ADVERSE NEURAL TENSION
ADVERSE NEURAL TISSUE TENSION

Theory and concept of Adverse Neural Tissue Tension (ANTT) will be discussed. Anatomy, biomechanics and pathology of the nervous system will be covered. What is a positive tension sign and is it relevant? We will use clinical reasoning to attempt to answer this question. The upper and lower limb neurodynamic testing including the Slump test will be covered. We will integrate the use of manual therapy techniques including but not limited to: soft tissue mobilization (STM), joint mobilization and joint manipulation with neural tension treatment. Lab time will include palpating nerves, performing neurodynamic testing and mobilization of the nervous system.

Course objectives

• Introduce the concept of Adverse Neural Tissue Tension.
• Be able to perform neurodynamic tests for upper and lower limbs, including the Slump test and modified slump test.
• Understand the theoretical meaning of positive tension signs and their relevance.
• Be able to perform a variety of manual therapy treatment techniques for adverse neural tissue tension pathology.
• Develop clinical reasoning skills to include ANTT.

Definition

Adverse neural tissue tension is an abnormal physiological and/or mechanical response from the nervous system that limits its normal range of motion or stretch capabilities. (Butler 1991)

Effects of treatment

• Improve vascularization of the nervous system and surrounding structures
• Improve axoplasmic flow
• Improve the mechanical properties of the nervous system

History

• Nerve stretching was in vogue in the late 1800’s in France and England. Techniques were usually used for the sciatic nerve and brachial plexus. Treatment was used for a variety of diagnoses like sciatica and ataxia.
• Braggard in Germany described ULNT for ulnar nerve. (1929)
• Cyriax developed the notion of dural pain.
• Phalen described carpal tunnel syndrome in the 60’s.
• Brieg’s work on biomechanics of the nervous system in the 60’s and 70’s.
• Sunderland’s work of the internal structure of the peripheral nerve and the role of ischemia in entrapment lesions. (1970’s)
• Maitland: Slump test (late 1970’s)
• Elvey: Upper limb tension test. (1979)
• Lundborg, Ryderik and Dahlin: recent work on vascular factors and axoplasmic transport in nerve injuries. (1980’s)
• David Butler PT, 80’s and 90’s. Published Mobilization of the Nervous System 1991 and The Sensitive Nervous System 2000.

**Anatomy and biomechanics**

• Peripheral and central nervous systems need to be considered as one.
• The connective tissues are continuous, although in different formats such as the epineurium and the dura mater.
• Neurons are connected electrically; an impulse at the foot ends up in the brain.
• The same neurotransmitters exist peripherally as well as centrally, and there is a flow of cytoplasm inside the axons (axoplasmic flow).
• The spinal canal is 5-9 cm. Longer in flexion than in extension.
• The epineurium forms a distinct sheath, well differentiated from surrounding fascia. Considerable ROM of the nerve trunk in relation to the neighboring fascia is allowed.
• The nervous system consumes 20% of the available O2 in blood, yet comprises only 2% of body mass.
• Blood supply is necessary for impulse conduction and for intracellular movement of the axoplasm.
• A critical vascular zone exists at T4-9. Here the spinal canal is at its narrowest and the blood supply is less rich in this area (may be relevant in the so-called “T4 syndrome”).
• Korr suggested that many disorders we treat and the responses from treatment may be related to axonal transport systems. He proposed treating both joint and neural structures.
• Spinal dura mater is layered with axially directed collagen fibers. It possesses great axial strength and little elasticity.
• Collagen fibers of pia and arachnoid mater are arranged in a lattice pattern that allows lengthening and shortening.
• Tension in peripheral nerves transmitted to spinal dura matter via perineurium, which is continuous with the dural sleeve.
• Nervous system is attached to surrounding tissue in a manner that allows mobility yet can impact resistance.
• Nerve root and dural sleeve: Sequestered discs can have an auto immune reaction to their nucleus pulposus. Centrally, lymphatic drainage of the nerve roots is poor, any inflammation to the endoneural space will predispose the nerve to fibroblast activity and development of adhesions.
• Nerves have an inherent elasticity and ability to elongate.
• Nerves move relative to their interfacing structures.
• Mechanical double crush (Butler). Upton and McComas 1973
Tension points: areas where there is minimal or no movement of the nervous system in relation to its surrounding interface. In the spine, tension points exist at C6, T6 and L4. Peripherally, tension points exist for example at the ulnar groove and the superior tib/fib joint.

Breig showed considerable movement of the lumbar sympathetic nerve trunk during SLR and ANS implicated as susceptible to injury in extension phase of whiplash injury in animal models.

Dura innervated by sinuvertebral nerve. Branches also innervate PLL, periosteum, blood vessels and annulus fibrosis.

Pathology

Mechanical interface: tissue or material adjacent to the nervous system that can move independently to the system. Example: the supinator muscle interfaces with the posterior interosseous branch of the radial nerve as it passes through the radial tunnel.

The ligamentum flavum and the facet joints interface with the posterior aspect of the spinal dura mater.

Pathological interfaces: osteophytes, ligamentous swelling, fascial scarring, tight plaster or bandage.

Tension points.

Cadaveric proof that cervical flexion can influence lumbosacral nerve roots (Breig).

Dorsiflexion can tension the nervous system up to and including the cerebellum (Smith, Breig)

“Sciatic brachialgia” - leg, arm and/or neck pain with SLR.

“Brachialgic sciatica” - sciatic pain with cervical flexion.

Where nerve branches enter a muscle at abrupt angles there is likely to be less movement.

Sympathetic symptoms: because of connections with CNS, these symptoms can be somewhat bizarre, like nausea, sweating, color and temperature changes.

Vulnerable anatomic sites of injury

Soft tissue, osseous or fibro-osseous tunnels. Example: posterior interosseous nerve through the arcade of Frohse, carpal tunnel, ulnar groove. At tunnel sites there is a greater potential of spatial compromise.

Where the nervous system branches, particularly if the branch leaves the trunk at an abrupt angle. To branch, a nerve sacrifices some of its gliding mechanism; hence it’s more susceptible to injury. Example: where the sciatic nerve branches to the common peroneal nerve.
• Where the system is relatively fixed. Examples: the common peroneal nerve at the head of the fibula, the dura mater at L4, the suprascapular nerve at the scapular notch.

• The nervous system may be exposed to friction forces as it passes in close proximity to unyielding surfaces. Examples: the brachial plexus as it passes over the first rib, the radial nerve in the radial groove of the humerus, the dural sleeve as it passes by the pedicles. Fascia can also be considered an unyielding surface. Examples: the greater occipital nerve as it runs through the fascia at the back of the skull, the lateral femoral cutaneous nerve through the fascia in the thigh.

• Tension points. Examples: C6, T6, L4, tibial nerve at the posterior knee. Various combinations of movement can create unknown tension points.

The most common nerve trauma a therapist faces is the mechanical and physiological consequences of friction, compression, stretch and occasionally disease.

Non-physiological movements, body posture and repetitive muscle contraction may be contributing factors to nerve injury.

**Double crush**

• Since the nervous system is a continuum, old and dormant injury sites are commonly exacerbated with the advent of a new injury.

• The consequences of altered axoplasmic flow may be seen as trophic changes in the target tissues like skin or muscle.

• Part of the explanation of tennis elbow and Achilles tendon rupture may be in interrupted axoplasmic flow.

• In trans-section or severe nerve injuries, axoplasm will actually “drip”.

• The flow of axoplasm will be slowed if the blood supply to the neuron is compromised.

• Fast axonal transport is not altered at pressures of 20 mm Hg, but accumulation occurred at the compression point after 8 hours. 30 mm Hg for 2 hours led to significant slowing. These are the pressures found with carpal tunnel syndrome. (Dahlin and McLean, 1986)

• Disruption of axoplasmic flow is reversible. Pressure of 50mm Hg for 2 hours is reversible within 24 hours. Pressure of 200 mm Hg for 2 hours is reversible
within 3 days. Pressure of 400 mm Hg for 2 hours is reversible within one week (Dahlin and McLean).

- High rates of afferent bombardment from afferent nerve endings in a facilitated segment increases the demand for energy in the affected neurons to the detriment of axoplasmic flow (Korr 1985)
- Inflammation lowers the threshold of firing and therefore increases the rate of firing.
- AIGS- abnormal impulse generating sites.
- Double crush syndrome: minor serial impingements along a peripheral nerve can have an additive effect and cause a distal entrapment neuropathy. The basis of the peripheral neuropathy is thought to be a change in axoplasmic flow. Of 115 patients with carpal tunnel syndrome and ulnar lesions at the elbow, 81 had electrophysiological and clinical evidence of neural lesions at the neck (Upton and McComas 1973).
- Double crush syndromes are well documented in literature for the peripheral nervous system, but keep in mind that it may work in spine patients as well. Example: patient with cervical whiplash who has thoracic and lumbar pain.
- Neural fibrosis and resultant disuse can lead to contracture formation.
- Once intraneural fibrosis has been established, there may be an irreversible component to the disorder and treatment may become more difficult.
- Clinically there are vulnerable sites in the body where lesions affect the elasticity and movements of the nervous system. (Tension Points)
- Sympathetically induced intraneural vasoconstriction may be an important factor in RSD and some chronic pain syndromes (Lundborg).
- Intermittent mechanical agitation for longitudinal sliding of a nerve trunk across an irritant is a major underestimated factor in the production of inflammatory processes (Triano and Lutges).

Clinically, mechanical sensitivity of the nervous system may be evident in patients whose arm pain can be brought on by SLR, or where palpation of the cervical spine can create lumbar pain and vice versa. This relatively common clinical finding will usually be in the presence of a positive Slump test.
Palpation of nerves

Normal nerves should feel round and hard.

An injury to a nerve may feel hard and thickened.

Nerves under tension will have less transverse movement. Direct palpation over a sensitized nerve may produce symptoms along the tract.

Palpation in a tension position may help differentiate between ANTT and local soft tissue problems. Example: palpation of the medial plantar nerve in a SLR position is worse than when the leg is in a neutral position. This will help to differentiate between a medial plantar nerve problem and plantar fascitis.

Nerve tenderness is common at the site of entrapment.

Methods of palpation
- Twang
- Tap
- Direct palpation

Upper extremity

1. **Ulnar nerve: (C7,C8,T1)**
   Located in axilla just medial to the median nerve, which in turn is just medial to the coracobrachialis. Ulnar nerve just lateral to long head of the triceps. Most palpable at groove between Olecranon and medial epicondyle of humerus commonly called the ulnar groove and also known as the “funny bone.” It enters the forearm between the two heads of the flexor carpi ulnaris and continues between this muscle and the flexor digitorum profundus half way down the forearm. Ulnar nerve then passes thru Guyon’s canal formed by the pisiform, hamate and the pisohamate ligament.

2. **Median nerve: (C6, C7, C8, T1)**
   In axilla located just medial to coracobrachialis and lateral to ulnar nerve. Lies just medial to brachial artery and biceps tendon at the crease of the elbow. In the forearm it passes between the two heads of the pronator teres. Becomes more superficial and lies between the tendons of the flexor digitorum superficialis and profundus almost to flexor retinaculum where it becomes more superficial and lies between the tendons of the flexor digitorum superficialis and the flexor carpi radialis.

3. **Radial nerve: (C5, C6, C7, C8)**
   In axilla lateral to median nerve. Crosses latissimus dorsi, deep to axillary artery and after passing inferior border of teres major, it winds around the medial side of the humerus and enters between medial and long head of triceps. It takes a spiral course down the arm in radial groove of humerus becoming most palpable at
insertion of deltoid. Above crease of elbow it runs between the brachialis and brachioradialis anterior to the lateral epicondyle, where it divides into superficial and deep branches. Deep branch of radial nerve (posterior interosseous nerve) winds to dorsum of forearm around radial side of radius between fibers of supinator. Muscular branches supply extensor carpi radialis brevis and supinator.

**Indications for tests of adverse neural tissue tension**

- Post-surgical patients.
- Chronic dysfunctions.
- Post whiplash, post fractures that are stable but where swelling is present (Colles or tib/fib fracture), carpal tunnel syndrome, de Quervain’s tenosynovitis (abductor pollicis longus and extensor pollicis brevis and possibly terminal branches of the musculocutaneous nerve), tennis elbow, TOS and repetitive strain injuries.
- When testing active shoulder flexion or abduction and the patient spontaneously performs the movement with the elbow flexed or the head sidebent towards the involved extremity.
- When the subjective examination suggests the possibility of ANTT (e.g. neck flexion produces back/leg pain while getting into or out of the car or reaching into back seat of the car and this reproduces neck/arm pain).
- Symptoms in the absence of joint signs.
- Chronic derangement or dysfunction.
- Symptoms that do not fit a normal pain pattern (e.g. knee or foot symptoms that are not responding to local treatment).
- Symptoms that exhibit latency.

**Contraindications to ANTT**

- Recent onset or worsening of neurological signs
- Cauda equina lesions (bowel/ bladder symptoms, saddle numbness)
- Tethered cord syndrome

Tethered cord syndrome is where the cord is tethered to the dura mater, which may in turn be tethered to the spinal canal. This is usually congenital and is associated with some form of spinal dysraphism. There is also increasing evidence that an adult version of tethered cord syndrome is not as infrequent as once thought. If the cord is tethered then the forces from spinal movements that would usually be transmitted away from the cord via the denticulate ligaments, meninges and nerve roots are transmitted directly to the cord.

*Precautions*

- Be careful with irritable acute nerve root and/or irritable unstable pathology.
• Standard base tests are only a starting point. You should be prepared to make up your own test based upon the patient’s history, area of symptoms and stage of the pathology.
• It is possible to have one of the ULNT positive while another test is negative.
• Assess patient status as each component of the test is added.
• Irritable condition.
• Latency.
• Elderly patient.
• Reproduction of symptoms, which do not settle in a reasonable period of time.
• Slump test produces symptoms or signs: discontinue test
• Slump test causes dizziness: discontinue test
• Slump test causes head pain: discontinue test
• Worsening disorder
• Neurological signs
• General health problems
• Circulatory disturbances
• Involvement of other structures (i.e. unstable disc with Slump test or irritable facet joint with ULTT)
• Pathologies which affect the nervous system (MS, diabetes, Guillain Barre)
• (+) Lhermitte’s: Patient long sitting and flex head. Complains of sharp pain in spine and/or upper extremities and/or lower extremities. Possible cause from dural/meningeal irritation, meningitis, or spinal cord involvement.

Positive ANTT test

• Reproduction of symptoms. Be careful, as this may not yet implicate the nervous system and further testing may be required.
• Asymmetrical limitation of movement. And/or decreased ROM in tension position of involved side at a particular joint that otherwise has normal ROM (frequently the elbow in the upper extremity).
• Differences in quality of movement (i.e. SLR has increased resistance through range compared to the other side).
• Relevant symptoms along the tract (i.e. burning over fibular head with SLR, which may indicate some limitation along the nerve tract).
• Test response changes with movement of a distant body part away from the site of symptoms. (i.e. passive neck flexion increases the response of posterior thigh pain with SLR) (release of shoulder depression abolishes pain in the anterior wrist crease)

Keep in mind that when using the good side for comparison, this could also be affected by the same disorder.

Guidelines

• Gentle handling skills
• Test uninvolved limb first
• Modify test with limited limb ROM
• Know the details of the patient’s symptoms before testing begins.
• Assess the symptoms in the starting position and again when adding each successive component
• Note the type and amount of resistance through range
• Know the neurological status before testing and monitor this frequently

Normal response and relevance

In normal subjects, tension testing may cause some discomfort and /or tingling. For example, in the ULNT 1, a “normal “ response may be a deep stretch/ache in the cubital fossa extending to the anterior aspect of the forearm, stretch across the anterior shoulder and/or tingling in the first three fingers. When we see a patient in the clinic they are usually there because of symptoms. And one of the symptoms typically is pain. In the vast majority of ANTT cases, the tension test will reproduce the patient’s symptoms. To implicate the nervous system, we move a body part distant from the site of the patient’s symptoms. If this alters the patient’s symptoms then we can call the test positive.

The gray area is when a “normal” response is elicited which is not the patient complaint and the ROM is full and possibly asymmetrical (the patient’s problem could be bilateral). The test is still positive but may not be relevant.

To further judge relevance of a neural tension test, we also must have knowledge about current goals and activity levels and must perform physical examinations with skill. For example, minor limitations and hyperalgesia in a test that loads the radial nerve may well be relevant in a professional tennis player with lateral elbow pain. Similar minor findings in a patient with a widespread and long- standing pain state such as fibromyalgia may be less relevant.

We must also prioritize our findings, which will in turn determine the order of our treatment. Try to get more “bang for our buck.” Think proximal and central. For example, you have a positive ULNT for radial nerve. You go to the neck and discover stiffness at C5,6 on the symptomatic side, but you also find a cervical extension/rotation syndrome. To just perform radial nerve glides would be a sin. Maybe perform A-P mobs grade 1-2 with arm in radial nerve tension position then train intrinsic cervical flexors to stabilize cervical spine and perform isolated upper trapezius strengthening.
**The T6 area in the clinic**

What is it about the T6ish area? The middle of the back is a very popular place to experience pain and tightness. A repeated clinical pattern exists where many people with positive straight leg raises (SLR) or slump tests often complain of pain around the T6 area. Additionally, if someone had a positive SLR or passive neck flexion, suggesting some alteration of meningeal biomechanics, palpation around the T6 area often revealed tenderness and stiffness, usually unbeknown to the owner of the back. People, post whiplash, often have this finding and indeed whiplash sufferers who complain of pain in the mid thorax may have a worse prognosis (Maimaris, Barnes et al. 1988). Many clinicians are well aware that if you gave the mid thorax a bit of a wriggle and shake, the whole body movement can improve and SLR and slump test findings will often improve, sometimes markedly.

**The science**

Intriguingly, some science on neuromeningeal biomechanics emerged at the time of developing neurodynamic theories. The spinal canal could be around 9 centimeters longer in flexion than extension - somehow the contained cord and meninges have to adapt. Check out the T6 area – it doesn’t move that much in relation to the surrounding canal – kind of like if you pull a piece of elastic from both ends, there is a bit in the middle that doesn’t move much in relation to its surrounds.

If there was something special about the area then you would expect that the anatomy would express something about the function. While there has been minimal study (and few would bother today), the major blood vessel for the thoracic cord and meninges comes in just under T6 – so it’s probably not a good place to have a lot of sliding around, and in addition, the canal here is quite narrow and the dura mater is thicker than anywhere else along the neuraxis. Maybe it is designed to be like the middle of a piece of elastic?

**T6 today**

Pause a moment and marvel– the spinal cord in the thorax may only be a little over a centimetre in diameter with the surrounding canal maybe around 1.4 centimetres and also containing meninges and cerebrospinal fluid (CSF) whose freshness is necessary for cord nutrition. This tiny area has a lot of work to do in representing the low back, pelvis and legs. I think it is best to add the cord into modern concepts of representation and neuromatrix which are often all brain based. I think that no matter what, if central sensitization is considered, then the physical health of the nervous system including the cord should be entertained. Is the T6 area a place that is perhaps biomechanically more at risk than other parts of the nervous system especially when you consider what humans do with their bodies these days? Physical problems with physiological consequences could result in significant nociception from the meninges, peripheral neurogenic contributions from sinuvertebral nerves innervating the meninges, and nerve roots plus mechanically
induced contributions to sensitisation. The slump test, especially in long sitting may be useful to check it out.

**Upper limb neurodynamic testing (ULNT)**

**ULNT 1**

*Median nerve bias.*
- Depress shoulder girdle by pushing fist into bed.
- Shoulder abduction to 110 degrees, arm resting on PT's upper thigh.
- While maintaining this position, the forearm is supinated then the wrist and fingers extended, including thumb.
- Laterally rotate the shoulder.
- Extend the elbow.
- Sidebend head to the opposite side, keep face parallel with the ceiling.

When adding components, make sure that earlier component positions are strictly maintained.
ULNT 2

*Median nerve bias*
- Patient lies diagonally across the table. Patient’s shoulder on PT’s upper thigh in 10 degrees of abduction. One hand holds the wrist, the other holds the elbow.
- Using the thigh, the PT depresses the shoulder.
- Extend the elbow.
- Laterally rotate the arm.
- Extend wrist, fingers and thumb.
- Abduct the shoulder.
- Sidebend head away.
ULNT 3

Radial nerve bias
- Patient lies diagonally across table. Shoulder in 10 degrees of abduction with elbow straight. Patient’s shoulder on PT’s upper thigh.
- Depress shoulder girdle with upper thigh.
- Medially rotate the arm then pronate the forearm.
- Flex wrist, thumb and finger, ulnar deviate wrist. This sensitizes the superficial sensory branch of the nerve.
ULNT 4

Ulnar nerve bias

- Depress shoulder by pushing fist into table. Patient elbow in PT iliac fossa.
- Wrist and finger extension particularly fourth and fifth digits.
- Forearm pronation.
- Fully flex elbow.
- Add lateral rotation.
- Abduct shoulder. As if holding platter, placing hand over ear.
- Sidebend head to opposite side.
Palpation of nerves lower extremities

Sciatic nerve (L4,5, S1,2,3)
The largest nerve in the body. It passes out of pelvis through greater sciatic foramen and extends from the inferior border of the piriformis, between the ischial tuberosity and the greater trochanter. Becomes more palpable just proximal to popliteal fossa.

Tibial nerve
Lateral to the popliteal artery at the crease of the knee, between the heads of the Gastrocnemius. Travels to medial aspect of the Achilles tendon, posterior to medial malleolus. Divides into the medial and lateral plantar nerves.

Common Peroneal nerve
Medial to the tendon of the biceps femoris, wraps laterally around fibular head along lateral lower leg and passes deep and just medial to peroneus longus where it divides into the superficial and deep peroneal nerves. (Intermediate dorsal cutaneous nerve) branch of the superficial peroneal nerve. Lateral to extensor digitorum longus, anterior and medial to lateral malleolus. Passes along the lateral part of the dorsum of the foot.

Femoral nerve (L2, 3, 4)
Emerges through fibers of psoas major at the distal part of the lateral border, and passes down between the iliacus. Passes under the inguinal ligament and is lateral to the femoral artery.

Saphenous nerve
The largest and longest branch of the femoral nerve. Lies between the tendon of the sartorius and the gracilis then distal to VMO.
**Straight leg raise**

*History*
Reports around 2800 BC suggest that extension of the legs should be used to examine sprained vertebrae. Most sources say that Charles Lasegue from the University of Paris was the initiator of the test in 1864. His pupil, Forst, brought it into prominence. Lasegue stated that pain in SLR came from compression of the sciatic nerve by the hamstrings. Lazarevic in 1880 was the first to recognize pain produced by SLR was due to sciatic nerve irritation. He also demonstrated that dorsiflexion intensified sciatica.

*Straight leg raise test*
Base test SLR: Flex the hip, keeping the knee extended. Add sensitizing movements and ASSESS as each component is added! Sensitizing movements are based upon area of symptoms. For the LE they are as follows:

- Neck flexion
- Dorsiflexion (tibial nerve) sometimes referred to as Braggard’s.
- Adduction
- Medial rotation (peroneal nerve tract)
- Plantarflexion/inversion (peroneal nerve tract)
- Dorsiflexion/inversion (sural nerve)
- Bilateral SLR
- Lasegue sign (hip vs. Sciatic nerve). Flex knee after symptoms.
- Alter cervical and/or lumbar spine positions

Start with spine, body and trunk in neutral position.
**Prone knee bend**

In 1919 Wasserman used to diagnose anterior thigh shin pain in soldiers. Base test PKB: patient prone. Flex knee. Add sensitizing movements and ASSESS as each component is added. Use variations as indicated.

- Hip extension
- Hip adduction (lateral femoral cutaneous nerve), or abduction (saphenous)
- Hip internal or external rotation
- Hip abduction, external rotation and extension with knee extension (saphenous nerve)

- PKB in side lying slump

*Normal response:*

Enough knee flexion to allow the heel to touch the buttocks
Pulling/stretch in quadriceps area
The Slump test

*History*
- Petren in 1909 was the first to employ knee extension in sitting as a tension test.
- Cyriax in 1942 used extension of the knee in sitting with cervical flexion to diagnose “sciatic neuritis”.
- Maitland popularized the test in 1979.

*Purpose:* To assess the mobility of the pain sensitive structures in the vertebral canal and intervertebral foramen (dura mater and root sleeve) and peripheral nerves as a possible source for the patient complaint. The test evaluates the limitation of motion and reproduction of the patient’s symptoms (Maitland).

*Method*
- Know patient’s symptoms before starting.
- Patient sits back on table with popliteal fossa touching plinth.
- Patient’s hands behind back.
- PT adducts patient’s legs.
- PT sits or stands next to patient’s side.
- Ask patient to “slump” or slouch. (May need tactile cueing in belly)
- Put arm around patient’s shoulders and apply light overpressure (mainly do not allow patient to come out of this position or desensitize the nervous system)
- Remember to assess patient’s symptoms as each component is added.
- Ask patient to flex chin to chest.
- Apply light overpressure.
- Ask patient to extend knee.
- Ask patient to dorsiflex foot. Then PT holds foot in this position.
- Finally, ask patient to extend head (look towards ceiling) assess for alteration on symptoms.
Neurodynamic quick tests

Functional tests which are easily assessed to implicate comparable structures and assist in focusing the objective examination.

**Purpose**

- To assist in proving or disproving a working hypothesis as it relates to structural differentiation
- To sensitize (increase tension on the nervous system), or desensitize (decrease tension on the nervous system)
- Use of functional aggravating factor; think of all causes, not just ANTT.

**Upper extremity examples**

- Shoulder abduction causes shoulder pain: perform shoulder abduction with cervical spine sidebend towards (desensitize ANTT) vs. Sidebending away from the symptomatic side to sensitize. Can perform with wrist flexion or extension.
- Reaching behind back causes anterior shoulder pain. Alter cervical or wrist component.
- Elbow extension causes shoulder pain: Increase ANTT by sidebending away or depress the shoulder, flex, extend wrist.
- Cervical flexion and/or sidebend, recreate shoulder and/or UE symptoms.

**Lower extremity examples**

- Lumbar flexion increases calf pain: Add neck extension to desensitize ANTT.
- Lumbar sidebending away causes leg pain: Add cervical sidebending towards to desensitize. Or if lumbar sidebending causes no symptoms then add cervical sidebending away to sensitize.
- Sitting with right leg crossed over the left increases right trochanteric pain: Add cervical flexion or trunk flexion. Reverse sequence of test.
- Squatting: With trunk flexion vs. extension. Addition of cervical flexion or sidebending away.

**Treatment considerations**

- Always palpate over all areas of symptoms
- No symptoms when treating acute and sub acute, but move into resistance. Once situation is stable, can go into symptoms.
- Muscles protect nerves
- Can use big levers, oscillations
- Can do simultaneous treatment while in stretch position (cross friction massage, joint mobilization, ionto, IF, heat etc.)
- Take off more distal component from site of symptoms to desensitize
• When doing Slump test, take break, walk around, then do opposite leg (consider irritability)
• Can do Slump test in neutral lumbar spine
• Add neural tension with normal muscle stretching and strengthening (position of feet, head on ball or total gym, seated knee extension, hamstring stretch)
• Teach patient how to do, how not to do, what to feel, what not to feel, **not to over do.**
• Need to constantly assess and re-assess during each stage of testing and treatment

**Effects of neural tissue stretching**

• Improve vascularization of the nervous system and surrounding surfaces.
• Improve axoplasmic flow.
• Improve the mechanical properties of the nervous system.
Mobilization of the nervous system with and without the use of soft tissue mobilization and/or joint mobilization with tension signs

Upper extremity techniques

• Anterior cervical spine- A-P mobs, side glides in and out of tension positions.

• STM on foam roll- use of belt and ULNT signs
• Circumferential contract/relax UE and LE
• STM to upper extremity and various trigger points with ULNT in tension position with and without active movement.
• Suboccipital release
• First rib depression
• Use contract/relax with ULNT
• Mobilize peripheral joints in tension position (i.e. radial head)
• Functional movement patterns (FMP) - active cervical rotation while therapist performs sustained pressure (SP) on scalenes/trapezius

![Image of therapy technique]

• FMP - cervical flexion

![Image of cervical flexion]

• Mobilize costovertebral joint - manually or with tennis ball
• Mobilize thoracic spine - manually, double tennis balls or roll
• Passive neck flexion with chin tuck and assist.
• Passive neck flexion in supine or long sit while mobilize sternum.

Straight leg raise variations

• Perform in adduction or abduction
• Perform with neck flexion or thoracic flexion

• Dorsiflexion (tibial nerve)
• Internal rotation (common peroneal nerve)
• Plantar flexion and inversion (superficial and deep peroneal nerve)
• Dorsiflexion and inversion (sural nerve)
• Bilateral SLR
• Perform knee extension while hip in 90 degrees, can adduct or abduct hip
- Active-assisted SLR with belt, supine or side lying
- Side lying SLR, alter trunk/neck position.
- Mobilize L-spine or sacrum with roll or tennis balls
- Hamstring stretch in doorway, STM on hamstrings in stretched position
- FMP - squat with STM to calf

**Passive knee bend variations**

- Hip extension with 90 degrees knee flexion (vary knee flexion depending on signs/symptoms)
- PKB in hip adduction and hip extension (lateral femoral cutaneous nerve)
- Hip abduction, lateral rotation, hip extension with knee flexion (saphenous nerve)
- PKB in Slump

**Slump test variations**

- Knee extension (KE) inn thoracic flexion (TF) and neck flexion (NF) without dorsiflexion (DF)
- DF in TF/NF/KE
- Hip flexion in TF and lumbar flexion towards NF/KE/DF
- Alternating neck and trunk position to move into or out of ANTT
- All above variations in side lying
- NF or DF in long sitting: unilateral or bilateral KE and DF. Can change trunk or neck positions. Add ER or abduction to desensitize
- Thoracic flexion in long sitting, feet against wall for DF or active DF
- Combine with PKB on one side, side lying

**Other treatment options, home exercises and self-mobilizations**

- Hand-heel rock
- Chin tuck.
- Chin tuck with mobilization of sternum/manubrium.
- Side lying over roll.
- Foam roll used in door jam to depress shoulder
- Pivot prone
- Thoracic and rib self mobilization over roll, tennis balls
- Bilateral SLR on wall
• Back stretch where legs go over head

• Standing hamstring stretch, cross legs, flex head
• Contract/Relax
• Unilateral P-A costovertebral in long sit.

• ULNT 3 radial nerve bias - behind back
• ULNT 2 median nerve bias

• ULNT 4 ulnar nerve bias
References