

## **EMG**

EMG/NCV – Electromyogram/Nerve conduction Velocity studies. In some cases, EMG/NCV's can help diagnose and can even help in cases of litigation. EMG/NCV can help to show if pain is really neuropathic. Pain is a subjective response. Orthopedic tests may be subjective (due to pt. verbalization of pain). EMG/NCV is an objective test, giving concrete evidence that a neurological problem/deficit is present.

The test says whether there is a neuropathy and what the particular level of involvement. For example, in a case of carpal tunnel, surgery should not be considered without concrete evidence of a neuropathy of the median nerve.

EMG/NCV's are typically sent out. Typically, a neurologist or a physiatrist performs them. The doctor gives their impression of the results (ex. – C7 radiculopathy instead of ulnar nerve entrapment).

The most common symptom found is weakness with an EMG. Pain down the leg is sensory, but an EMG looks at motor unit disease. Problems with pain, caused by pinched nerves, then damage to nerves can show motor as well sensory changes.

### *Motor Unit*

The motor unit involves the alpha motor neuron. There is a myoneural junction and a muscle fiber. Motor units can be large or small. There is one neuron innervating many fibers. The unit is small or large dependent upon # of fibers it innervates. The smallest motor units are in the intrinsic muscles of the eyes. Those contain as few as 15 muscle fibers. The largest motor units can innervate over 200 fibers, found in the large muscles of the leg. The difference between a small unit and large unit is fine movement (hand – small unit) and gross movement (leg – large unit). The fewer the fibers, the more detailed the movement.

If a person cannot perform fine movements, (clumsy), something may be going on in the motor unit.

Alpha Motor Neuron to the Myoneural Junction = Nerve Involvement...a lesion in this area is a neuropathy

*If the problem is in the muscle itself = Myopathy*

Ex.—Wasting of the shoulder --- What could be the cause? – Neuropathy or myopathy? – Girdle dystrophy is a myopathy limited to specific areas (shoulder and hip)... This condition is a genetic wasting of the muscles. There is no known treatment... In this example the chief complaint was weakness.

### *Needle EMG*

Needles are inserted into the muscle. When the needle is in the muscle, activity at first is insertional activity (due to insertion of the needle). This is believed to be from the release of calcium. In a couple of seconds, the readings will calm down to resting rates.

**A. Resting Potentials/Fibrillation Potentials:** Spikes in the readings after the insertion of an EMG can be a fibrillation. When a muscle generates this activity it **is not** a good thing. Tiny fibers contract independently. Some patients may feel them. The resting potentials indicate an irritation of the muscle at rest. This is created by a process of decentralization and refers to the central nervous system. The fibers in the proximity of the needle are being separated from the CNS. The fibers are separated from the CNS. This is evidence of a possible neuropathy. These potentials at rest may indicate a neuropathic condition, since normally there should not be spikes of increased amplitude when a needle is inserted.

Decentralization = Indicates a possible neuropathy. In this case, **it is loss of the alpha motor neuron.** Polio is a good example of this condition of decentralization. Polio destroys the alpha motor neuron. Polio attacks the alpha motor neuron, creating a LMN lesion.

Problem at the nerve root indicates a neuropathy. A neuropathy can cause fibrillation potential. The problem may also be caused by plexopathy, peripheral neuropathy, or myasthenia gravis at NM junction.

MUAP (motor unit action potential) are of interest to the examiner. MUAP's can be counted. A normal muscle under max contraction gives off a recording with evidence of action potentials. Contracting a muscle has asynchronous firing of motor units as not all fibers contract at once. Every motor unit does not contract or relax at any one given instance. When 1 unit fatigues, another takes over = asynchrony of firing. *Synchrony of firing means either loss of motor units, or so much stress through muscle that you are recruiting every fiber.*

When a motor unit action potential fires, all the fibers contract with 1 motor unit action potential.

The height of the peaks measures the amplitude. The amplitude is the amount of energy given off by the MUAP.

**B. Myopathy:** Ex.—Overhead Example: In the EMG on overhead with a myopathy, the height and number of potentials are different. There is increased # of potentials with lower overall height due to a myopathy. If muscle fibers are lost, you must recruit more fibers. Each fiber has less amplitude to accomplish the same task. More than 1 motor unit must be used, since they've gotten smaller. Since they've gotten smaller, the amplitude decreases. We end up with the myopathy presentation on the EMG. Progressive weakness forces more recruitment of units with less amplitude.

**C. LMN Disease:** The number of MUAP's is less and the amplitude is greater. The motor units decrease in #, but bigger in amplitude. Damage to a nerve leads to decentralized fibers. When the needle is put in, they will show fibrillation. They are sensitive to acetylcholine. There are increased receptor sites on the receptor cell to acetylcholine (as a response to lack of nerve stimulation) looking to contract the muscle and presenting with the pattern of LMN disease in the EMG. The LMN will attempt to recruit the fibers next to it. It will make the motor unit bigger. This will make the amplitude greater, but the # of units goes down (because of loss of innervation).

A progressive loss of motor units, as in a progressive neuropathy will cause loss of fine motor skills as these are recruited to merge with the larger fibers.

#### *Segmental Findings*

If the findings are indeed segmental, the myotomes will be affected. If the problem is at the nerve root, then exam of muscles should produce weakness. If the problem is indeed in the IVF, the posterior ramus (innervates back muscles) should be involved. A plexopathy, peripheral neuropathy will not show involvement of back muscles innervated, since back muscles are innervated by the posterior ramus.

#### *Myasthenia Gravis*

The weakness comes on progressively. This problem is due to restoration of acetylcholine (essentially they run out of it). Dr.'s can do repetitive nerve stimulation to test for this condition. Repetitive stimulation will cause the loss of acetylcholine to mimic the symptoms. Then the patient is given a drug to restore the acetylcholine and everything returns to normal. So the testing is to use up the acetylcholine producing weakness, then restoring the acetylcholine to restore the strength. This test shows the importance of acetylcholine and indicates myasthenia gravis.

#### NMS – 11/15/07

#### **EMG—Myopathy**

Example: The # of MUAP's increases, but the amplitude decreases. There has to be greater recruitment attempting to make the muscle twitch.

#### *Progressive Neuropathies*

During progressive neuropathies, spiked potentials during contraction means that you are dealing with a neuropathy. With a neuropathy, you will ultimately see atrophy and loss of fibers. The most common symptoms of motor unit myopathy is weakness. Progressive weakness will lead to atrophy. The amplitude of the potentials should increase, during a neuropathy. The fibers are picked up by a neighboring nerve. Nerve growth factor is produced readily causing a budding and attachment within weeks to another nerve

#### *Role of an EMG/NCV*

EMG and NCV are done during active state of the lesion. If a patient develops atrophy a huge red flag should go. The EMG can tell you whether there is evidence of a nerve lesion or not. A NCV will localize the lesion. It tells you where the problem is.

Before you do the EMG, always perform the orthopedic and neurological assessment. If an EMG is different than the patient presentation, refer back to your orthopedic and neurological assessment. You cannot ignore weakness or atrophy.

In a plexopathy, the patient may present with a neuropathy. The neuropathy is not segmental. A NCV would help localize where the lesion is.

Example 1: A patient with sclerotomy pain down their leg during an EMG would not indicate a neuropathy in the results of the test as the EMG would be normal.

Example 2: If you had a disc bulge and the bulge touched a ligament in your back, you could have pain referral. The pain referral could be down the leg, but the EMG would not show a myopathy or neuropathy because weakness is not present.

Example 3: If a patient demonstrates a neuropathy and the findings are segmental, the doctor can find weakness in the muscles relating to the segment. The muscles are all innervated by the same nerve root (myotome). This tells you the lesion is likely to be at the nerve root. The muscles are innervated by anterior primary ramus. One can also do EMG's along the posterior primary ramus, if the back muscles have a bad finding on the EMG, the lesion must then be the nerve root and the problem at the IVF! To summarize, evidence of a neuropathy can then be found in muscles innervated by the myotome via the anterior ramus and also in the muscles of the spine (back muscles) via the posterior ramus.

Example 4: A-----B-----C-----D-----E... (A=CNS...B=Nerve Root...C=Plexus...D=Peripheral Nerve...E=Alpha Motor and NM Junction)...If C-D is lesioned, then the problem is not at the nerve root, but the peripheral nerve or the plexus.

Example 5: A----- B (Neuron with Inflammation – Neuropraxia) --C----- D (Fiber)  
Neuropaxia/Neuritis is present. The nerve still maintains continuity. The action potential hitting the site of inflammation makes the conduction slow down until it passes the site of neuropraxia and then it speeds up to the fiber. Measuring conduction along the nerve, A-B = Normal, B-C = not normal, B-D = not normal....This indicates a neuropathy. An example would be carpal tunnel in the wrist.

Example 6:—Disc Herniation... You cannot do a NCV in the spinal canal. You can do a NCV outside the canal. If you have evidence of a normal EMG in the periphery with segmental problems, this gives evidence of a disc bulge/herniation.

### NCV

In an NCV you check for latency or slow conduction. A measuring electrode is placed on a distal muscle. A nerve is stimulated at a proximal point. The time frame is calculated from proximal to distal. A distal latency number is determined. You can look up the normal distant latency and compare this number to your results. If the # is increased, there is evidence of decreased nerve conduction velocity.

### SSEP

You can also do sensory studies, SSEP (somatosensory evoked potentials). SSEP's look at sensory nerves. They help find neuropathy and lesions. Both SSEP and EMG/NCV help to find the site of the lesion.

### Double Crush Syndrome

Ex.—Also Called Ganglio-Neuropathy....This condition has two injury sites. 2 areas of pressure are needed to manifest the problem. The two lesions can co-exist with double crush. Adjusting the C-spine in the treatment of carpal tunnel syndrome or treatment distally at the wrist can help to get rid of the problems. Addressing one lesion can help to get rid of the second site of lesion. This validates the treatment of some carpal tunnel problems with spinal adjusting. Conservative management of cervical problems is advised before surgery. An EMG can show a peripheral problem with double crush syndrome. . . .

### NMS – 11/19/07

\*\*\* 3<sup>rd</sup> exam will tentatively be the Monday of the week leading into Final Exams...20 old questions from the 1<sup>st</sup> 3 exams \*\*\*  
\*\*\* Read book chapters \*\*\*  
\*\*\* Illustration on overhead \*\*\*

### C-Spine

C-spine disc herniations can occur from insidious cause. Patients can have a disc herniation and not be able to remember what the cause is. They may tell you that they “woke up with it.” There may be no known onset. Luschka joint provide lateral support and protect the nerve root. Luschka joints can flare out due to disc lesion. Disc injuries can cause hypertrophy of Luschka joints and pinch the nerve. The L-spine does not have this protection since *it does not have Luschka joints.* The PLL spans the entire distance and is 3x thicker in the C-spine than in the L-spine. The nucleus lies more anterior in the C-spine than

in lumbar spine. The possibility of nucleus migrating posteriorly is less than in the L-spine. Annular fibers of C-spine can be thick and strong vs. those in lumbar which are weak.

#### *Article on Overhead*

Bogduk (1999)... “Little or no annulus in the L-spine region. There is no layering of concentric rings at concentric angles. Thin layer of posterior fibers supporting discs between uncinat processes, creating inherent weakness.”

#### **L-Spine Discs**

Disc lesions in L-spine are far more common. In lumbar spine disc injuries, *the patient can relate a trauma*. They can recall a particular event that induced the herniation (trauma related). *PLL is 1/3 width and tapers down in the L-spine, making it vulnerable to annular fibers. Annular fibers may be weaker in L-spine than in the C-spine. L and R posterior-lateral discs are susceptible to annular fibers and bulging. Lumbar postero-lateral is the most common site of disc bulge. The PLL ligament does not cover the entire posterior disc in the L-spine.*

L5 disc can entrap S1 nerve root. L4 disc can entrap L5 nerve root (due to cauda equine).

#### **Medial vs. Lateral Disc**

*Posterior-Lateral Herniation*: Disc bulges/herniates outward and to the side. This can hit either the medial or lateral side of the nerve root. There is 1 huge category (posterior-lateral) that is divided into 2 subcategories (medial & lateral). The medial and lateral subcategories refer to what side of the nerve is pinched.

There are people with nerve entrapment that are completely symptom free. It then becomes important to check for evidence of dysfunction.

#### **Disc as a Pain Producing Structure**

The disc like a muscle can be a pain producing mechanism. The sinuvertebral nerve innervates the disc. It has C-fibers and sympathetics. There can be pain without nerve root compression. The annulus is well innervated by sensory and autonomic fibers. There can be C-nociception by mechanical means and irritation of the sinuvertebral nerve. The sinuvertebral nerve pierces the outer 1/3 of the annular fibers. The disc itself thus can produce a pain pattern. There can be referred pain that is not dermatomal. Nerve root compression is not always necessary to produce referred pain. NCV's and EMG's can be used to help determine the source of the pain and if there's evidence of a nerve pathology.

#### **Dura**

Dura is innervated. The sinuvertebral nerve that comes off the nerve root innervates its own nerve sleeve and 1 segment above and below. Disc pieces from a bulge or herniation can hit the dura. The dura surrounding the nerve root (dural sleeve) is hit first with a disc protrusion on the nerve root. Dural sleeve protects the nerve roots and allows them to slide in and out. A SLR test will actually move the nerve root. The dura protects the nerve from this motion.

Example: Pressure on the disc causing a bulge that attacks the dura...The pain would be felt in multiple areas of the lumbar spine. This would be typical lumbar pain called “Lumbago” or “Lumbalgia.”

#### *Orthopedic Tests & Tests for the Dura*

Orthopedic tests can help to localize the site of lesion. 4 tests are universally accepted to test for disc lesions. You learn more from a – test than a + test. Orthopedic tests help give a focal diagnosis. There are bad at giving an etiological diagnosis. We expect an etiological diagnosis (what's wrong). Orthopedic tests were never created to tell us what's wrong (etiology). Orthopedic tests are + when they reproduce the chief complaint.

Ex.--+ cervical compression test indicates the problem could be more than a disc. The problem could be tumor, hypertrophied ligament, osteophytes, etc. The compression maneuver reproduces pain in the cervical area and/or arm that indicates more testing should be done to determine the diagnosis. We should use the orthopedic tests to help find if our preliminary diagnosis are correct, rather than use the test to help make the diagnosis.

Cyriax: If the 4 tests do not reproduce the problem, it can't be a disc (Vasalva, SLR, WLR, and Soto-Hall). The disc first encounters the dura. These tests are tests for dural integrity. If these 4 tests are – then it can't be disc. If you have a disc, one or more of these tests will be + and reproduce pain/chief complaint.

#### *Vasalva Maneuver (Dural Test)*

Reproducible pain with Vasalva indicates SOL in the spinal canal (tumor, bone fragment, disc, etc.)

## NMS – 11/20/07

### **Dural Sleeve/Sheath**

The dural sleeve is innervated by its sinuvertebral nerve. The sinuvertebral nerve contains C-fibers. C-fibers are pain fibers. Dural tissue can refer pain. This tissue is segmentally innervated. A lesion pressing on this area irritates the dural sheath. This is somatic tissue (sleeve) innervated by sinuvertebral nerve (C-fibers) that sets up a sclerotomal referral. On a CT scan, it may present with a disc bulge, but the presentation of signs and symptoms would show no evidence of lesion/deficits. People with a dural sleeve injury may have a + Valsalva. This means that there is an SOL within the canal, but we cannot further diagnose the condition. A + Valsalva **does not** necessarily mean + neurological deficits. It does mean that something is in the canal. When bearing down, blood pressure changes dramatically, heart rate drops, and parasympathetic tone increases (vagotonic). Sometimes people feel like they are going to faint with Valsalva due to increased vagal stimulation affecting the heart and the lungs. Aggressive Valsalva maneuver may then induce fainting. Patients in the cardiac area of a hospital may not be allowed to use the washrooms, due to the increased vagal tone and the fact that previous heart damage.

Valsalva does cause dural expansion. The sinuvertebral nerve innervates the dural sleeve by 1 maybe 2 sinuvertebral nerves.

### **Sciatic Neuropathy**

A neurological dysfunction is not determined by an orthopedic test. It is determined by sensory, motor, and reflex exam.

The dural sheath has to be impinged first. This pain can be sclerotogenous. The Valsalva expands the dura into the bulge. Many times you get a reproduction of sclerotogenous pain with a Valsalva.

*Occasionally this bulge will move and push on the nerve root. The patient then experiences tingling into the dermatome.*

### **Central Disc Bulge**

Disc bulge goes directly posterior. The PLL is innervated by many sinuvertebral nerve. Any spot on the PLL is multisegmental. A lesion of the PLL is completely unpredictable. The pain can be extrasegmental. It is a dull, achy, not following a sclerotome pattern. One day it can be more prominent one side than the other. The more confused they are, the more convinced that it is some form of nonsegmental somatic pain.

Multisegmental-Extrasegmental-Nonsegmental = All Synonymous

### **Lumbago**

Dull, diffuse achy pain across the low back. It is tough to find evidence of an SOL with lumbago.

If the problem is a disk, surely 1 of the dural tests will be + (Valsalva, SLR, WLR, Soto-Hall). In a patient with lumbago you cannot raise the side of involvement. Lumbago is generalized low back pain across both sides of back. Lumbago does not present with one sided complaints. You must then describe the test as leg raise and not SLR or WLR. SLR always means the involved leg. In lumbago, they don't have an involved leg, so you cannot do a SLR. You have to describe the test as right and left leg raise. If the patient does not have pain with the leg raises, then it may be safe to say that they don't have a disc bulge.

## NMS – 11/26/07

### **Disc Lesions**

Cervical disc lesions are more insidious (less history). Lumbar discs are more common and the patient can relate to an incident. Discs most often bulge postero-lateral. 25-30% lumbar spine disc lesions may go directly posterior (central lesions). With posterolateral discs, there is greater chance for radicular problems. You then need to distinguish whether the bulge is lateral to the nerve root or medial to the nerve root. If the bulge is lateral, they will lean away from the pain. If the bulge is media, they lean into the pain.

Cox-Flexion Distraction = technique is based on medial vs. lateral bulge. The technique changes based on the decision of whether the bulge is lateral or medial to the nerve. Clinical symptoms are important in the treatment.

#### *Medial Disc Lesion*

Medial disc bulge (medial to the nerve) most common are *leaning into the pain*...Ex.—R sided sciatica with medial disc bulge...patient leans into the pain...mobility of nerve is such that it is pulled away from the bulge...*This patient would have a + SLR (nerve tension is present). This patient will also have a + Well Leg Raise (pulls the nerve into the bulge – irritates the nerve by the bulge). Medial disc bulges have + SLR and + WLR.*

### Lateral Disc Lesion

Lateral disc bulge on R side...Patient leans to the L side (lean away from the lesion)...This is a lateral bulge (lateral to nerve root) & patient leans away from the pain (Lateral = Lean Away from pain). The disc is immobile, but the nerve root is mobile. When you lean away, the nerve root moves away from the bulge. The patient will have a + SLR, but will not have a + WLR. Raising the well side moves the nerve farther away from the bulge. This is huge difference between the two.

### **Dural Sheath**

The dural sheath is innervated by same nerve that innervates the disc and the PLL. This makes it very difficult to truly find the source of the pain. If the pain is actually from the dural sheath, **pain is usually one sided and will be confined to a unilateral sclerotome.** There will be **no neurological deficits** (no change in reflex and no paresthesia). **This will be a nasty dull ache on one side.**

### **Disc & PLL Pain**

Pain will be more diffuse through the lower back. It will be extrasegmental pain (Dr. Cyriax). The patient will not have a dominant side. The patient will demonstrate lumbago.

### **Lumbago**

Lumbago is non-segmental and diffuse pain throughout the back. If the pain is reproducible by Vasalva (+ Vasalva) reproducing lumbago pattern, it is likely that a disc may be in your differential diagnosis/diagnosis. A Vasalva is one of the best tests for SOL.

Orthopedic tests were never designed to give etiology, but they were designed to give the site only. Facets do not give + Vasalva. Facets are innervated by A and C fibers. Facet pain reproducible is by A fiber pain on orthopedic tests, but the other structures will not show the pain.

### **List of Orthopedic Tests**

**Dural Tests: Vasalva, Neck Flexion, SLR, WLR**

**Bechterew**

**Braggard**

**Spinous Percussion**

**Milgram**

**Kemp** – may not be a good orthopedic exam...may be used to check for improvement

**Ely**

**FABER**

**Iliac Compression**

### **Case Examples**

**Example 1:** CC: Back pain/spasm. L5/S1 dermatomal leg pain and paresthesia. Lumbar DJD; sprain injury two weeks earlier. + Milgram, + SLR, +Kemp, + Bechterew, + Braggard, -WLR, -Vasalva, -Soto-hall

Overview: Milgram's Test is a general test for lumbar pain. If there's any pain in the low back, it will be +. Milgram's can even be + in a DVT. + SLR, +Kemp, +Bechterew's (Bechterew's is a sitting sciatic stretch test ...Bechterew's is a challenging test for sciatica, but not a true test for disc lesion). L5/S1 radiculopathy is a focal diagnosis that tells you that the problem lies at the level of the nerve (it could be bulge, fracture, etc.). Bechterew's tells you that the lesion is sciatic or radicular, but you don't know why. Braggard test is + (dorsiflexion of foot indicates sciatic). - WLR & - Vasalva (throws out disc theory - universally accepted test that one will be + with a disc)... - Soto Hall (neck flexion - this will rule out dura)...Now with all this info we have 1+ dural test (SLR), but the rest of the dural tests are -. We need to find lesions with dermatome pain, involving dura that would not involve a disc.

### Choices:

- Dural sheath irritation: *Rationale:* Chief complaint is dermatome with paresthesia. Dural sheath will be sclerotogenous pain
- Central Disc: If disc is posterior, then this would present with sclerotogenous and not dermatome pain patterns. Also, Vasalva was -. This example also shows neurological deficits
- Osteophytic impingement: It could be possible due to the age of patient having DJD. Tests would make sense. Osteophytes can cause sciatica or radiculopathy. Osteophytes usually give a - Vasalva (usually there are located in lateral parts of IVF and not directly in it). Valsalva's show up when problem is in canal. Osteophytes can be an SOL, but many osteophytes are confined to lateral margins of IVF presenting with - Vasalva. This is the BEST CHOICE AVAILABLE FOR A DIAGNOSIS!

d). Lateral Disc: Probably not a lateral disc, due to 1 finding (- Vasalva). If it was a lateral disc, Vasalva should be +.

The spur was there pretreatment and will be there post treatment. The patient was flared up and the inflammatory reaction occurred. You can help the person, but they do have DJD and the condition can be exacerbated all over again. Spurs can become a problem, with an acute injury, increasing the inflammation.

**Example 2: CC:** Burning anterior thigh pain. + Ely, + Kemp, + Percussion, +Vasalva, + Neck Flexion, - SLR

Overview: Ely: Heel to Butt stretches femoral nerve (counterpart of sciatic nerve)

Kemps: Bad Test to perform

Percussion Test: Specific test (very specific) – Percussing the spine puts the pain right at the level

Vasalva: +

Neck Flexion: + (Possible dural)

SLR: - (this is sciatic test and not femoral)

Choices:

a). L2 disc herniation: May be a choice due to + Vasalva...the pain does not fit a complete dermatome, however chief complaint does not match a true dermatome.. Answer is A.

b). Sclerotogenous referral: No, due to burning pain (rules out sclerotogenous referral)

c). L3 IVF encroachment: Vasalva is generally not + for IVF lesions, but is + for canal lesions. Pattern also does not fit a dermatomal pattern.

d). Lumbar facet syndrome: Not a good choice, due to burning sensation, plus Vasalva + rules this out.

e). Neuralgia Paresthetica: Peripheral entrapment of lateral femoral cutaneous nerve. This is more common in men than women. Presentation is with a Bull's eye sign. The sign is an area of numbness in the middle of burning pain. The numb area is insensitive to pain. Posterior ilium patients are subject to this. Men wearing heavy tool belts are subject to this condition. This is a peripheral neuropathy, cause by entrapment within fascia. Choice E is not a good choice due to + Vasalva (indicating location of problem in the spinal canal). Also, there is a + percussion test causing pain. Can't be choice E because of Vasalva and percussion test.

SLR is mainly for neurological lesion of L4-L5-S1. Cyriax gave 4 dural tests, and the reason is why is due to a numerous amounts of possibilities.

### NMS – 11/27/07

Article read by Dr. Christy...

Patients cannot relate an event for a cervical disc injury. They may wake up in the morning and just feel it. The most common level is L4-L5 for a disc. This presents with stabbing pain, superficial and localized pain often associated with numbness and tingling. In advanced cases, there may be deficits. Uncommon disc herniations (central disc) may provoke lumbago. A central disc herniation, may compress cauda equinae and cause incontinence with bowel or bladder dysfunction requiring referral to medical specialist. Often, the central disc needs to be differentiated from muscular strain. **There is 1 good orthopedic test to help differentiate strain from central disc (Vasalva).**

### **Soft Tissue Lesions – Sprain vs. Strain**

*Strain:* Injury to connective tissue, generally contractile tissue. If a person injures muscle = strain vs. if a person injures ligaments = sprain. Loss of integrity of muscle and 2 different kinds of strain: 1). Transverse 2). Longitudinal

Transverse Strain: Damage to the muscle fiber itself. The fiber is torn across the middle. Many fibers are injured in the muscle. The cause is direct trauma. For example, getting hit with a baseball in the chest can cause a transverse strain. Damage occurs to the muscle tissues and can create pain and limitation of muscles. Another cause may be contracting a tissue against excessive resistance. Active shortening of the muscle can damage the muscle. Lifting something too heavy can then injure the muscle. There will be immediate pain and disability with spasm. One of the clinical signs is pain on contraction. When the muscle is at rest, there is no pain or minimal pain. **Pain on contraction is a big indication that the injury is a transverse strain.**

### **ROM Chart**

	Muscle	Joint
AROM	Pain	Pain
PROM	-----	Pain
RROM (Isometric Test)	Pain	-----

This chart is good for a transverse injury only. Active contraction will produce pain with this maneuver. The injury site will be irritated and inflamed during active contraction. RICE is the appropriate therapy.

*Trigger Points:* Trigger points usually manifest in people have chronic reports of pain. The points may not be painful on motion.

Longitudinal Injury: *Far more common. In a longitudinal injury, the injury is to the connective tissue/fascia rather than the muscle fibers themselves. This happens by eccentric contraction. This occurs by active lengthening during contraction. This is also called a negative repetition. For example, throwing a ball, the arm must decelerate. The muscles that decelerate the arm in a state of contraction are forced to lengthen. Eccentric contraction of hypotonic muscle occurs. If the muscle is not well conditioned and subjected to this eccentric force is it more susceptible to injury. If a muscle is hypotonic, there is excessive force exerted on the non-contractile elements of the tissue. This type of injury will damage the spindle.*

Travell and Simmons: Longitudinal injury is an orthopedic term. *Longitudinal injury is called a spindle injury or occult Injuries. This injury is deep within muscle tissue. The muscles ache and they hurt. They don't hurt on active contraction. The chart above does not apply to this type of injury. The pain is not able to be easily reproduced. The chart is based on concentric contraction (active shortening). The muscle fibers in eccentric injury are intact, and wouldn't produce pain during contraction. The Doctor can find the pain by deep palpation and challenging the muscle eccentrically. The area will be sore, and have nodules (trigger points). Trigger points refer pain to other areas. For example, rhomboid trigger points refer pain to the shoulder. Trigger points in the neck can refer pain into the hand. The muscle is allowed to stretch during eccentric contraction. The muscle overstretch and the fibers are hypotonic due to underutilization. The body will accept the new joint position. Hypertonic muscles are over utilized. The hypotonic muscles that are underutilized are injured when forced to work eccentrically causing damage to the spindle as the spindle leaves its normal orientation in the muscle. Gamma fires the spindle and stretch fires the spindle. The firing of the spindle, fires the 1a (annulospiral) that goes to cord and goes to alpha motor neuron causing active contraction. The stretch is not removed from the spindle, because the spindle is no longer attached. The spindle cannot be shortened because it is no longer attached. This is called a spindle injury. The spindle keeps firing. The alpha fires and gamma fires. The gamma goes back to the spindle and activates the 1a. This is chronic stimulation to the spindle that is no longer allowed to relax. This theory was proposed by Dr. Travell.*

Dr. Nimmo: Promoted Travell concepts of trigger and tender points. Tender points keep the pain at the site during palpation. *Trigger points can refer pain.* This is the clinical difference, because histologically they appear the same.

Ex.—Trigger point from infraspinatus referring pain to the hand (hand goes numb)...Ask – Quality of pain? Can the patient reproduce pain? Are there signs of neurological involvement? If they are all negative for neurological involvement, then these are indicators that the pain is from a trigger point.

Massage, acupuncture, trigger point therapy all bombard the cord with mechanical afferentation. All of these are modalities to decrease pain. Decreasing pain provides a huge benefit to the patient.

**Trigger point pain is not considered sclerotogenous. Sclerotogenous pain is segmental (relates to cord segment). Trigger point pain does not relate to a segment of the cord.**

**NMS 11/28/07**

### **Double Crush Syndrome**

Each nerve possesses a certain margin of safety designed to accommodate some chronic compression without causing symptoms.

Double Crush syndrome can occur in various peripheral entrapments of the ulnar, radial and median nerves in conjunction with whiplash injury. The normal cellular transport mechanism to the distal axon by the axonal transport system is impaired.

Rand Swenson (Double Crush Syndrome)..."With current experimental data, it is safe to say that two experimental lesion along the course of the nerve have greater effects than a single lesion."

Article on Overhead..."Neurological deficit following acetabular fracture" -- The prospect of functional recovery is worse than those having a single lesion."

Journal of Hand Surgery—Picture of Double Crush



“Axoplasmic flow has been altered because of pressure on the exiting nerve root and mild compression on the median nerve. The combination of proximal and distal impingement has caused the symptomatic threshold to be surpassed.” This is denervation. This means that you are experiencing a degeneration of the nerve as a cumulative effect of both proximal and distal compression. Neither one individually can do this, both together they can. The double crush will then show + EMG/NCV findings. In double crush, mild impingement of both (cumulative) effects create problems distal to the site of distal entrapment. Best results come from treating both sites. It would be wise to treat both the cervical spine and the carpal tunnel in the case of a carpal tunnel presentation.

#### Double crush Syndrome Recommendations (for Carpal Tunnel Presentation)

1. Radiological demonstration of cervical spondylosis or other vertebral abnormalities
2. Complaints of pain and stiffness in the neck
3. Previous history of neck injury commonly of the hyperextension “whiplash” type as a result of rear-end motor vehicle accidents
4. Clinical evidence of sensory abnormalities corresponding to dermatomes, rather than peripheral nerve distribution...
5. Proximal and distal symptomatology

+ Orthopedic findings in the C-spine may indicate a double crush. Treatment may be a trial period of chiropractic.

#### *Triple Crush*

It is possible to have more than 1, or 2 sites of injury. If you remove at least 1 site of crush, you can remove some of symptomatology.

#### *Muscle Wasting, Atrophy, Fasciculations, & Weakness*

These are all indications for immediate referral. If the losses are only sensory as many problems are, then it is acceptable to treat conservatively (especially double crush by treating multiple areas). If you treat only 1 site of a double crush, there is a higher possibility that the condition may recur.

#### **Peripheral Neuropathy vs. Radiculopathy Presentations**

##### *Peripheral Neuropathy*

Pressure on a peripheral nerve (ex. Crossing leg) will cause symptoms of numbness (foot goes to sleep). Numbness is caused by pressure on a peripheral nerve. Later the area becomes number and then digits start to curl. When the pressure comes off the nerve, we then feel the paresthesia. Paresthesia comes when pressure is taken off the nerve. When you try to walk on a paresthetic leg or attempt digital movement, the leg will be in a paresthetic storm (huge increase in paresthesia).

##### Order of Symptoms in a Peripheral Neuropathy

1. Numbness – when pressure is on the nerve
  - a. More numbness and digits start to curl
2. Paresthesia – when pressure comes off the nerve
3. Paresthetic Storm – Increased paresthesia during movement

##### *Radicular Neuropathy*

*If the problem is radicular, one gets **pain first**. The patient will have **paresthesia at the same time**. Pain is the #1 symptom. Taking the pressure off the nerve root, will cause relief of pain. During time of tingling and pain, moving fingers would have no effect. **There is no effect of digital movement on pain if nerve root is affected.***

##### Order of Symptoms in a Radicular Neuropathy

1. Pain & Paresthesia– Pain occurs first and paresthesia occurs in conjunction with pain
2. Relief of Pain – Occurs when taking pressure off the nerve root
3. Movement – Movement during pain has NO EFFECT!

#### **TOS**

The arm is going to sleep and tingling. They leave out pain when describing the injury, but the patient subjectively reports the arm does go to sleep. Patient experiences numbness with Hyperabduction/Wright’s Test. If the patient experiences numbness and nothing else, it may be indicative of neurovascular entrapment. Bringing the arm down from Wright’s test position, the patient experiences paresthesia. This presentation indicates a peripheral neuropathy.

##### *Costoclavicular Presentation*

When the arm is down and shoulder depressed the symptoms come on (numbness). With the arm down, numbness occurs. Carrying something makes the arm number. Propping the arm up they feel paresthesia. Shrugging the shoulder causes paresthesia and moving shoulder down numbness. This presentation indicates a peripheral neuropathy.

People with compressive neuropathies. Most of the complaints are numbness and not pain.

Billfold Syndrome: Men who sit with big wallets may produce radiating pain

Piriformis Syndrome: True piriformis syndrome emphasizes numbness as compared to pain.

### **Neuropathy vs. Neuritis**

Neuritis: the pain is constant once the inflammation sets in

Neuropathy: Aggravated by motion in to a particular position.

**NMS – 11/29/07**

**\*\*\* IN CLASS REVIEW \*\*\***

**Dural Tests:** WLR, SLR, Soto-Hall, Vasalva

**Other Tests:** Ely, Milgram, Kemp, Braggard, Bechterew's, Percussion

*WLR/SLR*

Dominant individual leg

Shouldn't use for lumbago

*Lumbago*

Dull, diffuse pain all across the back

*Nerve Tension Signs*

Nerve tension occurs with stretching of the nerve. Leg raises are tension signs. Braggard's & Bechterew's can also be tension signs

*Vasalva*

Vasalva is indicated as a dural test not nerve tension test.

*Examples*

1. Radiating Pain on Straight leg raise (+ SLR) and + Bechterew's = Pain from a nerve
2. + Vasalva points to a disc lesion in the canal and not an IVF lesion

*When do nerve signs appear?*

When nerve is damaged

*SLR*

Tests dura and nerve tension. You may need other tests to help make a diagnosis.

*Braggard's*

Braggard = is not a dural test (strictly a **tension test**)

***Bechterew's***

Bechterew's = nerve tension only

*Example*

With + SLR and + Braggard the pain is of neurological origin (we don't know why – it could be many things). + Vasalva test added to the above tests will then tell you if that is a disc. Vasalva is for SOL in the canal. So the three tests tell you that there is a problem of neurological origin that is in the canal that irritates both dura and nerve. The diagnosis is then likely to be a disc lesion.

*When do neuro signs appear?*

When there is a **nerve entrapment or true neuropathy**. It is possible a SLR can have pain down the leg with no evidence of a neuropathy. Ex—a patient has radiating pain with + Vasalva and + SLR with a – Braggard. This may be the disc bulging and irritating the dura. The dura sleeve is probably irritated producing pain referral, but not a nerve entrapment or neuropathy.

*Can a test be both dura and nerve tension?*

Yes—SLR can be used for both. Braggard is not used for dural signs, it is strictly a nerve tension sign. A Bechterew's test or sitting with leg extended (sitting sciatic stretch) may also be both dura and a nerve tension sign.

*What can be learned from orthopedic test?*

We cannot learn etiology. We can learn **location of the chief complaint**. We can learn what **movements produce the pain**. We cannot learn if the problem is neurological or not. We must do a neurological assessment to find a true neuropathy.

*What does the sinuvertebral nerve innervate?*

The PLL and the disc itself. The disc itself can be a source of pain. Discitis (inflammation of the disc) can be terribly painful. The sinuvertebral nerve contains C-pain fibers only. Sinuvertebral nerve also contains sympathetics. The neurotransmitter released into the disc is norepinephrine and substance P. The both combined are a good combo to yield inflammation. A chronic, constant bombardment yields chronic inflammation and lots of pain. The sinuvertebral nerve is capable of having a segmental pattern because it innervates dural sleeve (distinctly segmental sclerotome referral). The pain will be diffuse, deep, dull and achy because it only has C fibers. Differentiating between PLL, dura, or cord is hard to do since they are innervated by sinuvertebral and using C fibers.

*Facets*

Facets are innervated by A fibers and some C-fibers. A facet would produce sclerotome pain patterns. Putting the facet in a Kemps test will trigger the A fibers and cause sharp, localized pain.

*How does lumbago manifest?*

It is not a segmental pain pattern. **It is extrasegmental. It is a non-segmental pain pattern; therefore, it is not appropriate to consider it as a sclerotogenous pain referral.**

*Adjustment*

The adjustment focuses on inducing motion. Motion may start the inhibition of the area and help to decrease the pain.

*How do you know if lumbago is the result of irritation to the cord dura?*

Vasalva test, Soto Hall Test and other dural tests may be helpful.

*Central Disc*

It bulges directly posterior. The disc hits the PLL (first) and then cord dura (second) and then it can hit the cauda equinae.

*Posterolateral Disc*

Posterolateral hits the dural sleeve (first) and then the nerve root (second). These bulges hitting the root produce a true radiculopathy. If they hit their sleeve they will be sclerotogenous pain.

*Lateral vs. Medial Posterolateral Bulges*

To determine which one it is you can use a WLR and signs of antalgia (leaning into the injury with medial vs. leaning away with lateral injury).

*Dural Tests*

Dural tests can reproduce sclerotogenous pain patterns, neurological dermatomal pain patterns, and extrasegmental pain patterns and not exclusive for a particular type of pain.

*EMG/NCV*

Globally, these exist to tell us whether there is a pathology of the motor unit. A motor unit is 1 alpha motor neuron innervating a number of muscle fibers. If you have pain down your leg coming from a pinched nerve in your back, perform the EMG. The pinched nerve (mixed nerve) includes motor fibers. The mixed nerve (mixed at the IVF) will have a motor finding if there is a neuropathy. This allows you to quantify the injury. When the unit fires, there should be 1 MUAP's (despite the # fibers). The more fibers, the greater the amplitude on the EMG. The smaller the amount of fibers, the smaller the amplitude. In either case, it is still called a MUAP. We look at the height of the wave to determine the size of the motor unit.

1. Decreasing amplitude (motor units decrease) but increased MUAP's = Motor unit is decreasing in size and the patient is losing muscle fibers (the unit gets smaller and amplitude is less, but you have to use many potentials to perform the movement)
2. Increased amplitude with decreased MUAP's = Indicates that you increase the unit and indicative of a neuropathy ...The unit gets bigger due to recruitment.

Ex.—Patient with TOS – A true thoracic outlet is a neuropathy. EMG can be + for distribution for anterior primary ramus (motor). If the lesion was also at the nerve root, the problems would be sensory and motor, + the injury would show a myotomes pattern.

#### *Strain*

Transverse = damage the fiber

Longitudinal = *damage the connective tissue ... Difficult to reproduce! ... The only way to isolate this is to put an eccentric contraction through it. Transverse injury is more likely to develop trigger points. Myofascial strains or spindle injuries are present. The spindle has not been damaged. There has been no damage to alpha, gamma, or annulospiral. The spindle is no longer attached. This can be an occult injury, deep within the tissue. The ROM study fails to bring out the longitudinal strain.*

#### *Peripheral vs. Nerve root Pressure*

Peripheral Pressure = less pain complaints than nerve root. Raise arm and arm goes to sleep = peripheral

#### *Double Crush*

Upton and McCombus = Double Crush Syndrome (pressure on nerve on 2 places)