides sensation to the lateral dorsum of the hand.

Blood supply

Hand perfusion is mainly via the superficial palmar arch (predominantly from the ulnar artery) and the deep palmar arch (predominantly from the radial artery).

6. Anatomy of the carpal tunnel (canalis carpi)

The carpal tunnel is a passage on the palmar aspect of the wrist formed by bones and ligaments.

- Floor and side walls: formed by the carpal bones (creating a concave arch).
- Lateral boundary: tubercles of scaphoid and trapezium.
- Medial boundary: pisiform and hook of hamate.

- Roof: flexor retinaculum (transverse carpal ligament), which spans the carpal arch and forms the tunnel.

Contents of the tunnel

The tunnel contains 9 tendons and 1 nerve:

- Median nerve (most vulnerable structure)
- Four FDS tendons (flexor digitorum superficialis)
- Four FDP tendons (flexor digitorum profundus)
- One FPL tendon (flexor pollicis longus), in its own synovial sheath
  Important note: The flexor carpi radialis (FCR) tendon passes within the layers of the flexor retinaculum but does not pass through the carpal tunnel (it has a separate tunnel). The ulnar nerve and ulnar artery travel outside the carpal tunnel through Guyon's canal.

Biomechanics

Wrist position markedly changes intracarpal pressure. Normal tunnel pressure is 2–10 mmHg. Wrist extension can increase pressure up to 10-fold, and wrist flexion up to 8-fold.

7. Etiology and risk factors

Most CTS cases are idiopathic, but contributing factors can be grouped as systemic, local/anatomic, and occupational/environmental.

Systemic factors

- Hypothyroidism: modest association; may increase risk of requiring surgery.
- Diabetes: increased susceptibility to neuropathy; tendon glycosylation contributes.

- Obesity: increased fat volume and hydrostatic pressure changes raise risk.
- Pregnancy: common in the third trimester due to edema and hormonal changes; often resolves postpartum.
- Rheumatoid arthritis: synovial hypertrophy (pannus) narrows tunnel volume.

Local/anatomic factors

- Distal radius fractures: may cause acute or delayed CTS; incidence is higher after surgically treated fractures.
- Wrist shape: "square" wrist morphology (high depth/width ratio) increases risk.
- Space-occupying lesions: ganglion cysts, tumors, or abnormal muscle anatomy (e.g., lumbricals entering the tunnel).

Occupational/environmental factors

Repetitive wrist/hand motions, vibration tools, and prolonged wrist flexion/extension increase risk. The relationship between computer use (keyboard/mouse) and CTS remains controversial, with weak evidence.

8. Clinical staging

CTS is commonly described in three stages:

- Stage 1 (early): nocturnal awakening, relief by shaking the hand (flick sign), morning stiffness.
- Stage 2 (moderate): daytime symptoms begin; numbness worsens during repetitive tasks or sustained positions (e.g., phone use, driving); clumsiness and dropping objects may occur.

- Stage 3 (advanced): thenar atrophy is evident; sensory symptoms may become constant numbness, and pain may decrease due to severe nerve fiber damage.

9. Clinical findings and pathophysiology

CTS results from any process that reduces tunnel size or increases contents volume (e.g., synovial inflammation, fluid retention).

Median nerve compression findings

Sensory disturbance: paresthesia/hypoesthesia in the lateral 3.5 digits (thumb, index, middle, and radial half of ring finger). Spared area: central palm sensation is typically preserved because the palmar cutaneous branch of the median nerve passes outside the carpal tunnel over the flexor retinaculum.

Motor deficit and weakness

The median nerve recurrent motor branch supplies thenar muscles (abductor pollicis brevis, opponens pollicis, and superficial head of flexor pollicis brevis). Persistent compression causes thumb weakness and impaired coordination.

"Manus simia" (ape hand) deformity: due to opponens pollicis paralysis, thumb opposition is lost, the thumb lies in the plane of the palm, and thenar eminence atrophies.

Typical symptoms

- Numbness/tingling in thumb, index, middle, and half of ring finger; the little finger is usually spared (ulnar nerve).
- Nocturnal pain and awakening, often related to wrist flexion during sleep and fluid redistribution.
- Reduced grip strength and loss of fine hand function.

Examination findings

- Thenar atrophy in severe chronic cases (especially abductor pollicis brevis).
- Sensory loss on two-point discrimination or monofilament testing in affected digits.

10. Diagnostic methods

Diagnosis starts with detailed history and physical examination and is supported by confirmatory tests when needed.

Provocative tests

- Phalen test: full wrist flexion for 60 seconds; positive if paresthesia occurs (sensitivity ~42–85%).
- Tinel sign: tapping over the median nerve produces an "electric shock" sensation (sensitivity ~38–100%).
- Durkan test (carpal compression): direct compression over the carpal tunnel for ~30 seconds reproduces symptoms.
- CTS-6 score: validated clinical tool (symptom distribution, nocturnal symptoms, thenar atrophy, Phalen, Tinel, two-point discrimination loss).

Electrodiagnostic tests (EDX)

Nerve conduction studies (NCS) and electromyography (EMG) are considered the diagnostic gold standard, demonstrating slowed median sensory and motor conduction. In clinically obvious cases (high CTS-6), EDX may not substantially change diagnostic probability.

Imaging

- Ultrasound: shows increased median nerve cross-sectional area (CSA); >9–10 mm^2 at the pisiform level supports diagnosis.
- MRI: not routine; useful when a space-occupying lesion (tumor, ganglion) is suspected.

11. Differential diagnosis

Conditions to exclude include:

- Cervical radiculopathy (C6–C7): neck pain and radiating arm pain.
- Pronator syndrome: median nerve entrapment in the forearm; palmar sensation may be affected (unlike CTS).
- Thoracic outlet syndrome: brachial plexus compression.
- Peripheral polyneuropathy: e.g., diabetic glove-and-stocking sensory loss.
- CMC arthritis: pain at the base of the thumb worsens with movement.

12. Treatment

Treatment depends on disease severity.

A. Conservative management

First-line for mild to moderate cases:

- Neutral wrist splinting: minimizes tunnel pressure; especially recommended at night.
- Corticosteroid injections: reduce edema and inflammation with temporary benefit (often ~1–3 months).

- Medications: short-term oral steroids may help; routine NSAID use is not recommended.
- Physical therapy/exercises: nerve and tendon gliding may reduce symptoms; ultrasound and laser therapies are used but evidence is variable.

B. Surgical treatment

Indicated when conservative therapy fails, symptoms are severe, or thenar atrophy/motor loss develops.

- Principle: division of the flexor retinaculum (transverse carpal ligament) to enlarge tunnel volume and relieve pressure.
- Open release: standard incision with direct visualization.
- Endoscopic release: smaller incision; potentially faster return to work; complication risk depends on surgeon experience.
  Surgery is more effective than conservative therapy for long-term symptom relief.

13. Prognosis and follow-up

About one-third of patients may improve spontaneously without treatment. After surgery, 70–90% achieve good-to-excellent outcomes; nocturnal pain often resolves quickly. Possible complications include postoperative pillar pain, temporary grip weakness, or recurrence due to incomplete release. Recovery may be less favorable in diabetics or in cases with severe nerve injury.

14. Conclusion

CTS is a condition that can significantly impair quality of life and may arise from anatomical compression, systemic disease, or occupational strain. Diagnosis relies primarily on careful history and examination (particularly CTS-6 criteria) and may be supported by EMG and ultrasound when needed. Treatment ranges from splinting to surgical decompression depending on stage and severity. Early recognition and timely intervention, especially in high-risk patients (e.g., diabetes, hypothyroidism) or after distal radius fracture, are essential to prevent permanent median nerve damage.

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