

Brachial Plexus Injuries

Goals & Objectives

Course Description

Brachial Plexus Injuries is an asynchronous text-based online continuing education program for physical therapists and physical therapist assistants. The course presents contemporary information about brachial plexus pathologies including sections on anatomy, epidemiology, etiology, symptomology, assessment, diagnosis, evaluation, and treatment.

Course Rationale

The purpose of this course is to present contemporary information about brachial plexopathies to physical therapists and physical therapist assistants. Physical therapists and physical therapist assistants will find this information pertinent and useful when developing and implementing rehabilitation programs that address the challenges and needs specific to individuals with brachial plexus injuries.

Course Goals & Objectives

At the end of this course, the participants will be able to:

1. Identify the anatomical structures of the brachial plexus.
2. Identify the etiologies that cause brachial plexopathies.
3. Classify brachial plexopathies utilizing standardized systems.
4. Define Wallerian degeneration and nerve regeneration.
5. Recognize the clinical presentation of brachial plexopathies.
6. Identify the electrophysiological & imaging tools commonly used to assess brachial plexopathies.
7. Identify non-surgical and surgical interventions for treating brachial plexopathies.
8. Define thoracic outlet syndrome (TOS)
9. Identify causative factors for thoracic outlet syndrome.
10. Identify the clinical tests utilized to assess for TOS
11. Identify non-surgical and surgical interventions for treating TOS
12. Identify the etiology and risk factors for obstetrical brachial plexus injury
13. Recognize and classify clinical symptomology associated with OBPI

Course Provider – Innovative Educational Services

Course Instructor - Michael Niss, DPT

Target Audience – physical therapists and physical therapist assistants

Course Educational Level – Introductory / intermediate

Course Prerequisites – None

Method of Instruction/Availability – Online text-based course available continuously.

Criteria for Issuance of CE Credits - A score of 70% or greater on the course post-test

Continuing Education Credits – 3 hours

Determination of Credits – Mergener Formula: $.9 \times [-22.3 + (0.00209 \times 62,958) + (2.78 \times 15) + (15.5 \times 3)] = 180$ minutes = 3.0 hours

Fees - \$29.95

Conflict of Interest – No conflict of interest exists for the presenter or provider of this course.

Refund Policy - Unrestricted 100% refund upon request. The request for a refund by the learner shall be honored in full without penalty or other consideration of any kind. The request for a refund may be made by the learner at any time without limitations before, during, or after course participation.

Brachial Plexus Injuries

Course Outline

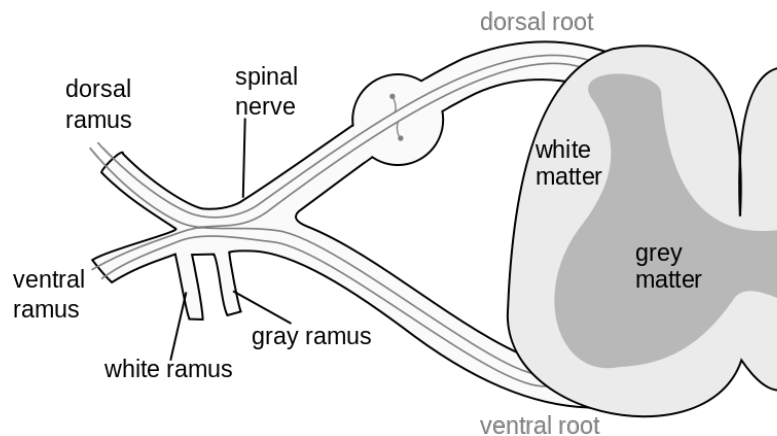
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Introduction

Society has seen a steady increase in brachial plexus injuries in the last half century. Many believe that this is due to advances in high-speed transportation technologies as well as increased participation in recreational activities worldwide. Developments in medicine, diagnostics, surgery, and rehabilitation now offer us new modalities to improve the clinical outcome of individuals with brachial plexus lesions.

Anatomy

To fully understand the different pathological processes involving the brachial plexus, it is necessary to have a good understanding of the developmental, structural, and functional anatomy of the brachial plexus and peripheral nerves. The peripheral nervous system develops from the neural crest cells, which form the dorsal nerve roots (sensory), and from the cells in the basal plates of the developing spinal cord, which form the ventral nerve roots (motor). Both nerve roots unite to form the mixed spinal nerve root that immediately divides into the dorsal and ventral primary rami.¹

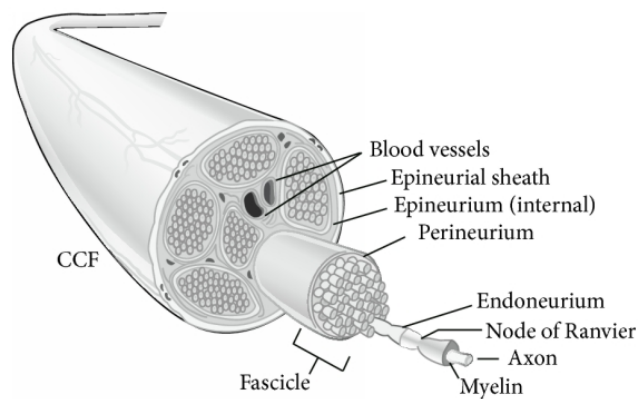
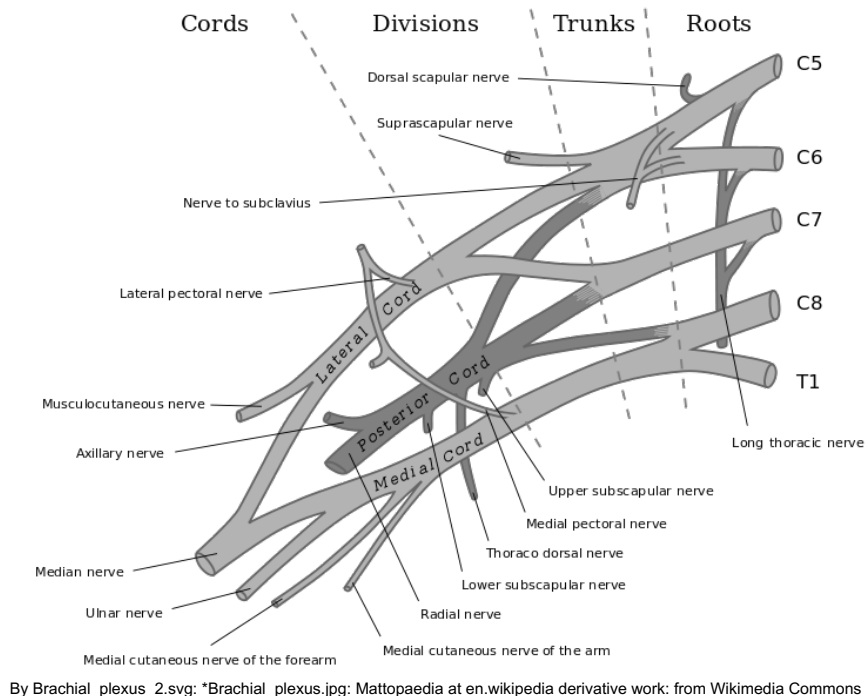


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The brachial plexus is formed in the posterior cervical triangle by the union of ventral rami of 5th, 6th, 7th, and 8th cervical nerve roots and 1st thoracic nerve root. This composite nerve network can be divided into roots, trunks, divisions, and cords. The roots, trunks, and divisions lie in the posterior triangle of the neck, whereas the cords lie in the axillary fossa. Cords are further divided in the major nerve branches of the upper extremity.²

Roots and trunks lie in the supraclavicular space; the divisions are located posterior to the clavicle, while cords and branches lie infraclavicularly. All three cords of the plexus lie above and laterally to the medial portion of axillary artery.²

Brachial Plexus Injuries



The endoneurium surrounds individual myelinated axons and groups of unmyelinated ones. Fascicles are collections of axons which are surrounded by perineurium. Myelinated peripheral nerve fibers are surrounded by Schwann cells. Every nerve fiber with the accompanying Schwann cells is surrounded by a layer of delicate connective tissue, called endoneurium.²

The peripheral nerve trunk is a collection of fascicles, and the epineurial (external) epineurium surrounds the nerve trunk proper. The endoneurium is longitudinally oriented while the perineurium and epineurium are circumferential. Plexuses of microvessels run longitudinally in the epineurium, and send transverse branches

through the perineurium to form a vascular network consisting primarily of capillaries in the endoneurium. Nerve trauma increases the permeability of the epineurial vessels, which are more susceptible to compression trauma than the endoneurial vessels. Higher pressure levels or more prolonged compression will also injure the endoneurial vessels, leading to intrafascicular edema, which may lead to secondary injury to the nerve.³

Etiology

Generally, lesions of the brachial plexus have a mechanical, metabolic, infectious, immunologic, pharmaceutical, radiation therapy, or neoplastic cause.⁴

Mechanical

Trauma

Most brachial plexus lesions result from a trauma to the neck and shoulder, often in form of stretch, compression, disruption, or tear injuries. Occasionally, traumatic plexus lesions may be associated with root avulsion. Causes of traumatic plexus lesions include motor cycle accidents, automobile crashes (seatbelt trauma, where usually the plexus of the non-fixated side is affected), falls, bicycle-related trauma, penetrating wounds (gunshots), or clavicle fracture during delivery. Traumatic plexus lesions can affect any part of the plexus and are classified as open or closed, upper or lower, supra- or infraclavicular, and as complete or incomplete. Traumatic injury of the brachial plexus can be devastating, resulting in partial or complete denervation of plexus-innervated muscles.⁴

Compression/traction

Compression of the brachial plexus may be due to extrinsic causes (e.g. positioning during anesthesia, obstetric brachial plexus palsy), or intrinsic causes (TOS, tumor growth, infection, aneurysm, posttraumatic pseudoaneurysms, scapular-thoracic dissociation).⁴

Extrinsic causes - Inadequate positioning during anesthesia: The most frequent cause of compression injuries is inadequate positioning during surgery, such as abduction of both arms and direct compression of the shoulder girdle. Inappropriate tear on nerves during muscle relaxation may be contributory. Steep head-down tilt position during urological surgery for example has been shown to cause injury of the upper and middle brachial plexus trunks. Patients are seated with the left arm abducted to approximately 90° and the right arm adducted. To prevent sliding of the patient on the operating table and secure patients, shoulders are supported with moldable beanbags. This results in injurious stretching, tearing or compression of the brachial plexus, particularly of the upper and middle trunks.⁴

Intrinsic Causes - Arteriopathy of the subclavian artery: Pseudoaneurysm of the subclavian artery may develop after fracture of the clavicle, trauma, balloon angioplasty,

or surgery. Pseudoaneurysm of the subclavian artery may cause compression of the brachial plexus resulting in upper extremity weakness and wasting.⁴

Metabolic

Diabetes is the most frequent metabolic cause of a brachial plexopathy. Diabetes may cause diabetic brachial plexus neuropathy, which is predominantly a monophasic, upper limb neuropathy with pain followed by weakness and involvement of the motor, sensory, and autonomic fibers. Diabetic brachial plexopathy is infrequent, compared with lumbosacral plexopathy. Diabetic brachial plexus neuropathy may begin focally and evolve into a multifocal or bilateral condition. Bilateral plexopathy can sometimes be the initial presentation of diabetes. Ischemic injury from microvasculitis has been proposed as possible pathogenetic concept of diabetic plexopathy.⁴

Infectious

Infectious diseases directly affecting the brachial plexus include HIV or Epstein-Barr virus. Post-infectious plexopathy may be due to herpes simplex, Dengue virus, hepatitis E, or herpes zoster. Possibly associated with plexopathy are infections by parvovirus or West Nile virus. Herpes zoster is the most frequent infectious cause of brachial plexopathy. Patients with herpes zoster plexopathy present with monoparesis, hyperalgesia, allodynia (pain through stimulus, which normally does not provoke pain), edema, color and skin-temperature changes, and skin eruptions. Herpes zoster, Dengue fever, and HIV infections may trigger pain and may be due to spreading of inflammation from the dorsal root ganglia to the ventral roots. HIV infections may cause brachial plexus neuritis, an immuno-mediated inflammatory reaction resulting in acute onset of shoulder pain, followed by weakness and wasting. Though viruses much more frequently cause plexus lesions compared to bacteria, some patients develop plexopathy due to infection with *Mycobacterium leprae* or *Ehrlichia canis*.⁴

Immunological

Although relatively rare, plexopathy of the brachial plexus has been reported in some patients receiving vaccination against influenza, hepatitis B, smallpox, human papilloma virus (HPV), or swine flu. The basis of the post-vaccination plexus lesion may be immune complex deposition at the nerve sheaths or vasculitis of the small arteries supplying the plexus network.⁴

Pharmacological

Infliximab, a chimeric monoclonal antibody, given for refractory rheumatoid arthritis or Crohn's disease, may be associated with brachial plexopathy. Chemotherapy with cisplatin or vinblastine may also cause plexopathy. Additionally, neuralgic amyotrophy may be caused by etanercept, which binds TNF-alpha, and is used as an antirheumatic agent but also in the treatment of psoriasis. Rarely, brachial plexus lesions may be caused by high thoracic paravertebral block, carried out for unilateral chest or

abdominal surgery. Each of these drug-induced plexopathies seem to be related to hyper-immune responses occurring in the body.⁴

Radiation

Radiation therapy may cause acute or subacute radiation syndromes. The most frequent radiation-induced complication affecting the brachial plexus is delayed radiation-induced brachial plexopathy, which may follow breast cancer or lung cancer radiation. The pathophysiology of radiation-induced nerve damage is not fully understood but there are indications that radiation causes neurofibrosis or ischemia.⁴

Neoplastic

Cancer may affect the plexus via a lymph node, direct infiltration, or may spread along the nerve roots to cause spinal compression. Direct infiltration of the brachial plexus is most frequently caused by invasive breast or lung cancer, B-cell lymphoma, or thyroid cancer. In neoplastic plexopathy, pain is frequently the presenting symptom. Benign tumors causing brachial plexus lesions include schwannoma, neurofibroma, meningioma, desmoid tumor, lipoma, or benign granular cell tumors.⁴

Idiopathic

The most frequent of the idiopathic plexopathies is Parsonage-Turner syndrome (PTS). PTS is a painful brachial plexopathy, clinically characterized by shoulder girdle pain of acute or subacute onset lasting 1-4 weeks. Typically, this stabbing pain is worse at night. When pain subsides, it is usually followed by episodes of polytope weakness, occasionally wasting, and sensory disturbances in a brachial plexus distribution with potential concomitant involvement of the lumbosacral plexus or the phrenic nerve. Both idiopathic and hereditary forms exist. It occurs in males two times more frequently than females. Trigger factors for PTS are previous mild infection, surgery, remote trauma, or vaccination.⁴

Lesion Location

Determination of the distance of the level of injury from the spinal cord offers very important information. In case of a preganglionic injury, the nerve is avulsed from spinal cord, separating motor neurons from the motor centers of the ventral horns of the spinal cord. Sensory neurons remain intact at the level of dorsal root ganglion, which explains why sensory nerve action potentials are preserved in preganglionic lesions. Preganglionic lesions are not repairable and alternative working motor nerves need to be transferred in order to restore part of the functionality of the upper limb. Contrarily, postganglionic lesions may be restored spontaneously or may be repaired surgically. It is not uncommon for both preganglionic and postganglionic injuries to coexist.²

Pre-ganglionic injuries

- Spinal roots are avulsed from the spinal cord
- Loss of motor function only

Post-ganglionic injuries

- Occur distal to the dorsal root ganglion
- Loss of both sensory and motor functions.

Clinically, brachial plexus injuries can be categorized as either upper plexus or lower plexus injuries. Brachial plexus injuries (BPIs) most commonly affect the supraclavicular zone. Infraclavicular and retroclavicular lesions are less common. Roots and trunks get more easily affected compared to cords and terminal branches.².

Epidemiology

Just over half of all adult brachial plexus injuries occur between the ages of 19 and 34 years old. Eighty-nine percent are male.

Narakas⁵ developed a rule of "seven seventies" that gives an approximate idea of the statistics involved in brachial plexus lesions:

- Approximately 70% of traumatic brachial plexus injuries are secondary to motor vehicle accidents; of these,
- Approximately 70% involve motorcycles or bicycles.
- Of the cycle riders, approximately 70% have multiple injuries.
- Overall, 70% have supraclavicular lesions;
- Of these, 70% have at least one root avulsion.
- At least 70% of patients with a root avulsion also have avulsions of the lower roots (C7, C8 or T1).
- Finally, of patients with lower root avulsion, nearly 70% will experience persistent pain.

Prognosis

The outcome of a traumatic brachial plexus injury is dependent upon many factors; primarily the patient's age, the type of the injured nerve, the level of injury, the time of surgical intervention, and concomitant diseases.²

- Age - Better prognosis in young patients
- Mechanism of injury - High energy injuries have worse prognosis. Avulsion injuries have worse prognosis than acute ruptures. Worse prognosis with concomitant vascular injury.
- Type of nerve - Exclusively sensory or motor nerves have better functional recovery than mixed nerves.
- Level of injury - Supraclavicular lesions have worse prognosis than infraclavicular. Upper trunk lesions have the best prognosis.

- Pain - Patients with persistent pain for more than 6 months after trauma have less possibilities for recovery.
- Time of surgical intervention - Fibrosis and degeneration of target organs at the time of surgical intervention are related to poor prognosis.
- Other factors - Concomitant diseases (infections, etc.) are related to worse prognosis

Classification

There are two commonly used classification schemes for peripheral nerve injury: the Seddon and the Sunderland systems. The Sunderland classification is more complex, but many consider it to be more useful.

Seddon Classification

The Seddon classification system describes three groups of nerve injuries: neurapraxia, axonotmesis, and neurotmesis.

Neurapraxia

Neurapraxia, is the mildest form of nerve injury. Axons are anatomically intact but nonfunctional; the nerve cannot transmit impulses and the body part is paralyzed. There is motor and sensory loss due to demyelination, without axon disruption or Wallerian degeneration. The conduction block due to neurapraxia usually affects motor fibers more than sensory fibers. Clinically, muscle atrophy does not develop except for mild atrophy due to disuse. Electrophysiologically, the nerve conducts normally distally but there is impaired conduction across the lesion because of focal demyelination. Loss of function persists until remyelination occurs. Recovery time ranges from hours to a few months; full function can usually be expected without intervention by about 12 weeks, often earlier, provided there is no ongoing compression. Motor paralysis can last as long as 6 months, but most lesions resolve by 3 months. Since axons may be remyelinated at different rates and to different degrees, function may be regained unevenly. Seddon coined the term neurapraxia to refer to lesions that recovered in weeks to months.³

Axonotmesis

In axonotmesis (tmesis = to cut), the axial continuity of some individual nerve fibers is interrupted, but perineurium and epineurium are preserved.² The axon is disrupted and Wallerian degeneration occurs. There is axon discontinuity, but the surrounding stroma is at least partially intact. Axonotmesis is commonly seen in crush and stretch injuries. Reinnervation depends upon the degree of internal disorganization, and the distance to the muscle. In neurotmesis, the nerve is completely severed or so internally disrupted that it does not regenerate spontaneously well enough to produce function.³

Typically, there is a complete motor and sensory loss. However, spontaneous recovery along the intact endoneurial tubes may occur if the distance from the injury site to the

nerve ending is not too great. Axon regrowth occurs at a rate of approximately 1 mm per day under ideal conditions.

Neurotmesis

In neurotmesis, which is the most severe grade of injury, the nerve trunk is completely disrupted and not in anatomical continuity. Most of the connective tissue framework is lost or badly distorted.³ Spontaneous recovery of the affected nerve axon cannot be expected. Without surgical intervention, this kind of injury may lead to the creation of a nonfunctional neuroma.²

Neurotmesis is typically seen with sharp injury, massive trauma, or severe traction with nerve rupture. There is loss of nerve trunk continuity with complete disruption of all supporting elements; reinnervation does not occur. Without surgery, the prognosis is extremely poor. Recovery is apt to be prolonged and incomplete. The affected body part can seldom become again what it was.³

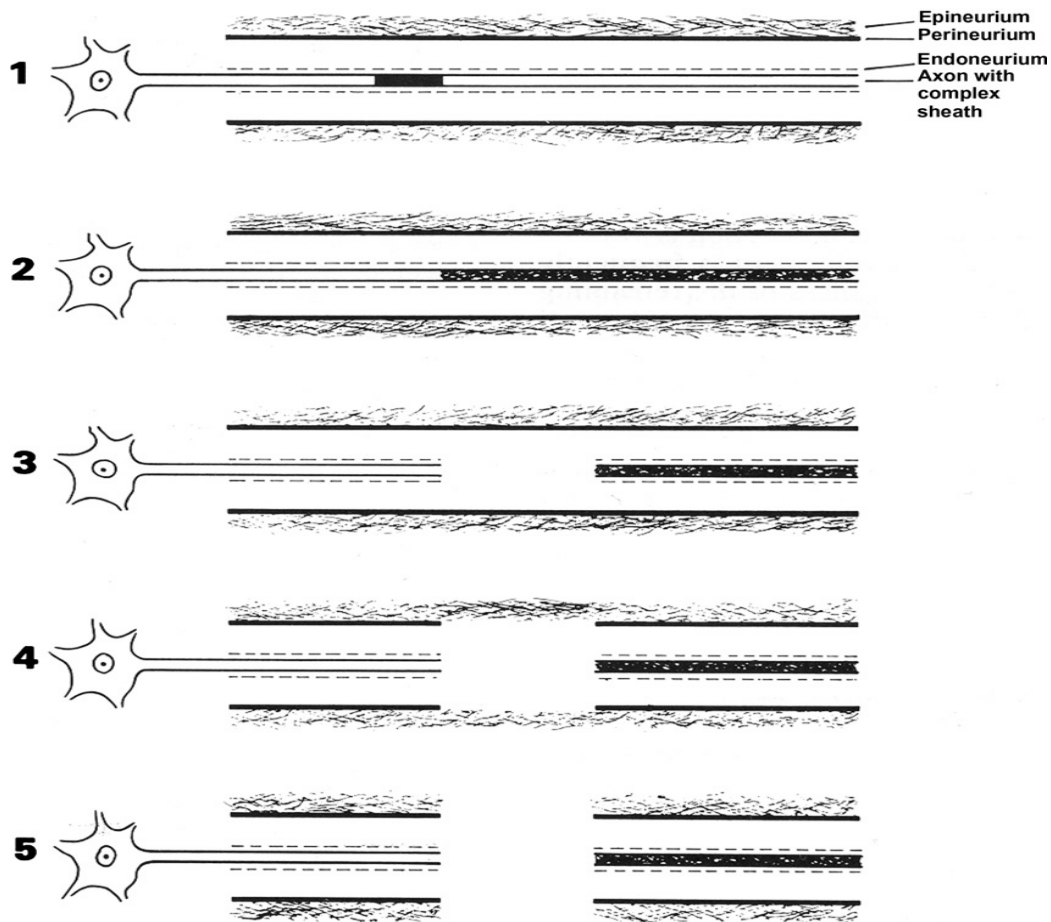
Sunderland Classification

Sunderland expanded Seddon's classification to five degrees of peripheral nerve injury:

- **First-degree (I)** - Seddon's neurapraxia and first-degree are the same.
- **Second-degree (II)** - Seddon's axonotmesis and second-degree are the same.
- **Third-degree (III)** - Sunderland's third-degree is a nerve fiber interruption. In third-degree injury, there is a lesion of the endoneurium, but the epineurium and perineurium remain intact. Recovery from a third-degree injury is possible, but surgical intervention may be required.
- **Fourth-degree (IV)** - In fourth-degree injury, only the epineurium remain intact. In this case, surgical repair is required.
- **Fifth-degree (V)** - Fifth-degree lesion is a complete transection of the nerve. Recovery is not possible without an appropriate surgical treatment.

Below is a diagrammatic representation of the five degrees of nerve injury as defined by the Sunderland classification system.³

Brachial Plexus Injuries



Modified from Sunderland S. Nerves and nerve injuries, 2nd ed. Baltimore: ***Williams and Wilkins, 1978.

1. Segmental demyelination causing conduction block but no damage to the axon and no Wallerian degeneration.
2. Damage to the axon severe enough to cause Wallerian degeneration and denervation of the target organ, but with an intact endoneurium and good prospects for axon regeneration.
3. Disruption of the axon and its endoneurial sheath inside an intact perineurium, loss of integrity of the endoneurial tubes will limit axon regeneration.
4. Disruption of the fasciculi, with nerve trunk continuity maintained only by epineurial tissue, severe limitation of axon regeneration, formation of a mass of misdirected axons (neuroma in continuity).
5. Transaction of the entire nerve trunk.

Sunderland	Seddon	Description	Recovery	Recovery Rate	Surgery
I	Neurapraxia	Temporary interruption to nerve transmission with restoration in weeks.	Complete	Fast (days-12 weeks)	None
II	Axonotmesis	Complete disruption of nerve transmission (axon) with regeneration and full recovery	Complete	Slow (3 cm/month)	None
III	Axonotmesis	Disruption of axon and connective tissue (endoneurium) causing disorganized regeneration	Varies	Slow (3 cm/month)	Varies
IV	Axonotmesis	Disruption of axon, endoneurium, and perineurium, with intact epineurium; but no regeneration	None	None	Yes
V	Neurotmesis	Complete severance of the nerve	None	None	Yes

Ganglionic Classification

Injuries can also be classified to pre- and post-ganglionic injuries. Pre-ganglionic injuries are injuries in which the spinal roots are avulsed from the spinal cord, while post-ganglionic injuries are those that occur distal to the dorsal root ganglion. Pre-ganglionic injuries are characterized by the loss of motor function only. In contrast, post-ganglionic injuries are characterized by the loss of both sensory and motor functions.¹

Millesi Classification⁶

- I Supraganglionic
- II Infraganglionic
- III Trunk
- IV Cord.

Leffert Classification of Brachial Plexus Injuries

- I Open (usually from stabbing)
- II Closed (usually from motorcycle accident)
 - IIa Supraclavicular
 - Preganglionic:
 - avulsion of nerve roots, usually from high speed injuries
 - with other injuries and LOC;
 - no proximal stump, no neuroma formation
 - pseudomeningocele, denervation of neck muscles are common
 - Horner's sign (ptosis, miosis, anhidrosis)
 - Postganglionic:
 - roots remain intact;
 - usually from traction injuries;
 - there are proximal stump and neuroma formation
 - IIb Infraclavicular Lesion:
 - usually involves branches from the trunks (supraclavicular);
 - function is affected based on trunk involved;

Trunk Injured	Functional Loss
Upper	biceps, shoulder muscles
Middle	Wrist and Finger Extension
Lower	Wrist and Finger Flexion

III Radiation induced

IV Obstetric

IVa Erb's (upper root) - waiter's tip hand; shoulder adducted and internally rotated with the elbow extended, forearm pronated, wrist flexed, and the hand in a fist.

IVb Klumpke (lower root); Arm supinated with the elbow bent and wrist extended.

Wallerian Degeneration³

Wallerian degeneration occurs when there is disruption of the axon. The distal portion of the axon degenerates and fragments. Myelin is transformed into neutral fat and phagocytosed by macrophages. Debris of the axon and the myelin sheath form ovoids that are gradually digested and disappear.

Proximal to the lesion, degeneration stops at the first internode in mild injuries, but may extend further proximally in severe injuries. Within hours of injury the ends of the severed axons seal over, and the sealed ends swell with cellular organelles because anterograde axonal transport in the proximal stump and retrograde axonal transport in the distal stump persist for several days. Resealing is a necessary prelude to axon regeneration from the proximal stump.

Axonal degeneration is not a passive process, but an active programmed response to disconnection from the cell body and the target organ. Loss of the axoplasmic cytoskeleton begins within about seven days in humans, accompanied by a program of auto-destruction.

In the distal stump, although the axon degenerates and disappears, the connective tissue basement membranes may remain, forming endoneurial tubes. Schwann cells proliferate and line the endoneurial tubes. These arrays of Schwann cells and processes within the basement membrane provide the pathway and scaffolding for axonal regeneration.

Wallerian degeneration begins within hours of injury and is complete by 6–8 weeks, leaving a distal stump comprising only endoneurial tubes lined by Schwann cells. The Schwann cells are not permanent; they disappear if axonal regeneration does not occur. Endoneurial tubes that do not receive a regenerating axon shrink and are eventually obliterated by scar tissue.

Nerve Regeneration³

In the central nervous system (CNS), recovery of function is accomplished by plasticity, using intact areas to take over the function of damaged areas; the CNS does not repair itself. The approach of the peripheral nervous system (PNS) to injury is to repair itself, and this is an essential difference between the two. Regeneration and repair processes go on at multiple levels following nerve injury, including the nerve cell body, the segment between the neuron and the injury site (proximal stump), the injury site itself, the segment between the injury site and the end organ (distal stump), and the end organ. The repair process may be disrupted at one or more of these sites. With mild injuries, regeneration and repair begin almost immediately. Remyelination in neurapractic injuries can occur fairly rapidly. With more severe injuries, there is an initial shock phase, after which regeneration and repair phases continue for many months.

Repair can occur through three mechanisms: remyelination, collateral sprouting distally from preserved axons, and regeneration from the site of injury. Collateral sprouts can provide reinnervation in partial nerve injuries, and when there are many surviving axons they may be very effective. With lesions involving less than 20–30% of the axons, recovery is predominantly by collateral sprouting from surviving axons, and occurs over 2–6 months. When more than 90% of axons are injured, the primary mechanism of repair is regeneration from the injury site.

The success of regeneration from the proximal stump depends to a large degree on the distance from the injury site. Even when good motor recovery occurs, sensory deficits, particularly in proprioception, may impair functional outcome.

Attempts at regeneration begin soon after injury. A cascade of events involving cell signaling molecules and neurotrophic factors occurs after nerve injury.

Schwann cells play an indispensable role in promoting regeneration by increasing their synthesis of surface cell adhesion molecules, and by elaborating basement membrane containing extracellular matrix proteins, such as laminin and fibronectin. Schwann cells produce neurotrophic factors that bind to receptors and are responsible for a signal that leads to gene activation. Within 30 min after injury, intracellular processes that promote repair and regeneration have already been activated.

Within days after injury, Schwann cells begin to divide and create a pool of dedifferentiated daughter cells. These dedifferentiated Schwann cells upregulate expression of nerve growth factor (NGF), other neurotrophic factors, cytokines, and other compounds that lead to Schwann cell differentiation and proliferation in anticipation of the arrival of a regenerating sprout.

Nerve growth factor receptors on the Schwann cells lining the endoneurial tubes in the distal stump increase. The action of NGF on these Schwann cell receptors stimulates regenerating axonal sprouts. After injury, macrophages migrate into the distal stump and may be involved in initiating Schwann cell proliferation. Neural cell surface

molecules and the extracellular matrix molecules laminine and tenascin are strongly upregulated by denervated Schwann cells and may foster axonal regeneration.

Neuronal survival is facilitated by the activation of trophic factors from multiple sources, including neurotrophins, neuropoietic cytokines, insulin-like growth factors (IGFs), and glial-cell-line-derived neurotrophic factors (GDNFs). These alterations help shift the neuron from transmitting mode to growth mode.

The neuron's capability to sustain regenerative attempts persists for at least 12 months after injury. The first attempts at repair in the proximal stump may begin as early as 24 hours, but when the injury is more severe it may be delayed for weeks. Regeneration depends on activity at a specialized growth cone at the tip of each axonal sprout. The growth cone produces a protease that helps dissolve material blocking its path. With severe injuries that disrupt the endoneurial tubes, regenerating sprouts may encounter formidable obstacles.

The process leading to growth cone formation begins within hours after injury, and many sprouts arise from each parent axon. Growth cones send out finger-like extensions, filopodia, that explore and sample the environment, acting as long distance sensors. Growth cones have remarkable abilities to detect navigational cues. Proper reading and integration of these cues is essential for precise rewiring of the regenerating nerve. The mobility of the growth cones at the ends of axon sprouts depends on receptors on the growth cones that receive guidance cues from the local environment. These navigation signals control growth cone advance, turning, and branching. Growth cones appear to be guided by at least four different mechanisms: contact-mediated attraction, chemoattraction, contact-mediated repulsion, and chemorepulsion. These mechanisms seem to act simultaneously and in a coordinated manner to direct pathfinding. The growth cone's actin filaments are common targets for this guidance signaling. Navigational cues trigger local accumulation of actin filaments that promote lengthening of filopodia. Guidance cues have been classified as either attractive or repulsive. Axons that successfully enter the endoneurial tubes in the distal stump stand a good chance of reaching the end organ. The growth cone contains multiple filopodia that adhere to the basal lamina of the Schwann cell and use it as a guide.

The most critical factor affecting nerve regeneration is the length of the gap between the proximal and distal stumps. Axons that cannot reach the distal stump are wasted; they may wander into adjacent tissue or become encased in the scar that invariably forms within the gap between the proximal and distal stumps. Scar within the bridging tissue impedes regeneration and leads to misdirection and aberrant regeneration, as axons sprout into functionally unrelated endoneurial tubes.

Clinically, an estimate of 1 mm/day or 1 in/month is generally used. The variability depends on several factors. Regeneration is better proximally and in younger individuals. Regeneration after surgical repair is slower than spontaneous regeneration.

Axon regeneration is not synonymous with return of function. Even after the axon reaches its target, a maturation process must evolve, including remyelination, axonal enlargement, and the establishment of connections with the end organ before functional recovery can ensue. Incomplete motor recovery after moderate to severe injuries may be due to a number of factors. Muscle fibers atrophy quite rapidly. Fibrotic changes can be detected in the muscle as early as 3 weeks post-injury. If the muscle is not reinnervated, fibrosis gradually progresses and will replace the muscle completely within about 2 years. Reinnervation must occur within approximately 12–18 months to provide a functional outcome. Even when there is successful reinnervation, the muscle seldom returns to normal strength. Intramuscular fibrosis may limit contractile efficiency and aberrant regeneration may reduce the synergy of contraction. Axonal misdirection, or pathfinding errors, is particularly problematic with proximal injuries to large mixed nerves.

Clinical Presentation

Brachial plexus injuries are often accompanied by other severe injuries. These injuries may hinder the diagnosis of a nerve injury until patient's recovery. For this reason, there should be a high level of suspicion in cases involving an injury of the shoulder girdle or any trauma involving fracture of the first rib or rupture of the axillary artery. A detailed examination of the brachial plexus and its terminal branches can be performed within a few minutes. Clinically, brachial plexus lesions present with weakness, muscle wasting, sensory disturbances, reduced tendon reflexes, and pain. The most frequent clinical manifestation of a brachial plexus lesion is muscle weakness and wasting. Weakness may persist during only a few hours or days in case of neurapraxia or persist for longer or even life-long in case of axonal lesions due to tearing or disruption.²

According to the location of the lesion within the plexus, various types of brachial plexus palsies are differentiated. These include supraclavicular lesions, which affect the trunks (upper, middle and lower trunk (primary strand) lesions), clavicular lesions (level of divisions), and infraclavicular lesions, which affect the cords. However, variable combinations of these lesion types may occur.²

Supraclavicular Injuries

In supraclavicular injuries, the shoulder typically presents in adduction and internal rotation; and the elbow/forearm is pronated. Suprascapular nerve injuries, which are most often located at or posterior to the suprascapular notch, are associated with the presence of tenderness over the notch, muscle weakness during shoulder abduction, and external rotation. Lesions at the level of spinoglenoid notch are related to isolated weakness of infraspinatus muscle. Palsy of long thoracic nerve is clinically evident with a defect during scapular abduction, while dorsal scapular nerve deficit will affect the stabilization of the scapula.²

Infraclavicular Injuries

Injuries at the infraclavicular level may have been generated by high energy trauma mechanisms at the shoulder level and may be potentially associated with rupture of the axillary artery. Axillary, suprascapular, and musculocutaneous nerves are most likely the affected nerves at that level of injury. The evaluation of median, ulnar, and radial nerve is performed by the examination of wrist and fingers. The musculocutaneous nerve as well as high lesions of radial nerve is examined by the flexion and extension of the elbow. The axillary nerve, which is a branch of the posterior cord, is examined with active shoulder abduction and deltoid muscle strength. Injury to the posterior cord may affect the function of radial nerve and the muscles that it innervates. Latissimus dorsi is innervated by the thoracodorsal nerve, which is also a branch of the posterior cord and is located closely to the posterior wall of axillary fossa. Pectoralis major receives innervation by the medial and lateral nerves, which are branches of medial and lateral cord, respectively. Lateral anterior thoracic nerve innervates the clavicle, whereas medial anterior thoracic nerve innervates the sternocostal head of the muscle. The muscle can be palpated as the patient adducts his arm under resistance. Proximally, the suprascapular nerve is a terminal nerve branch at the level of trunks.²

Upper Trunk Lesion

If the upper trunk (C5-6) is exclusively affected, weakness is found for shoulder abduction (deltoid muscle, innervated by the axillary nerve), external shoulder rotation (supra- and infraspinatus muscles, innervated by the suprascapular nerve as well as the subscapular muscle, innervated by the subscapular nerve), supination (supinator muscle, innervated by the radial nerve), lowering of elevated arms (latissimus dorsi, innervated by the thoracodorsal nerve), and elbow flexion (biceps muscle, innervated by the musculocutaneous nerve and brachioradialis muscle, innervated by the radial nerve). The brachioradialis muscle (radial nerve) is also affected. The rhomboid, levator scapulae, and anterior serratus muscles are spared since their innervating nerves originate before formation of the upper trunk. Sensory dysfunction is found in the C5 and C6 dermatomes. The biceps tendon reflex is diminished or absent.⁴

Middle Trunk Lesion

If the middle trunk (C7) is exclusively affected, weakness concerns muscles responsible for finger, wrist, and elbow extension as well as thumb flexion (flexor poll. longus, flexor poll. br. muscles) and thumb extension (extensor poll. long.). The pectoralis major muscle is also affected while the brachioradialis muscle remains spared. Sensory disturbances mainly concern the dermatome C7. The triceps tendon reflex is diminished or absent.⁴

Lower Trunk Lesion

If the lower trunk (C8-T1) is exclusively affected, weakness affects the median and ulnar nerve innervated intrinsic hand muscles. The triceps and the hand extensor muscle

remain intact. There is hypoesthesia of the dermatomes C8-Th1. There may be also regional hypoesthesia if the medial cutaneous antebrachii or the medial cutaneous brachii nerves are involved. Due to the length of the affected nerves, reinnervation of the distal muscles is less efficient than in case of an upper trunk lesion. Tendon reflexes are preserved. Some patients may additionally present with a Horner syndrome (ptosis, miosis, enophthalmus, and partial facial anhidrosis). Weakness in lower trunk lesions is usually more pronounced compared to weakness in upper trunk lesions since spinal roots (preganglionic fibers) may be additionally affected.⁴

Posterior Cord Lesion

If the posterior cord is exclusively affected, there is weakness of the deltoid muscle, the teres minor muscle, the latissimus dorsi, the subscapular, and the finger, wrist, and elbow extensor muscles. Sensory deficits concern the cutaneous branches of the axillary and the superficial radial nerves. The triceps tendon reflex is diminished or absent. There may be hypohidrosis or anhidrosis.⁴

Medial Cord Lesion

In case of a lesion of the medial cord, there is weakness of the forearm flexors and of all hand muscles (ulnar and median involvement). Sensory deficits concern the skin branches of the ulnar and the cutaneous antebrachii and brachii medial nerves. Contrary to a lower trunk lesion the pectoralis minor muscle is additionally affected. The triceps and biceps tendon reflexes are normal. There may be hypohidrosis or anhidrosis.⁴

Lateral Cord Lesion

In case of an exclusively lateral cord lesion there is weakness of the brachial biceps, brachialis, coracobrachialis muscles, and the finger flexors (excluding the thumb). Sensory deficits concern the cutaneous branches of the musculocutaneous and median nerves. The biceps brachii tendon reflex is diminished or absent. There may be hypohidrosis or anhidrosis.⁴

Motor Function

Below is a summary of the nerves, spinal levels, muscles and actions of the upper extremity:

Nerve	Level	Muscle	Action
Dorsal Scapular	C5	Rhomboid	Scapula stabilization
Long thoracic	C5	Serratus anterior	Scapula abduction
Suprascapular	C5	Supraspinatus, infraspinatus	Shoulder abduction & ext rot
Medial pectoral	C8	Pectoralis major	Shoulder adduction

Lateral pectoral	C7	Pectoralis minor	Scapula stabilization
Suscapular	C5	Subscapularis, teres major	Shoulder internal rotation
Thoracodorsal	C7	Latissimus dorsi	Shoulder adduction
Musculocutaneous	C5	Biceps brachii, brachialis	Elbow flexion
Ulnar	C8; T1	FCU, hand intrinsics	Wrist & finger flexion, finger abd
Median	C6,7,8; T1	Finger flexors, pronators, FCR	Wrist & finger flexion, pronation
Radial	C6,7,8	Supinator, triceps, extensors	Elbow, wrist & finger ext, sup
Axillary	C5	Deltoid, teres minor	Shoulder abduction

Electrophysiological Evaluations

Electrodiagnostic evaluation may confirm the diagnosis, pinpoint the lesions, determine the severity of axial discontinuity, and eliminate other clinical entities from differential diagnosis. They are valuable tools, which must be used in conjunction with meticulous physical examination and adequate imaging evaluation, and not as their substitute. In closed injuries, electromyography (EMG) and nerve conduction velocity studies (NCV) may be performed 3-4 weeks after the injury, when the conduction of the potentials has stopped along a nerve with postganglionic injury due to Wallerian degeneration. Serial testing in conjunction with repeated physical examination every few months can document and quantify ongoing reinnervation or denervation.²

Electromyography (EMG)

Electromyography (EMG) tests muscles at rest and during activity. Denervation changes (fibrillation potentials) can be seen as early as 10 to 14 days after injury in proximal muscles and as late as 3 to 6 weeks in distal muscles. The presence of voluntary motor unit potentials with limited fibrillation potentials signifies better prognosis than the cases where there is absence of motor unit potentials and many fibrillation potentials. The diagnostic benefit of EMG is related to the experience and the capability of the clinician to interpret the results of these tests. Early signs of muscle recovery may be detected on EMG (occurrence of nascent potentials, decreased number of fibrillation potentials, appearance of or an increased number of motor unit potentials). These signs contribute to expected clinical recovery in weeks or months. However, EMG recovery does not always ensure relevant clinical recovery. In addition, EMG evidence of ongoing reinnervation may not be detected in lesions where target end organs are more distal.²

Nerve Conduction Velocity (NCV)

Nerve conduction velocity (NCV) is used initially as a screening test for the presence or absence of conduction block. This test assesses both motor and sensory function via a

voltage stimulator applied to the skin over different points of the nerve to be tested. The evoked response is recorded from a surface electrode overlying the muscle belly (motor response) or nerve (sensory response).⁷

NCV and EMG obtained serially over time may map nerve recovery and identify a neurapraxic or axonotmetic lesion. The lack of spontaneous clinical or NCV/EMG recovery after three- to six months warrants surgical exploration depending on the level of injury. The problem remains that the most opportune time for surgical intervention has passed by then. The effects of chronic axotomy and muscle denervation render the tissue environment suboptimal for successful axonal regeneration. Since acute repair leads to better functional restoration, delays introduced by “wait-and-see” diagnostics can be costly. There is a great clinical need for accurate nerve injury diagnostics in the setting of acute injury.⁷

Imaging Studies

Radiographic (X-ray) Imaging

Standard radiographic imaging (x-rays) after a neck or shoulder girdle injury may reveal evidence of a concomitant neurological lesion. Radiographs of cervical spine, shoulder girdle, humerus, and chest should be obtained. Cervical spine radiographs should be evaluated for fractures, which could imply that spinal cord is in danger. Moreover, the presence of fracture in the transverse process of a cervical vertebra indicates possible root avulsion at the same level. Clavicle fractures are also indicative of a possible brachial plexus injury. Clavicle radiographs can reveal fractures of the first or second rib, which may suggest injury to the overlying part of brachial plexus.²

Magnetic Resonance Imagery (MRI)

Traditional Magnetic Resonance Imagery (MRI) is the imaging method of choice for non-traumatic plexopathies. This is mainly because MRI affords multi-planar images with excellent soft-tissue contrast that is superior to that afforded by both computed tomography (CT) and sonography. It also enables the differentiation between pre- and post-ganglionic injuries, which dictates the type of treatment the patient receives. In addition, MRI is non-invasive and does not employ radiation. The diagnostic accuracy of MRI is relatively high.¹

In addition, MRI is used in the diagnosis of entrapment syndrome (also known as thoracic outlet syndrome) and in the identification of its various causes, namely, hereditary motor sensory neuropathy (also known as Charcot Marie Tooth syndrome) and infective or inflammatory conditions of the brachial plexus such as brachial neuritis, chronic inflammatory demyelinating neuropathy, and other chronic infections such as leprosy and syphilis. Despite having many uses and benefits, MRI also has several drawbacks, which must be kept in mind to optimize the use of MRI for the patients' benefit. MRI has been shown to be less accurate in detecting nerve root avulsions

compared to CTM and MRM. In addition to all this, MRI is time consuming, expensive, and not universally applicable to all patients; for example, MRI may not be applicable to patients with metal devices, children, and claustrophobic patients who may require general anesthesia.¹ Moreover, there are artifacts caused by motion such as swallowing, tremor, respiratory, and cardiac movements of the chest, as well as cerebrospinal fluid flow interference which could downgrade the quality of imaging.²

Magnetic Resonance Neurography (MRN)

MRN is a special type of MRI that is tissue specific and capable of eliciting the morphological features of nerves, such as their caliber, continuity, and relation to nearby structures such as nerves, muscles and bones, as well as pathological features of the nerves (e.g., nerve fibrosis, inflammation, and edema). The diagnostic efficacy of MRN is high. MRN depends on the alterations in endoneural fluid content in the nerves since pathological processes increase this fluid relative to other cellular components. MRN findings include disruptions of the course of the proximal elements at the scalene triangle, fibrous band entrapments affecting the C8 and T1 spinal nerves and the lower trunk of the brachial plexus, gross distortions of the mid-plexus, hyperintensity consistent with nerve irritation at the level of the first rib, and distal plexus hyperintensity.

MRN is best used:

- When MRI and electrodiagnostic studies are unavailing regarding spinal or peripheral nerve pathology.
- If MRI shows multilevel disease and electro-diagnostic studies are unable to confirm these results, and
- In patients who are unable to undergo electrodiagnostic studies (i.e., patients on anti-coagulants or a coexistent disease that reduces accuracy such as diabetes).

In addition, MRN can localize trauma, radiation injury, and neoplasms of the brachial plexus and peripheral nerves. An important aspect of MRN is that it is most useful when the onset of symptoms is less than 1 year and is less useful when it has been more than 2 years.¹

Magnetic Resonance Microscopy (MRM)

Magnetic Resonance Microscopy (MRM) should be used for traumatic injuries, such as traumatic meningoceles and root avulsions. MRM would be most beneficial when traditional MRI and electrodiagnostics are inconclusive in their results, as well as for determining whether tumors of the plexus are primary or secondary and for the surgical planning in patients with plexus trauma or lesions. MRM is the imaging method that achieves myelogram-like images with MRI. Its use is mainly in the diagnosis of traumatic meningoceles and nerve root avulsion, where MRM was found to be superior to CTM. In addition, MRM is non-invasive, does not employ radiation, and is superior in the assessment of pseudomeningoceles compared to CTM. The rationale for using

MRM is mainly that the diagnostic accuracy of MRM is superior to CTM and MRI. In addition, MRM can be employed in the acute phase of injury unlike CTM, where lumbar puncture and use of contrast media carries a slight risk.¹

Computed Tomography Myelography (CTM)

CTM is the current gold standard for imaging nerve root avulsion injuries to the brachial plexus. The diagnostic accuracy of CTM is equal to or greater than that of standard myelography and MRI. CTM is also useful in evaluating tumors infiltrating the brachial plexus, since it is superlative in the detection of bony erosions of the spine as well as changes in the neural foramina. Obstetric brachial plexus lesions (OBPL) can be evaluated by CTM, however, it is invasive and requires sedation of the child. Nevertheless, currently, CTM is strongly recommended for any patient undergoing reconstruction for OBPL.¹

Sonography¹

Sonography can screen for most brachial plexus lesions and, therefore, it can be considered as a baseline or a screening investigation; however, it is hindered by the experience required by the technician and the complex anatomical location of the plexus.

Sonography does not have the same quality as MRI in evaluating soft tissues, such as the brachial plexus; however, there are many advantages of sonography, making it an important complimentary tool in imaging the brachial plexus. The first and most important advantage is patient satisfaction. Another reason is the interactive nature of sonography compared to MRI, which puts the patients to ease during the evaluation. Other advantages include the ability to conduct an ultrasound examination in virtually any patient, ability to perform real-time examination, availability of important information about the adjacent blood vessels through Doppler sonography, the ability to differentiate fluid and solid material better than MRI, ability to guide therapeutic interventions, ability to provide bilateral comparison, and availability of a more flexible field of view.

In addition, sonography costs much less than MRI and utilizes no radiation, which is a major advantage over most imaging modalities. Almost all pathologies affecting the brachial plexus can be visualized or at least screened for through sonography. Entrapment neuropathies due to a cervical rib, elongated C7 transverse process, and other causes of the thoracic outlet syndrome can also be detected. This is of paramount importance in children since it minimizes the radiation risk from standard radiographs. Sonography is also valuable in the detection of nerve tumors from the brachial plexus. Although certain features may aid in distinguishing benign lesions from malignant ones, it is not possible to determine this based on sonography alone. Sonography is also useful in guiding interventions (i.e., biopsy of a tumor and brachial plexus anesthesia) and in the postoperative follow-up of cases. In the case of traumatic brachial plexus injuries (pre- and post-ganglionic) sonography can detect root avulsion, nerve injury in the form of a neuroma, and scar tissue formation. Follow up of brachial plexus injuries is

a major area in which sonography is useful since it helps in monitoring the progress of lesions, such as tumors and traumatic nerve lesions.

Conservative Treatment

Treatment Goals

The goals of conservative treatment are to maintain the range of motion of the extremity, to strengthen the remaining functional muscles, to protect the denervated dermatomes, and to manage pain. Chronic edema may appear as a result of hypokinesia, loss of vascular tone due to sympathetic denervation, and any other soft tissue injury. Keeping the extremity raised and splitting and tensile banding may decrease edema. This should be followed by physical or occupational therapy otherwise stiffness may be the final outcome, especially in the hand.⁸

Pain Management

Managing pain is the most important consideration in the early management of BPI. The pain requires medications which are specific for neuropathic pain, such as tricyclic antidepressants, serotonin reuptake inhibitors, anti-convulsants such as carbamazepine, phenytoin, and lamotrigine, gabapentin and pregabalin, antiarrhythmics, baclofen and others. The mechanism of action of these drugs is thought to be the reduction of neuronal hyperexcitability, peripherally or centrally. Although traditional analgesics are not regarded as first-line drugs for treating neuropathic pain, agents such as nonsteroidal anti-inflammatory drugs, tramadol, and opioids may be useful. Topical agents such as lidocaine patches and capsaicin may be useful. Transcutaneous electrical nerve stimulation may be useful in some instances.³ Other modalities which have some role in neuropathic pain management are yoga, massage, meditation, cognitive exercise, and acupuncture.¹⁹ When these measures fail, the pain may be controlled with a peripheral nerve block or with an indwelling catheter. Desensitization techniques may help reduce pain and allodynia.³

Strength and Range of Motion

Injury to the peripheral nerves frequently lead to notable muscle atrophy and significant functional deficits. The neuromuscular junction undergoes physiological changes after nerve injury. It is important to maintain the muscles in a normal physiological length to prevent vascular and lymphatic stasis, contractures, and joint stiffness. Therapeutic interventions should include range of motion and strength activities and appropriate static or dynamic splinting.¹⁹

Surgical Treatment

A variety of surgical procedures are available to improve the functional outcome. Which one is appropriate depends on the type of lesion. The following are some of the more common surgical techniques.

Direct Repair

Direct nerve repair is the preferred surgical treatment for severe axonotmesis and neurotmesis injuries. An end-to-end epineural suture repair may be performed only when there is a sharp laceration of the nerve and minimal tension exists on both sides of the disruption.⁷

End-to-side nerve repair is a procedure in which an injured nerve ending is coapted to the side of a functioning donor nerve nearby. An epineural window serves as a connection point between the two nerves. The end-to-side nerve coaptation can be used to repair severed digital nerves, mostly caused by previous hand injuries, when direct tension-free coaptation is not possible.²¹

Nerve Grafting

The autologous nerve graft is the gold standard for nerve injuries that cannot be repaired by direct tension-free coaptation. The most common source of autologous nerve grafts is the sural nerve. This autologous graft is quite easily harvested and almost always has an appropriate diameter for digital nerve reconstruction. Other common sources of nerve grafts are posterior interosseous nerve and medial antebrachial cutaneous nerve. However, the use of autologous nerve grafts carries the risk of donor site morbidity including sensory loss within the area of harvest and the corresponding peripheral nerve field, painful neuroma, and scar formation. Increased operating time and limited harvest sites are also disadvantages.²¹

Nerve Transfer

A nerve transfer is the surgical coaptation of a healthy nerve donor to a denervated nerve. This is usually reserved for important motor nerve reconstruction although it can equally be applied to critical sensory nerves. Nerve transfers use an expendable motor donor nerve to a less important limb muscle. The nerve is cut and then joined to the injured distal end of the prioritized motor nerve. There are several benefits of utilizing a nerve transfer. In most cases there is only one neurorrhaphy site; with nerve grafts, there are two. In addition, nerve transfers minimize the distance over which a nerve has to regenerate because it is closer to the target organ and is more specific. Pure motor donors are joined to motor nerves and sensory donors to sensory nerves, optimizing regeneration potential. As opposed to a tendon transfer, when a nerve transfer is

successful, recovered function is similar to the original muscle function because synchronous physiologic motion may be achieved. With quicker nerve recovery, more rapid motor reeducation is also possible. The goal is to maximize functional recovery with fast reinnervation of denervated motor targets. The most common applications of motor nerve transfers include the restoration of elbow flexion, shoulder abduction, ulnar-innervated intrinsic hand function, and radial nerve function. The disadvantages are finding an expendable donor nerve near the target muscle with a large enough motor fiber population from which to “borrow”. Importantly, the donor nerve target should be synergistic with the redirected target for the brain to accommodate the rewiring of the newly redirected fibers.⁷

Timing of the Surgical Repair

The most critical point when planning a surgical procedure in brachial plexus injuries is the delay between the accident and the intervention. Indications for emergency operative procedures include vascular injury, open penetrating injuries, and open infected crushing/stretching wounds. Almost emergent surgical operation during the first or second week is recommended for complete traumatic palsy of the C5-T1 root. After the 3rd month surgical operation is recommended for traumatic palsy injuries with no clinical sign of functional restoration or electromyography signs of denervation. Another group of patients recommended for surgical exploration is that with clinical and EMG signs of recovery of distal branches instead of proximal axons. Perioperative assessment of the lesion is more accurate after Wallerian degeneration has occurred. Lesions due to iatrogenic etiology should be surgically explored at an earlier stage, especially when electromyography reveals complete denervation with no signs of functional recovery. Nerve reconstruction is not recommended for traumatic lesions more than 9 months after the accident although there have been a few reports of successful procedures after 9 months.⁸

Surgical Outcome

Functional recovery is typically better with direct end-to-end repair than it is with nerve grafting or transfer. Operations done early have a better outcome than those done later. Although muscles are irreversibly damaged after about 18 months, the sensory fibers and sensory receptors survive for a much longer period, and surgery done much later, even years later, may restore protective sensation to an insensate part.³

Secondary Surgical Procedures

In the absence of spontaneous recovery or when the first surgical procedure does not provide satisfactory outcomes then a second operation may be required. In such cases there should be specific signs of neurological denervation or no possibility of neurological recovery, or sufficient time should have passed with no functional

improvement. Arthrodesis, tendon transfer, and functional free muscle transplantation are alternative secondary surgical options.

Arthrodesis⁸

In complete brachial plexus traumatic injuries that present with unstable and painful shoulders, arthrodesis could be a definite solution. Prior to considering shoulder arthrodesis, certain parameters must be taken into account. First, good thoracic-shoulder functionality is of great importance. Second, the motion mobility of the peripheral hand is important as shoulder arthrodesis has no clinical effect on a paralytic hand whatsoever. The acromioclavicular joint, sternum-clavicular joint, and scapulothoracic joint should be intact. Any dysfunction may affect the success of arthrodesis.

The shoulder is typically fused with approximately 20 degrees of abduction, 30 degrees flexion, and 30 degrees of internal rotation to allow the patient to be independent in his daily life with a mean range of 60 degrees abduction and flexion through the scapulothoracic joint.

Tendon Transfers⁸

Tendon transfers are useful in restoring upper extremity function after BPI. An indication for tendon transfer is upper or lower brachial plexus traumatic injury with only partial paralysis. After tendon transfer, muscle strength is not restored to preinjury levels, in most cases with the loss of at least one grade on the measurement of muscle strength.

Shoulder

Many tendon transfer techniques have been described for treating partial shoulder paralysis. Among the most common procedures are the following:

1. Trapezius to deltoid transfer to restore abduction of the shoulder;
2. Latissimus dorsi transfer to improve external rotation.
3. Anterior transfer of the posterior branch of the deltoid muscle to restore nonfunctional anterior segment.

Elbow

Restoration of elbow flexion is of great importance for a good clinical and functional outcome. Depending on the level of injury and the degree of reinnervation there are different types of surgical procedure. The surgical goal is to restore good muscle strength through a range of elbow motion (30 to 130 degrees). The most commonly used procedures are as follows:

1. Transfer of the common origin of the flexor forearm muscles to a proximal section. This type of procedure provides nonsatisfactory outcomes when used in cases of complete elbow paralysis.
2. Transfer of latissimus dorsi muscle to the tendon of the biceps brachialis provides great muscle strength, but this muscle is often denervated;

3. Transfer of pectoralis major brachial branch tendon to brachial biceps. A fused shoulder is required for the best postoperative result;
4. Transfer of triceps tendon to biceps provides good results not only with respect to muscle strength but also aesthetically.

It is important to understand that tendon transfer on a stiff joint is pointless. In the case of a stiff joint, intensive physical or occupational therapy is required to achieve an acceptable range of motion even with surgical release. In the case of complete sensory loss, tendon transfer is not recommended due to the fact that a hand with no tactile sensation is functionally very limited.

Functioning Free Muscle Transplantation (FFMT)

Free functioning muscle transfer (FFMT) is another reconstructive option for severe and delayed nerve injuries including those that have failed after primary reconstruction, providing an uninjured donor nerve can be located. The procedure transfers a healthy muscle and its neurovascular pedicle to a new location to assume a new function. This can be used in a situation where both the nerve and muscle are damaged due to either severe acute injury or changes from chronic axotomy and muscular fibrosis. The muscle is powered by transferring a viable motor nerve to the nerve of the FFMT and restoring the circulation of the transferred muscle with microsurgical anastomosis to recipient vessels. Within several months, the transferred muscle becomes reinnervated by the donor nerve, eventually begins to contract, and ultimately gains independent function. Function provided by the FFMT most commonly is elbow flexion but also includes elbow extension, finger and wrist extension, and grasp, for example, in cases of complete brachial plexus avulsion with limited donor nerve options for nerve transfers. Current indications for FFMT in brachial plexus injuries include the time when reinnervation of native musculature is not possible (i.e., traumatic loss of muscle), late reconstruction (i.e., >12 months), previously failed reconstruction, or acute injuries to restore prehension.⁷

Thoracic Outlet Syndrome

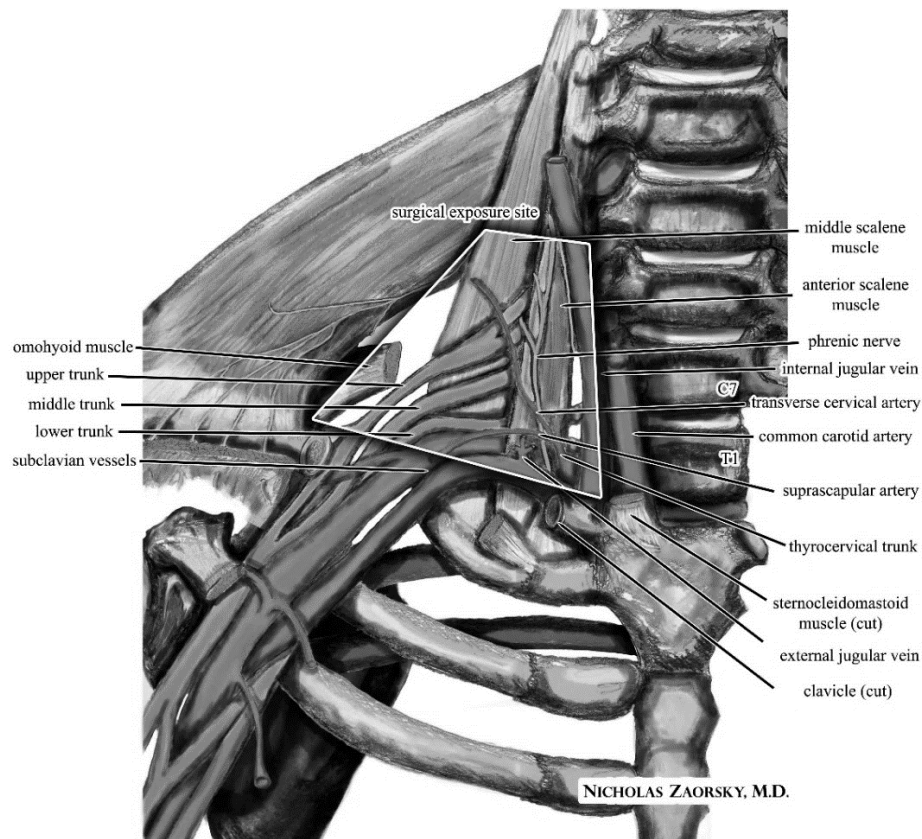
Thoracic outlet syndrome (TOS) is a disease which involves compression of the neurovascular bundle as it exits the thoracic girdle. For many years, the existence of such a syndrome was poorly understood and therefore its acceptance as a medical diagnosis was controversial. Today, however, the syndrome is better recognized; its symptoms and pathophysiology are more clearly defined.¹⁰

Thoracic outlet syndrome is a complex entity that is characterized by different neurovascular signs and symptoms involving the upper limb. Compression is thought to occur at one or more of the three anatomical compartments: the interscalene

triangle, the costoclavicular space and the retropectoralis minor spaces. The clinical presentation can include both neurogenic and vascular symptoms.¹¹

Anatomy

The thoracic outlet is anatomically defined by the space between the first thoracic vertebra, first rib, and manubrium of the sternum. Anteriorly, the subclavius tendon lies next to the subclavian vein. Next, the scalene separates the subclavian vein from the subclavian artery. The brachial plexus travels posterior and laterally to the artery and is accompanied by the middle scalene muscle.¹⁰



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It is a very small space, already occupied by the anterior scalene, the subclavius, and prevertebral muscles. The thoracic outlet is also dynamic; the volume changes with respiration and any activity of the neck, thorax, or arm. Additionally, cervical ribs or anomalous first ribs, which tend to be more cephalad or fused with the second rib, can further affect the dimensions of the thoracic outlet. As the space continuously contracts and expands, there may be impingement of the brachial plexus or subclavian vessels by the osseous structures. Simultaneously, fibrosis and scarring may occur, which can then cause encroachment or inflammation.¹⁰

Pathophysiology^{10,11}

There are three types of TOS: neurogenic, venous, and arterial. Neurogenic thoracic outlet syndrome (nTOS) makes up approximately 95% of all patients suffering from this syndrome. The pathophysiology of neurogenic TOS most often is a combination of neck trauma (including microtrauma secondary to repetitive activities) plus an anatomic predisposition that results in pathology of the scalene muscles and compression of the brachial plexus.

Impingement of the neurovascular bundle supplying the upper limb occurs most frequently at the scalene triangle. This triangle is formed between the anterior and middle scalene muscles. Scalene muscle injury is emerging as the most common etiology of thoracic outlet syndrome. It is thought that this injury can be caused by a single significant traumatic event, or secondary to repetitive trauma over time. Overhead work activities may lead to this repetitive trauma, such that assembly line work, hair dressing, and cash register operation are jobs that are sometimes associated with this syndrome.

TOS most frequently occurs following a single episode of neck trauma such as a motor vehicle accident. The individual typically experiences neck pain within days followed by symptoms in the hand and arm within weeks. The trauma may cause scalene muscle hemorrhage resulting in edema in the region of the anterior and middle scalenes. This can eventually become fibrosed, scarred and result in spasm of the scalenes. As scarring and spasm develop in the scalene, the muscles compress the brachial plexus. This compression leads to the symptoms of pain and paraesthesias in the upper extremity. Those individuals with congenitally narrow thoracic outlet spaces, the presence of a cervical rib or cervical bands may be at greater risk of developing TOS following injury.

There are anatomic variations in the scalene triangle. The space between the anterior and middle scalenes can vary from very narrow to wide. NTOS is sometimes associated with pectoralis minor pathology. Those with pectoralis minor involvement will often have tenderness over the pectoralis minor tendon accompanying their symptoms of NTOS. Anomalies in the subclavius muscle, often referred to as subclavious posticus, may also play a role in the development of thoracic outlet syndrome. This supernumerary muscle sometimes inserts on the superior surface of the sternal end of the first rib, runs laterodorsally and inserts on the superior margin of the scapula. This muscle may be implicated in TOS as this aberrant muscle runs on the anterior surface of the subclavian vein and crosses over the brachial plexus. The incidence of cervical ribs and anomalous first ribs in the general population is rare, approximately 0.76%. Seventy percent of cervical ribs are found in women and anomalous first ribs are equally divided between men and women. The majority of cervical ribs and anomalous first ribs are asymptomatic. If an individual with a cervical rib or anomalous first rib experiences a neck injury this can predispose one to the develop TOS, the majority being neurogenic. It is rare for patients with a cervical rib or anomalous first rib to spontaneously develop NTOS.

Epidemiology

Thoracic outlet syndrome is one of the most common types of neurovascular bundle compression disorders. Most cases involve the inferior trunk of the brachial plexus, usually among middle-aged women. The male to female ratio is 1:3, and more than 80% of patients are between the ages of 25 and 40 years. TOS also occurs in young children and teenagers, but the diagnosis is often missed because TOS is not commonly thought of as a disorder that affects children. In addition, children are likely to attribute their symptoms to simple muscle strain.¹²

Clinical Presentation¹¹

Neurogenic TOS (NTOS) involves compression of the brachial plexus trunks or cords, comprised of nerves that come from the C5-T1 spinal levels. The clinical picture is one of nerve irritation. Individuals with this syndrome often experience pain, paraesthesias and numbness in the neck, shoulder, arm and hand. The paraesthesias are most often reported in all 5 fingers but worse in the fourth and fifth digits and medial forearm. These symptoms are made worse by elevated, overhead, or outstretched positions of the arm. Individuals will often have pain over the trapezius and the neck, occipital headaches and may even experience anterior chest wall pain.

On physical examination there may be tenderness over the scalene muscles and subcoracoid space. There is often decreased sensation to light touch in the fingers, especially over the fourth and fifth digits. A positive Tinel's sign may be elicited over the area of the brachial plexus above the clavicle in the scalene triangle, with reproduction of paraesthesias in one or more of the nerve root distributions.

Clinical Assessment

There are provocative tests that can reproduce symptoms of Neurogenic TOS by putting stress on the neurovascular bundle. These tests can be used to help with diagnosis. In order for the test to be considered positive it must elicit the patient's symptoms.¹¹

Elvey's Test

The brachial plexus tension test (BPTT or Elvey's test) has proven to be a useful clinical test. Elvey's Test involves the individual lying in supine. The shoulder joint is abducted and externally rotated. The examiner then adds shoulder girdle depression, forearm supination, wrist and finger extension and, finally, elbow extension. The position of lateral neck flexion to the contralateral side is used in the examination of the symptomatic arms if the other maneuvers are of full range and fail to provoke a symptomatic response.¹¹

Upper Limb Tension Test (ULTT)

The Elvey's Test has recently been modified and referred to as the modified Upper Limb Tension Test (ULTT). It involves having the patient in a seated position and performing the maneuvers actively, rather than passively. This allows both sides to be tested simultaneously and the unaffected arm can serve as a control for the affected limb. The patient abducts their shoulders to 90 degrees with the elbows in extension. The second position involves the patient actively extending their wrists. The third position involves the patient tilting their head to their shoulder. Position one and two will elicit symptoms on the ipsilateral side. Position three will elicit symptoms on the contralateral side. A positive test is pain down the arm with reproduction of patient's symptoms. When a positive response is found it means that there has been compression of the nerve roots or branches of the brachial plexus in either the cervical spine, pectoralis minor space or the thoracic outlet.¹¹

Roos Test or Elevated Arm Stress Test (EAST)

Another test that has proven to have benefit in the diagnosis of NTOS is Roos Test. This test is sometimes referred to as the Elevated Arm Stress Test (EAST). This test involves having the patient stand and abduct their arms to 90 degrees, elbows flexed at 90 degrees and slightly posterior to the frontal plane. The patient is then instructed to open and close their hand slowly for three minutes. If the patient is unable to keep their arms in this position for 3 min, experiences heaviness, profound weakness or reproduction of their symptoms the test is considered positive.¹¹ Clinicians can actually see decreased ability to open and close their hands or lowering of the arm during the test in patients with neurogenic TOS. Patients rate the amount and location of discomfort, pain, fatigue, and numbness. Unfortunately, those with carpal tunnel syndrome, ulnar neuropathy, or fibromyalgia will demonstrate symptoms as well.¹⁰

Adson Test

Another common test for TOS is Adson Test. In order for this test to be considered positive the examiner must find a decrease in pulse along with a reproduction of symptoms. Adson Test involves locating the patient's radial pulse and the patient then rotates their head towards the test shoulder. The patient then extends their head while the examiner laterally rotates and extends the patient's shoulder with the elbow in extension. The patient then takes a deep breath in and holds it. The test is considered positive if there is loss of the radial pulse and reproduction of symptoms.¹¹

Electrodiagnostic Studies

There is controversy regarding the use of electrodiagnostic studies for the diagnosis of TOS. Nerve conduction velocity (NCV) prolongation is seen in some patients with long standing NTOS that results in atrophy of the muscle. However, in many instances, nerve conduction results are often found to be normal. Electromyography (EMG) may reveal neurogenic damage, such as increased motor unit action potential and decreased recruitment at maximum effort; however, EMG is typically not sensitive enough to pick up less severe NTOS.¹¹

Diagnosis

TOS is often a diagnosis of exclusion. Other causes such as herniated cervical disks, rotator cuff injuries, peripheral nerve entrapment, chronic pain syndromes, psychological conditions, multiple sclerosis, hypercoagulable disorders, atrial fibrillation with distal emboli, and upper extremity deep venous thrombosis all need to be considered because they can mimic the symptoms of TOS.¹⁰

A more objective examination is the lidocaine scalene block test. Under image guidance, either computed tomography, ultrasound, or fluoroscopy, the anterior scalene muscle is injected with lidocaine. Patients with nTOS should have some decrease or complete relief of symptoms for four hours.¹⁰

Conservative Management

The initial management of neurogenic TOS is non-operative. Approximately 60–70% of patients with nTOS can be successfully treated with avoidance of activities that precipitate symptoms, ergonomic modifications to the workplace, and selective use of pharmacologic agents such as nonsteroidal anti-inflammatories, antidepressants, and muscle relaxants.¹⁰

Physical or occupational therapy is also a very important component for these patients. The most critical point for being successful in TOS treatment is identifying actions that either relieve or exacerbate symptoms. Brachial plexus nerve compression and muscle imbalance in the cervicospinal region should be taken into consideration together. Postures and positions that cause compression at brachial plexus will affect the soft tissue surrounding it. Wrong postures and positions will cause pain to continue and to get aggravated, therefore, correct posture should be ensured and maintained. Most often, a flexed posture is seen in patients with TOS. Their head is positioned forward of the body, thoracic kyphosis is present, scapulas are in protraction and shoulders are in inner rotation. Most patients with TOS cannot switch to an ideal erect position instantly due to restriction of short and stiff muscles; elongation of stiff muscles for improved posture causes pain. Extension exercise programs should be implemented with these patients to gain regular muscle length required for ideal posture. In patients with TOS, since the head leans toward front of the body, stiffness occurs in the scalenes, suboccipital, sternocleidomastoid, upper trapezius, levator scapula muscles in postures where abduction and thoracic kyphosis occurs in scapulas. Protraction of the scapulas also shorten the serratus anterior muscle and cause weakness of both serratus anterior muscle and middle and lower trapezius muscles.²⁰

Other modalities include heat and ultrasound to facilitate effective stretching of the scalene and pectoralis minor. Gentle stretching, relaxation and biofeedback exercises are advocated. NTOS can be made worse with strengthening exercises with heavy weights and neck traction.¹¹ Conservative management should be attempted for 8–12 weeks before considering surgery or other interventions.

Surgery

A common surgical treatment for TOS is scalenectomy with the goal of decompression of the interscalene space. This is either done alone or in combination with first rib resection. Some surgeons advocate combining these two procedures to decrease the need for further surgeries. Some surgeons perform scalene muscle removal for suspected upper plexopathies and transaxillary first thoracic rib resection with suspected lower plexopathies. The indications for surgery include a sound clinical diagnosis, disabling symptoms with loss of function and an insufficient response to conservative measures.¹¹

Post-operative Therapy¹⁰

Rehabilitation may be initiated 2 weeks after the surgery. Therapy focuses on postural training as well as strength training in the shoulder, chest, and neck.

During weeks 1–3, therapists work on mobilizing the thoracic and cervical spine using thoracic extension and cervical flexion. This also begins to tease the patient's stretch and pain sensations. Patients concentrate on diaphragmatic breathing and perform gentle scapular posterior depression exercises. Finally, they start posture training.

In Weeks 4–6, patients add mild resistance training to improve scapular stability and mobility of the glenohumeral joint and rotator cuff.

Obstetrical Brachial Plexus Injury

Obstetric brachial plexus injury (OBPI), is also known as birth brachial plexus injury (BBPI), birth related brachial plexus injuries (BRBPI), or neonatal brachial plexus injuries (NBPI). Other medical terms that might be found in medical reports describing this injury include: Erb's Palsy (upper plexus injury), Klumpke's Palsy (lower plexus injury), Erb-Duchenne Palsy, and Horner's Syndrome (when sympathetic fibers are affected).

Epidemiology

The incidence of obstetric brachial plexus injury (OBPI) is about 1.51 per 1000 live births in the United States and reports vary from 0.38 to 5.8 per 1000 live births. The reported proportion of injuries that remain permanent varies from 50 to 90%.¹⁸

Etiology and Risk factors

Frequently cited risk factors for OBPI include macrosomia (defined as birth weight greater than 4500 g), instrument-assisted delivery, and shoulder dystocia (downward

traction of the fetal head during birth.¹⁸ However, other studies could not identify any reliable risk factor and regard OBPP as an unpredictable complication of delivery.⁴

Classification

The most common type of obstetric brachial plexus injury (73 – 86% of cases) involves damage to the upper spinal nerve roots (C5, C6, and C7). This is known as Erb's palsy or Erb-Duchenne palsy. Damage to the lower nerve roots (C8 and T1) is call Klumpke's palsy. Injury to this type occurs much less frequently than the upper (approximately 2-3% of children with OBPI). Other atypical and complete palsies also occur, and represent approximately 10% of all the OBPI cases.¹³

Erb-Duchenne Palsy - Injury to C5, C6, and C7:

C5: Patient is unable to abduct and externally rotate the shoulder.

C6: Patient is unable to flex the elbow joint.

C7: Patient is unable to extend the wrist joint.

Klumpke's Palsy - Injury to C8 and T1:

C8: Patient is unable to clench the fist.

T1: Intrinsic muscles of the hand are paralyzed.

Assessment

The Mallet grading scale remains the most commonly used system for assessing OBPI.¹⁴

This is a score with tests for five functions:

1. shoulder abduction,
2. external rotation with the arm against the side of the body,
3. putting the hand to the nape of the neck,
4. putting the hand on the back as high up as possible, and
5. hand touching the mouth.

This is not a sliding scale, as 5 grades are recognized: from function not possible (grade 1) to normal function (grade 5). Usually only grades 2, 3 and 4 are depicted in publications.¹⁵

Mallet Grading Scale

Grade I

- Flail shoulder

Grade II

- Active abduction $\leq 30^\circ$
- Zero degrees of external rotation
- Hand to back of neck impossible
- Hand to back impossible

- Hand to mouth with marked trumpet sign

Grade III

- Active abduction 30–90°
- External rotation up to 20°
- Hand to back of neck with difficulty
- Hand to back with difficulty
- Hand to mouth possible with partial trumpet sign (over 40° of shoulder abduction)

Grade IV

- Active abduction over 90°
- External rotation over 20°
- Hand to back of neck easy
- Hand to back easy
- Hand to mouth easy with less than 40° of shoulder abduction

Grade V

- Normal shoulder

Treatment

The evaluation and treatment of OBPI varies with the cause of the injury. When spontaneous recovery is not noted within the first few weeks following an injury sustained during delivery, it is essential that a medical professional who specializes in treating BPI be consulted. When spontaneous improvement does not result during the first three months post injury, significant functional recovery usually is not expected despite aggressive physical therapy and surgery. Early treatment most likely will include occupational and/or physical therapy to help maximize use of the affected arm while preventing contractures. If there is no evidence of improvement in function by the age of 4-6 months, further evaluation to address the potential for a surgical repair may be indicated. The clinical recovery noted following the initial surgery determines the need for other surgical procedures. The evaluation and treatment of other types of traumatic BPI usually proceed at a more rapid pace because such injuries are more commonly associated with the severance of nerve fibers. Maximizing functional use of the injured area is generally the overall goal of affected individuals, families and medical professionals.¹⁶

Prognosis

Most of the recovery from an OBPI occurs during the first three months post injury. Infants who recover partial antigravity upper trunk muscle strength in the first 2 months of life typically have a full and complete recovery by the age of 2. BPI may occur in combination with other injuries or impairments. Injuries to the upper brachial plexus affect the shoulder and upper arm (Erb's palsy) and spare the hand. These injuries are the most common and have the best prognosis because most of these injuries are related to nerve fiber stretching (neurapraxis) rather than tearing (avulsion or rupture). Injuries to the lower brachial plexus affect the forearm and hand (Klumpke's Palsy). These injuries have a worse prognosis. Extensive injuries that involve the entire plexus

result in anesthesia and total paralysis of the hand, arm, shoulder and the diaphragm and chest wall musculature on the side of the injury. Such injuries have the poorest prognosis.¹⁶

About 2/3 of children with this injury will recover to quite functional levels simply by maintaining looseness of joints while their nerves slowly heal. Some children have nerve injuries severe enough that they require surgical reconstruction with nerve grafts and nerve transfers to achieve even adequate function. One almost universally common outcome, even in children with otherwise good recovery, is that the motions of external rotation of the shoulder and supination of the forearm are weaker, later to recover, and often incomplete.¹⁷

Even beyond these direct functional weaknesses, because the arm is positioned poorly, joint contractures and imbalance of these motions can interfere with other upper extremity movements like elbow flexion, even when elbow flexion itself is well recovered. More importantly, lack of full motion leads to long term changes in the structure, growth, and posture of the shoulder requiring further musculoskeletal surgery, or a child with permanent deformity or disability. Surgery cannot completely correct this deformity. Any gains in active and passive range of motion during the first year of life may improve these long-term shoulder outcomes.¹⁷

Orthopedic Related Issues¹⁸

The presentation of weakness of the deltoid and external shoulder rotators caused by the common C5 injury seen in OBPI immediately affects growth of both the muscles and bones. Formation of contractures and consequent asymmetric muscle action on the developing bony elements of the shoulder results in bone deformation of the scapula and humerus. The scapula not only elevates and rotates laterally, but also becomes hypoplastic with flattening of the glenoid fossa and hooking of the acromion process. The clavicle and acromion process impinge upon the humeral head due to the abnormal positioning of the scapula and associated acromio-clavicular triangle (ACT), with its sides defined by the clavicular shaft and the acromion process and its base by an imaginary line connecting their medial ends. Functionally debilitating effects include medial rotation and posterior and inferior subluxation of the humerus within the glenoid fossa.

The abnormal migration of the scapula disrupts the normal anatomic relationships of the humeral head, the glenoid fossa and the acromio-clavicular triangle. Impingement of the distal acromio-clavicular triangle against the humeral head limits external rotation of the arm and shoulder. Without addressing the joint derangement, procedures such as humeral osteotomy are likely to fail or have significant rates of recurrence.

The medial rotation contracture (MRC) is the most significant secondary shoulder deformity in children with severe OBPI, requiring surgery in more than one third of patients whose injury did not resolve spontaneously. This deformity is caused by fibrosis and contractures developed as a consequence of the neurological injury.

The current approach to treating persistent MRC in OBPI patients is derotational humeral osteotomy or anterior capsule release. Both of these surgical approaches have limitations. Humeral osteotomy attempts to improve the patient's passive range of external rotation, but ignores the bone deformity at the root of persistent MRC, and does nothing to address the attendant subluxation of the humeral head within the glenoid fossa. Anterior capsule release may result in excessive external rotation positioning of the humerus with attendant loss of internal rotation and midline functioning.

Supplemental Information

[Update on Nerve Repair by Biological Tubulization](#)

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[Brachial Plexus Injuries in Adults: Evaluation and Diagnostic Approach](#)

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Brachial Plexus Injuries

Post-Test

1. The brachial plexus is formed by the union of the _____ nerve roots. (p. 3)
 - A. Dorsal rami of C5, C6, C7, & C8
 - B. Ventral rami of C5, C6, C7, C8, & T1
 - C. Lateral rami of C5, C6, & C7
 - D. Posterior rami of C4, C5, C6, C7, & T1
2. Fascicles are collections of axons which are surrounded by endoneurium. (p. 4)
A True B. False
3. Most brachial plexus lesions are caused by _____. (p. 5)
 - A. Trauma to the neck and shoulder
 - B. Pseudoaneurysm of the subclavian artery
 - C. Radiation therapy
 - D. Neoplastic cancers
4. Approximately 70% of traumatic brachial plexus injuries are secondary to motor vehicle accidents. (p. 8) A. True B. False
5. An injured nerve with disruption of the axon, endoneurium, and perineurium, an intact epineurium, and no regeneration is classified as _____. (p. 12)
 - A. Seddon neurapraxia
 - B. Sunderland II
 - C. Sunderland IV
 - D. Seddon neurotmesis
6. Wallerian degeneration begins within hours of injury. (p. 13) A. True B. False
7. The success of regeneration from the proximal stump depends to a large degree on the distance from the injury site. (p. 14) A. True B. False
8. A patient presents with weak thumb, finger, wrist, and elbow extensors, and decreased triceps reflex. Where is the most likely location of the lesion? (p. 17)
 - A. Upper trunk
 - B. Middle trunk
 - C. Posterior cord
 - D. Lateral cord
9. Which of the following would be the best choice for assessing a possible nerve root avulsion injury? (p. 22)
 - A. Electromyography
 - B. Nerve conduction velocity
 - C. Computed tomography myelography
 - D. Sonography

10. Which surgical procedure typically has the best functional recovery? (p. 24-25)
- A. End-to-end repair
 - B. Nerve grafting
 - C. Nerve transfer
 - D. Neurolytic transposition
11. Neurogenic thoracic outlet syndrome is most often caused by impingement of the neurovascular bundle in the _____. (p. 29)
- A. Acromioclavicular joint
 - B. Intervertebral foramen
 - C. Inferior costoclavicular notch
 - D. Scalene triangle
12. Which test for TOS has the patient seated and actively performing shoulder abd to 90 degrees, wrist extension, and lateral head tilt? (p. 30-31)
- A. Elvey's test
 - B. Upper limb tension test
 - C. Roos test
 - D. Adson test
13. Most often, a flexed posture is seen in patients with TOS. (p. 32) A. True B. False
14. A child with Erb's palsy typically has difficulty performing _____. (p. 34)
- A. shoulder internal rotation and elbow extension
 - B. finger flexion
 - C. shoulder adduction, elbow extension, and wrist ulnar deviation
 - D. shoulder abduction and external rotation, elbow flexion, and wrist extension
15. The Leffert Classification Scale is the most commonly used system for assessing obstetrical brachial plexus injuries. (p. 34) A. True B. False

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