

# **NERVE INJURIES**

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Nerve injuries are quite common and may have serious implications for the patient. Most nerve injuries result from either acute injury or chronic cumulative trauma. For the family, especially for the person involved, it can be a bewildering situation with uncertainty about what is happening and what the future holds.

This resource book aims to improve understanding of what a nerve injury is, what causes it, how it affects the individual and what to be expected. However, it cannot replace the advice of the Physiotherapist and the team who are looking after a person who has had an injury.

**For family members and carers:** This resource book is predominantly aimed at the person with nerve injury but family, friends and carers play a huge role in helping recovery after nerve injury.

## **The Problem**

An injury to a nerve can result in a problem with the muscle or in a loss of sensation. In some people it can also cause pain. To understand the nerve injury and recovery, it is important to understand the different types of nerve injury. The type of nerve injury will determine the type of treatment that will be needed.

## **Anatomy**

Nerves connect your brain and spinal cord to the muscles and skin giving you movement and feeling. If there is an injury to the nerve, there will be an interruption in the information being conveyed to the skin or muscles to and from the brain. The larger nerves in your arm and leg, which are about the size of a pencil are made up of tens of thousands of nerve fibers, similar to the telephone cable and the nerve fibers are grouped together in fascicles. Some nerves like the median and ulnar nerve in

your arm have motor and sensory fascicles giving you movement and feeling to your hand.

## **Nerve Injury**

Two nerve injury classification systems have been described and they are outlined in Figure 1. A first degree injury or neurapraxia will recover quickly within days after the injury or it may take up to 3 months. The recovery will be complete with no lasting muscle or sensory problem. A second degree injury or axonotmesis will also have complete recovery however the recovery will be much slower than a first degree injury. The nerve must grow back to reinnervate the muscle or skin and nerves grow back at the rate of an inch per month, therefore the time for recovery will be much longer than with a first degree injury. A third degree injury will also have slow recovery however only partial recovery will occur. The amount of recovery will depend on a number of factors; for example, the more scarring in the nerve the more likely there will be poorer recovery and the potential mismatching of sensory and motor fibers and the less likely that the nerve will fully recover. A fourth degree injury occurs when there is dense scar tissue within the nerve completely blocking any recovery and a fifth degree injury is when the nerve is completely separated, like with a cut nerve. Both a fourth and fifth degree injury require surgery for recovery. A sixth degree injury is a combination the other types of nerve injury and recovery and treatment will vary depending on which type of nerve injury is present.

## **Nerve Recovery and Regeneration**

Following nerve injury, the nerve will try to repair itself by sprouting regenerating nerve units. These regenerating units will then try to grow down the nerve to reinnervate muscle or skin. If they make a correct connection, motor nerve to muscle or sensory nerve to skin, then recovery of muscle function and skin sensation will occur. If however, the regenerating nerve fibers do not make a correct connection then no recovery will occur. Nerves will regenerate at the rate of 1 inch per month. While sensation can be regained even after long periods of denervation, muscle reinnervation will not occur after long periods of time without nerve innervation. Therefore it is necessary to get nerve to muscle as quickly as possible if it is not going

to recover on its own. Often electrodiagnostic tests, including electromyography (EMG) and nerve conduction studies are used to see if the muscle is recovering. An EMG will show muscle recovery before you can see the muscle contracting. If no evidence of recovery is seen by 3 to 6 months following nerve injury, surgery is usually recommended.

### **Post-surgery Physiotherapy**

After surgery, you will have a soft bulky dressing at the surgical site for comfort and support. The dressing will be removed 2 or 3 days after surgery and depending on the surgery you may need a splint to hold your arm or leg still for a longer period of time. You may shower over the area and the stitches will be removed about 2 weeks after surgery. You will be instructed in range of motion exercises as indicated depending on the type of surgery that you had. If a nerve repair, nerve graft or nerve transfer was done, the area may be immobilized with a splint for 2 to 3 weeks, although some restricted movement is advised to prevent tight scar from developing around the nerve. In cases of a brachial plexus reconstruction, you will be in a shoulder immobilizer for 4 weeks to protect the repair of the pectoralis major muscle that was detached to allow for surgery on your brachial plexus. Supervised therapy will begin 3 to 4 weeks after surgery. As the nerves begin to grow, nerve recovery is monitored using a Tinel's sign. When the nerve is lightly tapped, you will feel an "electric tingling" feeling as the nerve regenerates.

If you have had surgery to get back sensation, supervised therapy is necessary to instruct you in specific sensory reeducation exercises. When you have some return of feeling, you will begin sensory reeducation to help you maximize the your sensation. As the feeling comes back, you may find some uncomfortable tingling or discomfort, similar to what you feel when your foot falls asleep or when the feeling comes back after you have had a local anesthetic. As your muscle reinnervates, you will be instructed in strengthening exercises to increase your strength. Initially, to relearn to use the reinnervated muscle you may be instructed exercises in gravity- eliminated positions. When the muscle recovers more strength, you will be instructed in exercises with more resistance including weights.

## **Making sense of the hospital process**

While the problems many people experience following a nerve injury can be straightforward, some people with complex injuries will need treatment from a very wide range of professionals.

The physician may make the initial diagnosis and refer the patient to a specialist. If the injury is complex, the patient will normally be admitted to a ward under a consultant physician and be treated by a wide range of professionals. Most people with nerve injury in hospital will be under the direct care of a doctor, often a neurosurgeon, but sometimes a plastic surgeon.

A person who has had a severe head injury or spinal cord injury may not be conscious. As a result, the immediate next-of-kin may be put in a situation where they have to try to understand both the immediate and long-term decisions involving a very wide range of hospital staff. When the critical phase is over, the patient will be referred for rehabilitation.

Understanding the effects of a nerve lesion can be difficult. An added burden is in trying to understand the health care system which can often be very complicated. Some people who have had associated injuries need treatments from a very wide range of specialists in the medical, nursing and therapy professions. Therefore, it is not surprising that those who have had strokes and their families need a lot of time to take in essential information about the immediate effects of the stroke and the long-term treatment programs.

## **NEUROPATHY**

### **What Is Neuropathy?**

Neuropathy means disease or abnormality of nervous system. Neuropathies can be sensory, motor or autonomic. Sensory nerves give us information about the position of our joints, pain and temperature. Motor nerves stimulate muscle contraction and movement. Autonomic nerves control functions that our bodies don't consciously regulate, such as sweating, certain bowel and bladder functions, and heart rate.

Symptoms of neuropathy depend on both the type of nerves affected and the mechanism that causes damage to the nerves. The most common presenting symptom is the combination of numbness and tingling in the toes and feet. Less common neuropathies can cause weakness or clumsiness, and it may be difficult to do certain activities, such as raising an arm over the head, getting up from a seated position or walking up stairs.

If you experience such symptoms, your doctor will likely refer you to a neurologist, who specializes in the diagnosis and treatment of these disorders. Neurologists with specialized training in neuromuscular diseases usually have the most experience in the diagnosis and treatment of neuropathy. A neurologist should take a detailed history about the type of symptoms and timing, perform an in depth neurological examination and order various tests such as an EMG (electromyography) and nerve conduction studies. This will help determine the cause and best course of treatment. Remember that symptoms can be similar in different types of neuropathies, which can make diagnosis challenging. This is why a thorough work up is so important.

### **What Does A Complete Neurological Exam and Workup Consist Of?**

**Complete health history:** This includes questions about your symptoms, including type, onset, duration and location. Specific details about what brings on the symptoms, what relieves them and the types of sensations that occur serve as clues to the diagnosis. A complete list of medications should also be provided in case the medication itself is the cause of the neuropathy.

**Neurological evaluation:** In addition to the history of the symptoms, the neurologist will also examine reflexes, strength and the ability to feel various sensations. Again, this aids in an overall diagnosis.

**Blood tests:** Certain lab tests can help determine the cause of the neuropathy. This may include tests for vitamin deficiencies, immune responses, blood sugar levels and the presence of toxins or infections.

**EMG:** An EMG, or electromyography, electronically measures and records muscle activity. This tells the neurologist the location of any muscle, nerve or neuromuscular junction damage as well as its cause.

**Nerve conduction studies:** This test measures the size and speed of electrical signals as they pass along the nerves. This tells the neurologist of any abnormality of the nerves. EMG and nerve conduction studies usually go hand in hand.

**MRI:** An MRI (magnetic resonance imaging) may be performed to rule out any other causes of the neuropathy, such as trauma or nerve entrapment and sometimes to show inflammation along the nerves.

**Lumbar puncture:** A spinal tap or lumbar puncture can determine the presence of protein and cells in the spinal fluid. This test is usually done if the doctor thinks the nerves are affected by inflammation.

**Nerve, muscle or skin biopsy:** A small piece of nerve, muscle or skin can help determine the cause of the damage. These tests are only done if the doctor suspects very specific conditions.

### **What Is Idiopathic Neuropathy?**

Idiopathic means of no known cause. This type of neuropathy is very common, making up about a third of all neuropathies. This diagnosis simply means that the exact causative factor is unknown. This may sound confusing, but an experienced neurologist can tell you about the prognosis and treatments of this common condition. Symptoms include numbness, tingling and pain starting in the toes and feet. Balance when standing or walking may be affected. There may also be muscle cramps. Once the neurologist rules out other causes and identifies the neuropathy as idiopathic, a

treatment plan is formulated, which usually consists of over-the-counter pain medications, as needed, and safety precautions due to balance issues and loss of sensation. The neurologist should also give reassurance that patients with idiopathic neuropathy have very slow progression and do not develop disability with time.

### **What Is Diabetic Neuropathy?**

Diabetic neuropathy occurs in patients with diabetes mellitus who have uncontrolled blood sugar levels. Sensory, motor and autonomic nerves can be affected, so symptoms can include numb and painful feet, weakness, indigestion, constipation, dizziness, bladder problems and impotence.

Workup may include EMG and nerve conduction studies, blood work to check sugar levels and a thorough neurological assessment. Treatment depends on which nerves are affected and the

type of symptoms and problems that the person experiences. The first step is to maintain blood

glucose levels within normal limits through compliance with diabetic medications and diet. It is vital to prevent further damage and problems from occurring. Further intervention can include proper foot care, treating indigestion and constipation with medications and dietary management,

possible antibiotics for any bladder infection and pain relief.

### **What is hereditary neuropathy?**

Hereditary neuropathies are genetic in origin, meaning that they are passed through the genes.

Examples include Charcot-Marie-Tooth Disease and Hereditary Neuropathy with liability to Pressure Palsies.

Charcot-Marie-Tooth (CMT) Disease is the most common hereditary disorder and affects both

motor and sensory peripheral nerves. Symptoms include weakness and atrophy in the feet and

lower legs and in the hands in more severe cases. Deformities of the foot result from loss of muscle bulk and changes in the shape of the bone structure. These are usually called “pes cavus.”

Symptoms usually appear in the teenage years or early to mid-adulthood. Progression is very slow. There are many different types of CMT depending on the part of the nerve affected. Some types of CMT are caused by damage to the nerve itself, other types are caused by damage to the coating of the nerve, which is called myelin. Treatment includes physical and occupational therapy, use of leg braces and use of other assistive devices to facilitate safety and to help offset some of the deformities that can occur.

Hereditary Neuropathy with Liability to Pressure Palsies is a disorder that makes someone more susceptible to pinched nerves, like carpal tunnel syndrome. These patients sometimes sustain excessive damage to nerves from moderate trauma, like sleeping the wrong way on a nerve.

Symptoms can last longer and occur more frequently than a limb just “falling asleep.” Diagnosing a hereditary neuropathy may include a comprehensive history that reveals the episodes of numbness or weakness and nerve conduction tests that show a very specific abnormality. Genetic blood testing is used to confirm the diagnosis. Treatment consists of education about the risk factors, and ergonomic training to avoid pressure related and repetitive movement injuries from everyday activities.

### **What Is Immune or Inflammatory Neuropathy?**

There are conditions where nerves are “attacked” by someone’s own immune system. Several types of inflammatory neuropathies may occur. The two most common are Chronic Inflammatory Demyelinating Polyneuropathy (CIDP) and Multifocal Motor Neuropathy (MMN). There are also several variants of CIDP.

CIDP causes weakness and sensory abnormalities that usually develop over several months. The

disease can stop progressing, and relapses and remissions can occur. The severity of CIDP can vary from mild to severe and it can affect any age group and either gender. Normally, CIDP is not painful, although some patients complain of unusual or troubling sensations. MMN is a disorder characterized by weakness and muscle atrophy that usually affects the hands. MMN can cause minimal weakness in just a few muscles, or may be relatively severe weakness in all limbs. Because it only affects strength, but not sensation, MMN is sometimes mistaken for ALS (Amyotrophic Lateral Sclerosis, or Lou Gehrig's disease). Unlike ALS, MMN is an immune neuropathy and responds to the same treatments. In both MMN and CIDP the attack occurs against the myelin sheath, which surrounds the nerve and provides insulation for nerve conduction. The attack can also interfere with specialized "ion channels" in nerves that help electrical signals pass up and down the long processes. As a result, electrical impulses that carry the electrical signals are damaged. The diagnosis of these disorders depends heavily on nerve conduction studies that prove nerve signals are abnormal. They also depend on a complete examination of the nervous system by a specialist who can recognize the unusual patterns of muscle weakness and sensory loss. A lumbar puncture is sometimes needed to check for high protein levels and, in rare cases, a nerve biopsy may be required.

For MMN, a specialized blood test called anti-GM1 antibodies is useful if it is positive.

One common treatment for MMN and CIDP is called IVIG (Intravenous Immunoglobulin). Many, but not all, cases will improve and the symptoms will decrease within a few weeks to a few months after IVIG is started. IVIG is given at intervals ranging from every two weeks to every other month. Responses tend to wear off so treatments need to be repeated. Because diagnosis can be difficult, some doctors consider the first few treatments with IVIG as part of the diagnosis. Patients who do not respond probably have a different type of neuropathy. Since IVIG is also very expensive, and the diseases can stabilize, it is important that the neurologist check for responses regularly to determine whether ongoing treatments are needed and what doses are necessary.

A steroid called Prednisone is often used to treat CIDP as well. Prednisone causes more side effects than IVIG but also works well to treat the disease. Prednisone cannot be used for MMN, since it does not work for that particular neuropathy. There are other medications that can treat immune neuropathy, and these may be used when the condition is severe or difficult to treat.

### **Treatment of Painful Neuropathies**

Pain is an important symptom of neuropathies that affect sensory fibers. The pain can be described as burning, lancinating, tingling, or shock-like. There are several medications that are specialized for managing nerve type pain. These include Elavil, Neurontin, Lyrica® and Cymbalta®, to name a few. More severe cases may require stronger narcotic medications.

### **Other Causes of Neuropathy**

There are numerous other types of less common neuropathies that we have not discussed above.

Neuropathy may be caused by certain vitamin or mineral deficiencies, medication toxicity such as chemotherapy, alcohol abuse, certain infections, and as a symptom of other systemic illnesses. Other causes include:

- Diabetic
- Alcohol
- Infection
- Autoimmune
- Nutritional
- Toxin induced
- Inherited
- Thoracic outlet syndrome
- Burners/Stingers

## COMMON TRAUMATIC PERIPHERAL NEUROPATHIES

### Compressive/Traction Injury of the Nerve

• 3 categories of nerve injury

▫ Neuropraxia : a transient episode of motor paralysis with little or no sensory or autonomic dysfunction. Full recovery expected.

▫ Axonotmesis: a more severe nerve injury with disruption of the axon but with maintenance of the sheath. Motor, sensory, and autonomic paralysis results. Recovery can occur if the compressing force is treated in a timely fashion and if the axon regenerates.

▫ Neurotmesis: The nerve and its sheath are disrupted. Although recovery may occur, it is never complete, secondary to loss of nerve continuity.

### Axillary Nerve

• Derived from the posterior cord of the brachial plexus at the C5/C6 level with occasional contribution from C4

### Axillary Nerve Injury

- Uncommon nerve injury representing <1% of all nerve injuries.
- More commonly injured with shoulder dislocation (19%-55%), humeral fracture (58%) or shoulder surgery
- EMG evidence of nerve injury in up to 80 percent of individuals
- Nerve injury may be subclinical due to masking by pain of shoulder fracture or dislocation

### Axillary Nerve Course

- Travels below the coracoid process across the anterior inferior subscapularis muscle then posteriorly through the quadrilateral space and divides into two branches.

- The anterior branch passes around the surgical neck of the humerus
- The posterior branch travels inferior to the glenoid rim before dividing into the upper lateral brachial cutaneous nerve and the nerve to the teres minor

• **Quadrilateral Space bordered by:**

- long head of triceps medially
- humeral shaft laterally
- teres minor superiorly
- teres major inferiorly
- subscapularis anteriorly

• **Branches**

- Anterior branch: motor innervation to the deltoid.
- Posterior branch: motor innervation to the teres minor
- Superior Lateral Cutaneous Nerve: sensory innervation over the inferior portion of the deltoid (upper, lateral arm)

• **Mechanism of injury**

▫ **Traumatic:**

- Direct - contusion to anterolateral deltoid or fracture
- Indirect - traction with shoulder dislocation

▫ **Compression:**

- Quadrilateral Space Syndrome: rare condition. compression of the posterior humeral circumflex artery and axillary nerve as they pass through the quadrilateral space etiology is unclear.

▫ **Iatrogenic (e.g., rotator cuff surgery)**

## •Symptoms

- Variable. May not complain of frank weakness.
- Early fatigue or weakness especially with overhead activities or abduction.
- Numbness at the lateral upper arm.
- Night pain is frequently reported.
- If insidious onset and intermittent, think Q.S.S.

## •Differential

▫cervical pathology (usually C5/C6), rotator cuff injury, vascular compression, suprascapular nerve injury.

## Axillary Nerve Examination

- Deltoid or teres minor atrophy with late presentation.
- Palpate the deltoid for contraction during the initiation of abduction.
  - Be sure to evaluate all three heads of the deltoid because nerve injury may not be uniform throughout.
- Evaluate sensation in the upper lateral arm.
- Weakness with
  - External rotation
    - 45% of strength is from teres minor
  - Abduction
  - Forward flexion
- If posterior deltoid and teres minor spared, lesion is distal to the quadrilateral space

## •Diagnostics

- Imaging
  - Radiographs to rule out fracture of the humerus.

MRI may reveal indirect indicators of nerve injury (e.g., fat and water composition in muscle).

▫NCV/EMG

Obtain at least three weeks after the injury

Repeat at three months if no clinical improvement

**•Conservative treatment**

▫Rest

▫Physical therapy

active and passive range of motion

strengthening

▫Electrical stimulation of the deltoid is an optional treatment

**•Indications for surgical consultation**

a symptomatic patient with no clinical or EMG/NCV evidence of recovery by 3 to 6 months

penetrating injury or iatrogenic cause.

- Surgery should occur within six months of injury as some studies have demonstrated poorer prognosis with delayed intervention.

**Axillary Nerve -Prognosis**

•Full recovery with nonoperative treatment in 85-100% within 12 months if associated with dislocation or fracture.

▫Poorer prognosis with respect to recovery of deltoid function if from direct contusion

Many of these patients returned to contact sports and activities despite deltoid paralysis.

•Return-to-function

▫depends on the associated injuries involved but full shoulder range of motion and good strength are recommended.

## Radial Nerve

- Arises from the posterior cord of the brachial plexus (C5-T1)
- Symptoms about the elbow may mimic lateral epicondylitis.
- 5-10% of patients with lateral epicondylitis have associated radial nerve entrapment

## Course

- Runs with the deep artery before passing into the cubital fossa and descends between the brachioradialis and brachialis. At the level of the lateral epicondyle, it branches into the superficial and deep branches
- Deep branch
  - courses around the neck of the radius and enters the posterior compartment terminating as the posterior interosseous, which continues under the supinator anterior to the proximal radius in the radial tunnel.
- Superficial branch
  - passes anterior to the pronator teres, piercing the deep fascia at the wrist and entering the dorsum of the hand.

## Branches

- Forearm branches:
  - motor innervation to the triceps brachii, anconeus, brachioradialis, extensor carpi radialis longus
- Posterior Interosseous Nerve:
  - motor innervation to the extensor carpi radialis brevis, extensor carpi ulnaris, extensor digiti minimi, extensor digitorum, supinator, extensor indicis proprius, abductor pollicis longus, and extensor pollicis longus and brevis
- Posterior Cutaneous Nerve:
  - sensory innervation to posterior arm and posterior forearm
- Inferior Lateral Cutaneous Nerve:
  - sensory innervation to lateral arm
- Superficial branch:
  - sensory innervation to proximal dorsal 3 ½ digits and dorsal hand

•**Mechanism of injury**

▫Traumatic

Direct -humeral shaft fracture

Indirect

▫Compression

Multiple possible sites of compression.

Repetitive pronation/supination increases forces.

May occur with tourniquet use, improper use of axillary crutches, and “Saturday night compression palsy”.

## **Symptoms**

- Symptoms about the elbow may mimic lateral epicondylitis.
- 5-10% of patients with lateral epicondylitis have associated radial nerve entrapment.

## **Posterior Interosseous Nerve Compression**

- Compressive injury seen in throwers and with repetitive use
- prodrome of lateral forearm or elbow pain
- weakness or paralysis of the wrist and digital extensors
- No sensory component
- throwers may note decreased control and velocity

### **•Examination**

- weakness with extension of the wrist, thumb and index finger
- pain with resisted extension of the middle finger or resisted supination of the forearm
- compression 4 cm below the lateral epicondyle may reproduce pain (arcade of Frohse)
- symptoms worsen if misdiagnosed as lateral epicondylitis and counterforce brace applied over site of entrapment

### **•Diagnostic Injection**

- lidocaine injection 4 cm distal to the lateral epicondyle will result in temporary PIN palsy and temporary relief of pain
- This should not occur with lateral epicondylitis

## **Wartenberg's Syndrome**

- May be caused by any tight fitting strap

### **•Symptoms**

▫pain and decreased sensation over the dorsoradialhand, dorsal thumb, and index finger

**•Examination**

▫Positive Tinel's over superficial radial nerve

▫No weakness

▫Pseudo-Finkelstein test can be positive

**•Imaging:**

▫Plain radiographs are generally negative in the absence of trauma but may reveal calcifications.

▫MRI is only helpful if a mass lesion is suspected.

**•NCV/EMG**

▫may be diagnostic in PIN.

▫Negative test does not rule out entrapment and sensitivity for PIN questioned.

**Radial Nerve Treatment**

PIN

- rest (may be assisted with splinting)
- avoidance of provocative movements
- NSAIDs
- physical therapy

Wartenberg's Syndrome

- Discontinue compressive force
- rest (may be assisted with splinting in supination)
- NSAIDs

**•Indications for surgical consultation**

▫lack of clinical or EMG improvement after 3 months of conservative treatment.

## •Prognosis

- Generally responds to conservative treatment.
- Limited case series show a good prognosis with surgical decompression.

## Median Nerve

- Arises from two cords of the brachial plexus, lateral (C6 and C7) and medial (C8 and T1). Occasional input from C5.

## Course

- Descends in close relation to the brachial artery on the medial side of the arm.
- Enters the cubital fossa medial to the brachial artery before passing between the heads of the pronator teres where it gives off the anterior interosseous branch.
- It then descends between the flexor digitorum superficialis (FDS) and the flexor digitorum profundus (FDP) giving off the palmar cutaneous branch before passing within the carpal tunnel to reach the hand

## Branches

- Forearm branches
  - motor innervation to the pronator teres, flexor carpi radialis, and palmaris longus
- Anterior Interosseous Nerve
  - motor innervation to the FDP (radial half), flexor pollicis longus (FPL), pronator quadratus
- Terminal motor branches
  - motor innervation to the FDS, abductor pollicis brevis, opponens pollicis, flexor pollicis brevis, lateral two lumbricals
- Palmer Cutaneous Nerve
  - sensory innervation to lateral palm
- Digital cutaneous branches
  - sensory innervation to volar and distal dorsal surfaces of the radial 3 ½ digits

## • Mechanism of injury

- Traumatic: Direct or indirect
- Compression:
  - Most commonly within the carpal tunnel (Carpal Tunnel Syndrome)
  - Also occurs at multiple sites including within the pronator teres (Pronator Syndrome) and along the anterior interosseous nerve (Anterior Interosseous Syndrome)

## Median Nerve – Carpal Tunnel Syndrome

- Etiology

- Most common nerve entrapment
- Occurs with compression within the carpal tunnel
  - flexor tenosynovitis
  - repetitive flexion or grasping can provoke symptoms

- Symptoms

- Paresthesias and weakness in the radial three and a half digits of the hand.
- Increased symptoms with repetitive movements. Pain may radiate proximally.
- Nighttime symptoms are common.

### **CTS Examination**

- CTS

- Visual exam
  - Thenar muscle wasting may be present in advanced cases
- Physical
  - Tinel's sign -palpation of the median nerve over the carpal tunnel eliciting symptoms
  - Phalen's sign -wrist flexion for 60 seconds producing paresthesias in a median nerve distribution

### **Median Nerve –Pronator Syndrome**

- Most common in patients engaged in repetitive elbow flexion, forearm pronation, and gripping
- Caused by compression at four potential sites:
  - Bicipital aponeurosis
  - Pronator teres hypertrophy –most common
  - Impingement at FDS
  - Compression by persistent median artery or enlarged bursa

- Symptoms

- Vague, aching pain in the volar aspect of the elbow and forearm
- Worsens with activity
- Two important sensory symptoms help to distinguish pronator syndrome from carpal tunnel syndrome
- If sensory involvement present involves thenar eminence
- Not typically associated with nighttime symptom

### •Examination

- Pronator compression test
  - compression over the pronator teres reproduces symptoms.
- Tenderness over the pronator
- Tinel's sign
- Reproduction of symptoms with resisted pronation in the extended elbow.
- Numbness in the thenar eminence

### Median Nerve -Anterior Interosseous Syndrome

- Motor nerve branch off median nerve 5cm distal to medial epicondyle
- Innervates FPL, pronator quadratus, and FDP to 2nd and 3rd digits
- Can occur with any activity requiring repetitive forearm pronation and wrist flexion
- Compression can occur at:
  - pronator teres
  - fibrous bands in FDS (most common)
  - accessory head of FPL
  - vascular anomalies

### •Symptoms

- Weakness in pinch and decreased pronation strength with elbow flexed
- Deep constant pain in the proximal volar forearm preceding gradual weakness of the FPL and FDP.
- Distinct lack of sensory complaints
- Due to anatomic variation of AIN, distinguishing between pronator syndrome and AIN syndrome sometimes difficult

### •Examination

- Weakness of the FDP and FPL manifesting as inability to make an appropriate circle with the index finger and thumb

### Diagnostics

#### •Imaging:

- Plain radiographs may reveal osteophytes or bony abnormalities causing compression.
- MRI should be obtained if suspicion exists for compressive masses.
- NCV/EMG are warranted with CTS and AIS but are unreliable with PS.
- New advancements utilizing the relative sensory latency differences may allow earlier detection of CTS.
- Can still have CTS with negative studies.

### Treatment

#### •*Carpal Tunnel Syndrome*

- Splinting the wrist in neutral position including at night, rest, physical therapy and anti-inflammatory medications.
- Corticosteroid injections into the carpal tunnel may relieve symptoms.

#### •*Pronator Syndrome*

- Rest, anti-inflammatory medications, and immobilization with elbow flexion at 90° and the forearm in neutral for 3-6 weeks.
- Corticosteroid injection is debatable.

#### •*Anterior Interosseous Syndrome*

- Same as PS.

- Indications for surgical consultation include failed conservative management and progressive symptoms.

- Specific time periods for trial of conservative treatment

- 6 months for CTS

- 3-6 months for PS and AIS

- these vary in the literature.

### **Median Neuropathy Prognosis**

- CTS

- Prognosis is variable but generally responds well to conservative treatment within a few months with very good prognosis expected if surgical treatment required.

- PS

- 50% resolution in 6 to 8 weeks with conservative treatment. Very good prognosis if surgical decompression required.

- AIS

- Frequently resolves with conservative management. Good prognosis if surgical intervention required.

These hand exercises may be used for hand problems that involve the median nerve such as carpal tunnel.

Do these exercises slowly and smoothly in the order listed. Move to the next exercise position only when you feel no pulling, pain or numbness.

Hold each position for 5 to 10 seconds.

Repeat each exercise as prescribed by your physiotherapist

### **Exercises**

Sit up straight in a firm chair. Hold your head up straight with your arms at your side.

Bend you elbow at a right angle or 90 degrees.

1. Make a full fist with all your fingers.



2. Hold your wrist straight and straighten your fingers so your thumb is to the side of your index finger.



3. Bend your wrist back and stretch your fingers with your thumb out to the side.



4. Turn your hand so your palm is facing you and continue to bend your wrist back and stretch your fingers with your thumb out to the side.



5. Continue as in exercise 4 but extend your wrist back a bit further.



6. Continue as in exercise 5 while pushing your thumb out gently with your other thumb.



## **Ulnar Nerve**

- arises from the C8 and T1 roots. Contribution from C7 is not uncommon.
- 2nd most common compressive neuropathy of upper extremity is ulnar neuropathy at the elbow

### **Course**

- Descends distally, medial to axillary artery, piercing medial inter muscular septum at mid-arm level. After superficially passing posterior to the medial epicondyle of the humerus within the cubital tunnel, it enters the anterior compartment of the forearm between the heads of the flexor carpi ulnaris (FCU).
- Descending between the FCU and flexor digitorum profundus(FDP), the nerve gives off the palmar cutaneous branch mid-forearm and then the dorsal cutaneous nerve.
- After passing through Guyon's canal at the wrist, the nerve splits into superficial sensory and deep motor branches.
- The nerve may move as much as 7 mm anterior-medially, and lengthen as much as 4.7 mm during flexion.

### **Branches**

- Forearm branches
  - motor innervations to FCU and FDP (ulnar half)
- Superficial Motor Branch
  - motor innervations to the Palmaris brevis
- Deep Motor Branch

▫motor innervations to the hypothenar muscles, adductor pollicis, all interossei, medial two lumbricals, and deep head of flexor pollicis brevis

•Dorsal and Palmar Sensory Branch

▫sensory innervations to medial palm/dorsal hand and volar and distal dorsal surfaces of ulnar 1 ½digits

•**Mechanism of injury**

▫Trauma: Direct and indirect trauma (i.e. traction, friction)

▫Compression

□Elbow

□ulnar groove (e.g., bone spurs), below the medial epicondyle(i.e. cubitaltunnel) and above the medical epicondyle(i.e. “arcade of Struthers”)

□Wrist

□Guyon’s canal. May also occur with muscular hypertrophy of the FCU, ulnar artery aneurysm, lipoma, etc.

**Cubital Tunnel**

•Roof –fascia of FCU and arcuate ligament of Osborne (a.k.a cubital tunnel retinaculum)

•Walls –medial epicondyle and olecranon

•Floor –elbow capsule and UCL

**Ulnar Nerve –Cubital Tunnel Syndrome**

•Common in baseball pitchers due to the large valgus stress at the elbow and repetitive flexion/extension

•**Mechanism**

- Compression of nerve at edge of flexor carpi ulnaris aponeurosis or arcuate ligament
- Distance between olecranon and medial epicondyle increases by 1cm
- UCL buckles medially
- Bulging combined with tightened arcuate ligament □ nerve compression

### •Symptoms

- Pain at the medial joint line or paresthesias during late cocking or early acceleration may occur
- Radiation to the hand is common
- Snapping or popping sensation
- Sleeping with elbows fully flexed can increase symptoms
- Throwing arm feels clumsy/heavy

### •Examination

- Inspect for elbow flexion contracture or valgus carrying angle
- Palpate ulnar nerve in cubital tunnel and check for tenderness
- Tinel's sign
- Check for subluxation by palpating nerve with flexion
- Elbow flexion test –hold elbow in maximal flexion and wrist in extension for 1 min
- Dorsal symptoms rule-out ulnar entrapment at wrist
- Motor findings only in chronic cases (weak 5th digit abduction, intrinsic atrophy)

## Ulnar Tunnel Syndrome

- AKA Guyon's Canal Syndrome, handlebar or cyclist's palsy
- Caused by compression on the hypothenar eminence or from prolonged hyperextension of the wrist.
- Also seen in racquetball and wheelchair athletes

## •Symptoms

- paresthesias and pain in the fourth and fifth digits
- decreased grip strength (40% of the grip strength is derived from ulnar nerve musculature)
- pain at the volar wrist ulnar aspect
- Fourth and fifth digit abduction and adduction weakness imply poorer prognosis.

## Guyon's Canal

- Fibro-osseous tunnel bordered by:
  - Volar carpal ligament
  - Pisiform
  - Hook of the hamate
- Nerve lies superficially; soft tissue coverage is sparse

## Ulnar Tunnel Syndrome

- Other causes of compression at Guyon's Canal:
  - Ganglioniccysts (30% -*Shea and McClain*)
  - Tumors
  - Blunt injuries with or without fracture
  - Ulnar artery thrombosis
  - Idiopathic

## •Examination

- Pain and parasthesias in 4<sup>th</sup> and 5<sup>th</sup> digits
- Decreased grip strength
- Pain and tenderness over volar aspect of pisiform and hamate
- Tinel's at Guyon's canal
- Wartenberg's sign –weakness of 3<sup>rd</sup> interosseous
- Clawing in profound cases (weak ulnar intrinsics vs unopposed FDP)

▫Hypothenar and interosseal atrophy

Froment's sign: weakness of adductor pollicis brevis with substitution by FPL

## **Ulnar Nerve Diagnostics**

- Imaging:

- Choice of imaging is based on lesion location.
- Plain radiographs may reveal bony changes about the elbow (ulnar sulcus or cubital tunnel view) and osteophytes or hook of hamate fracture on carpal tunnel views of the wrist.
- MRI studies and angiograms may reveal occult hamate fracture and/or ulnar artery thrombosis.

- NCV/EMG:

- May help localize the injury and monitor recovery.
- Utility at Guyon's canal is limited due to technical difficulty of the exam.

## **Treatment**

- Conservative treatment

- Cubital Tunnel Syndrome:

- rest, ice, anti-inflammatory medications, and physical therapy.
- Splinting in mild flexion (elbow pad worn in "reverse," or fiberglass volar flexion "block" splint) or an elbow pad to avoid pressure on the cubital tunnel may help.

- Ulnar Tunnel Syndrome:

- same as above, plus wrist splinting in position of function with slight dorsiflexion.
- Corticosteroid injection into Guyon's canal may be considered but often yields only transient relief.
- In cases of cyclist's palsy, use of padded gloves, specialized grips, altering and frequently changing hand position, and appropriate bicycle fitting.

- Indications for surgical consultation**

- failure to respond to conservative treatment
- persistent motor weakness
- intrinsic paralysis or progression of symptoms

□ Surgical intervention for cubital tunnel syndrome frequently includes decompression of the nerve and/or anterior transposition for neuropathy associated with elbow deformity or subluxation of the nerve.

• Prognosis is individual and multifactorial

## **Tibial Nerve**

- derived from the L4-S3 roots as part of the sciatic nerve.

### **Course**

- Branches from the sciatic nerve in the distal thigh and continues through the popliteal fossa entering the calf between the two heads of the gastrocnemius muscle.
- After passing deep to the soleus, it continues in the posterior compartment between the tibialis posterior and the soleus muscles.
- At the medial ankle, the nerve becomes superficial, before passing into the foot through the tarsal tunnel. Within the tunnel it splits into the medial and lateral plantar nerves.
  - The medial plantar nerve divides into muscular and cutaneous branches.
  - The lateral plantar nerve passes between the quadrates plantae and flexor digitorum brevis before dividing into superficial and

### **Branches**

- Direct branches
  - motor innervations to the semimembranosus, semitendinosus, biceps femoris(long head), plantaris, popliteus, gastrocnemius, soleus, tibialis posterior, flexor hallucis longus, and flexor digitorum longus
  - sensory innervations to posterolateral calf via sural branches.
- Medial Plantar Nerve
  - motor innervations to the abductor hallucis, flexor digitorum brevis, flexor hallucis brevis and first lumbrical
  - sensory innervation to the medial sole and medial 3 ½ toes.
- Lateral Plantar Nerve
  - motor innervation to the quadrates plantae, flexor digiti minimi, adductor hallucis, interossei, abductor digiti minimi, and lateral three lumbricals
  - sensory innervations to the lateral sole and lateral 1 ½ toes.
- Medial Calcaneal Nerve

▫sensory innervations to the plantar and medial heel.

### •Mechanism of injury

▫Traumatic

▫direct or indirect trauma (most commonly to the distal tibia or ankle), overuse

▫Compression

▫mass lesion (e.g., varicosity, synovial thickening, ganglion cyst) or exostosis, accessory flexor digitorum longus, varicose veins, over pronation, rear foot valgus deformity, muscular hypertrophy

▫Systemic disorders: rheumatoid arthritis, diabetes, etc

## Tarsal Tunnel Syndrome

### •Etiology:

▫Entrapment at tarsal tunnel is the most common entrapment neuropathy in the foot and ankle, and extremely common in sport

▫Injury or entrapment of tibial nerve at the distal thigh, knee and leg are rare

▫Majority of TTS cases are secondary to external pressure, mass lesion, or bony/ligamentous trauma to ankle

▫Foot and ankle position (i.e. rear foot pronation) also shown to influence tarsal tunnel compartment pressure

▫Sports such as sprinting or jumping (repetitive dorsiflexion of ankle and rear foot pronation) can compress nerve

▫Idiopathic fibrosis or ligamentous thickening also found to be causes

### •Symptoms

▫burning pain on the medial plantar foot usually of insidious onset

▫worse with prolonged standing or walking

▫proximal radiation to the calf is not uncommon

▫motor weakness is generally not reported but weakness of toe flexion may occur

▫occasional night pain

### **Examination**

- Advanced cases may reveal atrophy of intrinsic foot muscles.
- Specific tests:
  - Pain with extremes of dorsiflexion
  - Tinel’s sign behind medial malleolus at the tarsal tunnel
  - Decreased sensation along the planter aspect of the foot
  - Tenderness and/or mass or swelling at the tarsal tunnel
  - Occasionally weakness with great toe plantar flexion.

### **Diagnostics**

- Imaging
  - Plain radiographs may reveal exostoses, malunions, or osteophytes
  - MRI to rule out mass lesions.
- NCV/EMG for TTS may show prolonged conduction.
  - Approximately 90% of patients with tarsal tunnel syndrome have abnormal findings on EMG/NCV studies

## **Treatment**

- Conservative treatment

- Rest
- Change in footwear or running posture
- Orthotic with medial support
- Splinting
- NSAIDs
- Corticosteroid injection.

- Indications for surgical referral include mass lesions and failed conservative treatment after 3-6 months.

## **Prognosis**

- Conservative treatment is successful in the majority of cases.

- Return to Play

- If surgical intervention is required, approximately 80-90% patients will experience improvement or resolution of symptoms.

- This estimate drops to 75% if the specific cause is not known.

## **Less common tibial nerve injuries**

- Medial Plantar Nerve (MPN) injury (“jogger’s foot”)**

- Burning heel pain, aching in the arch, and decreased sensation in the plantar foot behind the great toe

- compression occurs distal to the tarsal tunnel, most commonly in the abductor tunnel behind the navicular tuberosity.

- Exam shows tenderness of the MPN at the entrance to the abductor tunnel or overlying naviculocalcaneal ligament and weakness of intrinsic foot musculature. Difficult to differentiate from plantar fasciitis.

- Lateral Plantar Nerve (LPN) injury**

▫Symptoms may include decreased sensation at the lateral one third of the plantar foot. There may be weakness of the abductor digiti quinti but this is difficult to determine.

## **Common Fibular Nerve**

- derived from the L4-S2 roots as part of the sciatic nerve.
- Also referred to as the peroneal nerve
- Most common compressive neuropathy in the lower extremity

### **Course**

- Branches from the sciatic nerve in the upper popliteal fossa before giving off a lateral sural cutaneous branch, which becomes part of the sural nerve.
- Traveling posterior to the fibular head, the nerve enters the peroneal (fibular) tunnel between the two heads of the peroneus(fibularis) longus. Upon entering the tunnel the nerve splits into the superficial and deep fibular nerves.
- Traveling within the lateral compartment, the superficial branch runs between the fibula and the peroneus longus muscle and continues distally along the anterior intermuscular septum eventually piercing deep fascia at the distal third of the leg to become subcutaneous.
- Deep fibular nerve (DFN) pierces the anterior inter muscular septum and travels on the interosseous membrane in the anterior compartment before crossing the distal end of the tibia and continuing under the extensor retinaculum and through the anterior tarsal tunnel (a flattened space between the inferior extensor retinaculum and the fascia overlying the talus and navicular) to enter the foot.

### **Branches**

- Direct branches
  - motor innervations to biceps femoris(short head)
  - sensory innervations to lateral leg
- Lateral Sural Cutaneous
  - sensory innervations to the lateral and posterior leg
- Deep Fibular Nerve
  - motor innervations to tibialis anterior, extensor hallucis longus and brevis, peroneus(fibularis) tertius, and extensor digitorum longus and brevis

▫sensory innervation to 1<sup>st</sup> web-space.

- Superficial Fibular Nerve

▫motor innervations to peroneus longus and brevis

▫sensory innervations to dorsum of foot and toes except lateral 5<sup>th</sup> toe and 1<sup>st</sup> web space.

- Mechanism of injury**

▫Traumatic

Direct contusion

Repetitive motion injury

Stretch injury -mostly occurring where the nerve passes through the peroneus longus muscle.

May occur with knee dislocations, fibular fracture, proximal tibiofibular instability and severe ankle inversion.

▫Compression

Most common at peroneal tunnel.

Also occurs with internal masses (e.g., fabella), casting, weight loss, and after prolonged bed rest or prolonged positions such as squatting, kneeling or sitting cross-legged.

▫Iatrogenic: During surgical approach (i.e. knee arthroscopy) or due to positioning.

- Common Fibular Nerve Injury**

- Most common cause of compressive neuropathy in the lower extremity

- Occurs with compression at the peroneal tunnel

▫Repetitive exercise of plantar flexion and ankle inversion tenses peroneus longus and compresses CPN against fibular neck

▫Associated with high-mileage runners and cyclists

▫Habitual leg crossing, prolonged hospitalization, knee surgery, ganglion cyst, etc

- Symptoms**

- Partial or complete foot drop
- May be insidious or acute (may present as tripping or falls)
- Lateral lower leg and dorsal foot paresthesias are common

#### •Examination

- weakness of ankle and toe dorsiflexors and ankle eversion
  - Worse after exercise
- hypoesthesia to touch and pain in the lower two thirds of the lateral leg and dorsum of the foot
- Tinel's sign may be positive (fibular head)

### Superficial Fibular Nerve

#### •Superficial Fibular Nerve (SFN) injury

- Etiology usually undetermined but may be associated with fascial defect.
- Associated with lateral compartment syndrome.
- Stretch injury -prolonged kneeling or squatting, repetitive ankle sprains

#### •Symptoms

- Pain or paresthesia over the lateral calf, lower leg, and/or dorsum of the foot with resisted ankle dorsiflexion and eversion.
- No motor symptoms

#### •Symptoms

- Pain or paresthesia over the lateral calf, lower leg, and/or dorsum of the foot with resisted ankle dorsiflexion and eversion.

#### ▫Examination

- Percussion along course of nerve may result in positive Tinel's
- Muscle bulge due to fascial defect
- Weakness infrequent; associated with more proximal entrapment



## **Deep Fibular Nerve**

- Deep Fibular Nerve injury (Anterior Tarsal Tunnel Syndrome)
  - Pain over the dorsomedial aspect of the foot with radiation into or numbness within the first web space.
  - Occurs with compression of the nerve at the ankle as it passes the talonavicular joint.
  - Associated with anterior compartment syndrome.

### **•Examination**

- weakness of the extensor digitorum brevis muscle
- sensory deficit in the first web space
- ankle eversion normal
- Tinel's sign may be positive at the DFN (anterior compartment, mid-distal tibia)

## **Diagnostics**

### **•Imaging**

- Plain radiographs to rule out fracture or compressive exostosis
- MRI should rule out mass lesions.

### **•NCV/EMG**

- Considered the gold standard with fibular nerve injuries.
- Compartment pressure testing may be warranted if clinically suspected.

### **•Conservative treatment**

- avoidance of continued compression by object or position (this may include use of protective pads)
- rest, anti-inflammatory medications and physical therapy.
- Wearing a looser shoe for DFN compression may help.
- Corticosteroid injections are also utilized
- AFO if foot drop or contractures

### •Indications for surgical referral

- nerve laceration
- compression by a mass lesion
- lack of clinical improvement with conservative measures
- concurrent anterior compartment syndrome with DFN lesions may require decompression.

### Prognosis

- Resolution should be expected within 2-6 months depending on the etiology.
- SFN is less likely to respond to conservative treatment and usually requires surgery.
- If surgical intervention necessary, generally a good response is expected but this may vary with site of compression and degree of palsy.

### Suprascapular Nerve

- Originates from the superior trunk of the brachial plexus (C5 and C6 with variable contribution from C4)
- 1-2% of all shoulder disorders causing pain are due to supra scapular nerve entrapment
- Reported in throwing athletes and those exposed to repetitive trauma
- Although isolated nerve injuries of the supra scapular nerve are uncommon, most frequently injured peripheral branch of the brachial plexus in athletes.
- Up to 45% examined volleyball players had evidence of infraspinatus muscle impairment

### Nerve Anatomy

- Travels laterally across the posterior triangle of the neck, through the *supra scapular notch* and under the superior transverse scapular ligament before sending off branches to the supraspinatus.
- then travels around the lateral margin of the base of the scapular spine (*spinoglenoid notch*) to enter the infraspinatus fossa where it branches to supply the infraspinatus muscle

## **Nerve Function**

- Innervates the supraspinatus and infraspinatus with sensory branches to the acromioclavicular and glenohumeral joints.

- Although generally considered to have no cutaneous sensory component, sensory innervations of the proximal-lateral one-third of the arm is reported in 15% of patients.

## **•Mechanism of injury**

- Trauma

- Direct -scapular or clavicular fracture, shoulder dislocations, penetrating trauma

- Indirect -traction or repetitive overuse

- Proposed “sling effect”referring to friction of the nerve against surrounding structures of the suprascapular notch with depression and retraction, or hyper abduction, of the shoulder

- Compression: usually via surrounding ligaments or mass lesions (e.g., cyst lipoma, fibrous band)

- Iatrogenic

## **•Symptoms**

- dull pain at the posterior aspect of the shoulder exacerbated by overhead maneuvers and/or weakness of the affected shoulder, especially external rotation and abduction.

- Atrophy present in up to 80% of patients. Onset is usually insidious but may follow acute event.

- Suprascapular notch lesion-atrophy of both infraspinatus and supraspinatus

- Spinoglenoid notch lesion-atrophy of the infraspinatus only

## **•Differential**

- cervical spine pathology, brachial plexopathy, biceps tendonitis, adhesive capsulitis, impingement syndrome, rotator cuff and intra-articular gleno humeral pathology

## **Examination**

- Visual inspection may reveal atrophy of the infraspinatus or supraspinatus.
  - Atrophy of the infraspinatus better visualized from above or behind.
- Physical exam may reveal:
  - Tenderness at the site of injury. Cross-arm adduction may increase pain.
  - Physical exam findings are similar to impingement syndrome.

## **Diagnostics**

- Considered a diagnosis of exclusion.
  - May try injection of a local anesthetic into the suprascapular notch for diagnostic purposes but is nonspecific.
- Imaging:
  - Radiographs are usually normal.
    - An AP radiograph directed caudally at 30° or a Stryker notch view may be useful to assess the shape of the suprascapular notch.
  - MRI is best for revealing both mass lesions and other rotator cuff or soft tissue pathology.
  - Ganglion cysts causing suprascapular nerve compression are often associated with labral tears.
  - Ultrasound may also be useful but is operator dependent.
- Nerve conduction velocities and electromyography (EMG) may confirm diagnosis and/or exclude other pathology but results may not support clinical findings.

## **Treatment**

- Initial treatment is conservative
  - avoidance of aggravating activities, anti-inflammatory medications and rehabilitation for stretching and strengthening of surrounding structures.
- Structural lesions (e.g. labral pathology) correlating with clinical symptoms should be treated operatively.

▫Non operative treatment of ganglion cysts has a high failure rate and cysts are frequently drained surgically.

•If symptoms not due to known structural lesion or atrophy continue for more than 6 months, surgical exploration should be considered.

▫Follow up EMG may aide in the decision to proceed with surgical options.

## **Prognosis**

- The natural history of the injury varies greatly
  - Generally resolution of symptoms in the absence of a mass lesion is expected within 6-12 months of the diagnosis.
- The patient may continue their chosen sport/activities at a competitive level despite muscle atrophy if appropriate strength permits safe participation.
  - Return to play is individual and activity specific.

## **Spinal Accessory Nerve**

- The spinal accessory nerve is a pure motor cranial nerve.
  - Enters the foramen magnum and then exits the jugular foramen before passing through the upper third of the sternocleidomastoid. The nerve then assumes a subcutaneous course in the posterior cervical triangle to the trapezius.
  - Provides motor innervations to the trapezius and sternocleidomastoid(SCM).

### **•Mechanism of injury**

- Traumatic:
  - Direct -penetrating or blunt trauma to the posterior neck (hockey or lacrosse stick)
  - Indirect -traction
- Compression: Mass lesions or external (e.g., backpack)
- Iatrogenic: Subject to insult during lymph node dissection, carotid end arterectomy, etc.

### **•Symptoms**

- pain around the shoulder, weakness, difficulty with abduction and overhead activities, and sagging of the shoulder.
- Impaired athletic function, particularly if sport involves use of upper extremity at or above eye level
- Radicular pain due to traction from the drooping shoulder may also occur.

### **•Differential**

- Long thoracic nerve injury, cervical pathology.

## **Examination**

- Visual inspection
  - Asymmetry of the neck line, drooping of the affected shoulder, loss of normal scapulohumeral rhythm and “lateral”scapular winging.

▫Atrophy of the trapezius and SCM with associated spasm of the levator scapulae and rhomboids may be present

.

•Physical exam

▫Weakness with abduction and forward elevation and inability to shrug the affected shoulder.

▫Winging of the scapula may occur but is generally not as severe as that seen with long thoracic nerve injury.

▫Winging associated with spinal accessory nerve injury is not exaggerated by forward elevation as is seen with long thoracic etiology.

▫Scapular stabilization against the back by the physician may relieve symptoms.

**How to differentiate from long thoracic nerve palsy:**

•Abduction

▫winging

•Forward elevation

▫no winging (winging present in long thoracic)

•Winging of superomedial border (inferior border with long thoracic)

•**Imaging:**

▫Radiographs of the cervical spine, chest and shoulder are indicated but usually negative.

▫MRI only if ruling out mass lesion.

•NCV/EMG are generally diagnostic.

▫Repeat studies at 6-week intervals may be helpful in assessing recovery and need for surgical intervention.

**Treatment**

•Conservative treatment

▫anti-inflammatory drugs, electrical stimulation, limitation of overhead activities and physical therapy focusing on shoulder girdle and scapular rehabilitation.

▫sling may be used as needed but be wary of frozen shoulder.

▫braces to stabilize the scapula have not been highly effective.

**•Indications for surgical consultation**

▫symptoms and atrophy continuing for more than 6 months, penetrating trauma and iatrogenic injury.

**Prognosis**

•Injuries due to blunt trauma typically recover in less than one year with conservative treatment.

▫With injury due to penetrating trauma or laceration, there is improved prognosis if surgical intervention is not delayed more than 6 months.

•Return to play is individual and activity specific.

## **Long Thoracic Nerve**

- The long thoracic nerve arises from the anterior branches of C5 through C7
- 20% has contributions from intercostals nerves

### **Nerve anatomy**

- At 22 to 24 cm in length, it descends posteriorly to C8 and T1 rami, passes beneath the clavicle and continues distally on the external surface of serratus anterior
- Innervates the serratus anterior which protracts and rotates the scapula as well as stabilizing the scapula in abduction

### **•Mechanism of injury**

- Traumatic
  - Direct -contusion to shoulder or lateral thorax
  - Indirect -stretching of the nerve can occur with the head tilted/turned away, arm raised overhead
  - Traction position common in throwing a baseball, serving in tennis, spiking in volleyball and reported in yoga
- Compression: Multiple possible areas for compression by surrounding structures, crutches or inflamed bursa.
- Iatrogenic

### **•Symptoms**

- classic winging of the scapula or popping of the scapula during movement secondary to compromised gleno humeral biomechanics.
- shoulder, neck, and/or scapular area pain lasting up to a few weeks followed by insidious weakness with overhead activities or forward elevation (e.g. loss of throwing speed/power)

### **•Differential**

▫cervical disk disease, rotator cuff pathology, brachial neuritis (Parsonage-Turner syndrome), adhesive capsulitis, gleno humeral instability or arthritis, acromio clavicular joint arthritis, thoracic outlet syndrome.

## **Examination**

- Visual inspection

- winging of the affected scapula exaggerated by forward elevation or pushing off a wall.

- atrophy of the serratus anterior may be visible.

- Physical exam

- weakness with forward elevation often limiting movement to 110 degrees.

- altered scapula humeral rhythm may be found.

- Winging is associated with both long thoracic and spinal accessory nerve injuries.

- Winging associated with spinal accessory nerve injury is generally not exaggerated by forward elevation as is seen with long thoracic etiology.

## **Diagnostics**

- Considered a clinical diagnosis.

- Imaging:

- Plain radiographs generally normal.

- CT and MRI generally not useful unless ruling out other pathologies.

- NCV/EMG may confirm the diagnosis and track recovery.

- Include the spinal accessory nerves in the EMG.

## **Treatment**

- Conservative treatment

- rest, reassurance, anti-inflammatory medications or medications for neurogenic pain, maintaining range of motion, and strengthening of scapular stabilizers.

- Bracing may be considered for improved function or pain control. Evidence inconsistent.

**•Indications for surgical consultation**

▫symptoms persisting beyond one year with no improvement on EMG and iatrogenic injury.

**Prognosis**

- Isolated atraumatic long thoracic nerve palsy generally resolves in one to two years.
- Return-to-Play decision individualized and should be based on the athlete's strength and the demands of their sport.