

**NEUROLOGICAL MODELS → Where to start in Christy supplement (pgs 29-39)**

Nervous System in an excited state.

Hypertonicity → facilitation to a well functioning system

Radiculopathy → pinched nerve (dermatome, myotome deficit)

Facilitation model

Sensory Model (dorsal cord)

Autonomic models (lateral horns)

Motor Model (anterior cord)

**Reflexes**

SA → Somatosomatic → SE            SA+SE

SA → Somatovisceral → VE        SA+VE

VA → Viscerosomatic → SE        VA+SE

VA → Viscerovisceral → VE        VA+VE

With the names above the left part of the name is always in reference to the afferent branch. The Second part of the name is the efferent branch.

→SA fiber is an annulospiral fiber and C fibers

→SE fiber is a gamma that goes to the muscle

→Next you have another SA fiber coming from the muscle and then synapsing inside the cord on a fiber that goes to the heart (VE)

→Now the heart is in a state of dis-ease and it goes back to the cord with VA fibers.

Afferentation causes everything –

**Mooney & Robertson → “The Facet Syndrome”**

Back pain is rarely caused by a pinched nerve

- Injected prisoners with saline solution → and found that following facet injection the prisoners started having pain, and then radiated into extremities. However, most patients did not have radiating pain below the elbow or knee.

- o **Scleratomal**

- The pain was described as deep, dull, and ache, yet absence of neurological signs.
- Strength, muscle tone, EMG, reflexes are all normal
- no paresthesiae,
- no Tinel sign (peripheral pain pattern not present)

**Feinstein and Inman – “Sclerotome pain”**

Sclerotome – involved in connective tissue and is evolved from a somite

- This is segmentally related to the lesion and is hard to reproduce due to the A fibers over powering the C-fibers.

C-fibers are responsible for sclerotomal pain and dural pain. Over the counter pills will eliminate this pain. These fibers do not adapt.

A- fibers → sharp burning, discriminated, adaptable

SOMA TO SOMA → there are all kinds of referrals. (heart to the arm and jaw)

SAD → is dermatome

Viscero referral

Referral to different kinds of problems.

Dermatomal is the least often seen.

### **Handout**

**DAVID SEAMAN → How does subluxation Affect the Nervous System?**

Addl info → [www.chiroweb.com/archives/22/04/19.html](http://www.chiroweb.com/archives/22/04/19.html)

Addl info → [www.chiroweb.com/columnist/seaman](http://www.chiroweb.com/columnist/seaman)

Talking about sclerotomes (does person have neurological deficits...they shouldn't)  
Clearly, neurocompression at the IVF is not required to induce pain referral into the extremities.

Sclerotome and dermatome are TOTALLY DIFFERENT

Trigger points referral – rhomboid referral goes into the shoulder

Feinstein has pictures and documentation of the sclerotomes (shown in class)

### **Joint Complex Innervation → from above articles**

Nociceptors and mechanoreceptors are the primary sensory receptors that innervate joint structures, including synovia, joint capsules, bone, ligaments, tendons, muscles, and blood vessels. The predominant receptor is the nociceptor. **More than 90 % of joint innervation is done by nociceptors.**

Nociceptors afferents, for example, are either A-delta or c-fibers  
C-fibers are the primary fibers that innervate the spinal joints. **There are Fewer mechanoreceptors in the joints than in the muscles. Joints are predominately C-fiber/ group IV innervated.** When the spine is hurt you will increase nociceptors and decrease mechanoreceptors since you are injured and less movement will be present.

Although the spinal nerve travels through the IVF it is rare for the bone on nerve subluxation to occur. (David Seaman – says 99% is scleratome)

The subluxation does not necessarily have to be an irritant on the nerve.

### **Dr. Christy trying to explain Dr. Seaman's work.**

From the extremity you have the SA that comes up to the spinal cord and enters the dorsal columns. Then the synapse occurs and crosses the cord and goes up the cord (spinothalamic, A delta or spinoreticulothalamis, c-fibers)

16 PAIN: MECHANISMS AND MANAGEMENT

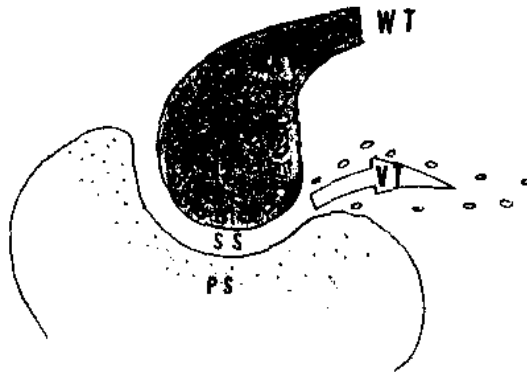


Figure 1-14. Synapse.

The wiring transmission (WT) functions by electrical stimulation of the preganglionic bulb (PR), which is charged with numerous vesicles (V) containing acetylcholine. When released across the synaptic space (SS) they cause depolarization of the postsynaptic membrane (PS) causing electrical-chemical direct neurotransmission.

The release of particles out of the synapse forms the vehicle that migrates (VT) in the interspaces to locate their receptors.

NEUROANATOMY OF PAIN MECHANISMS 17

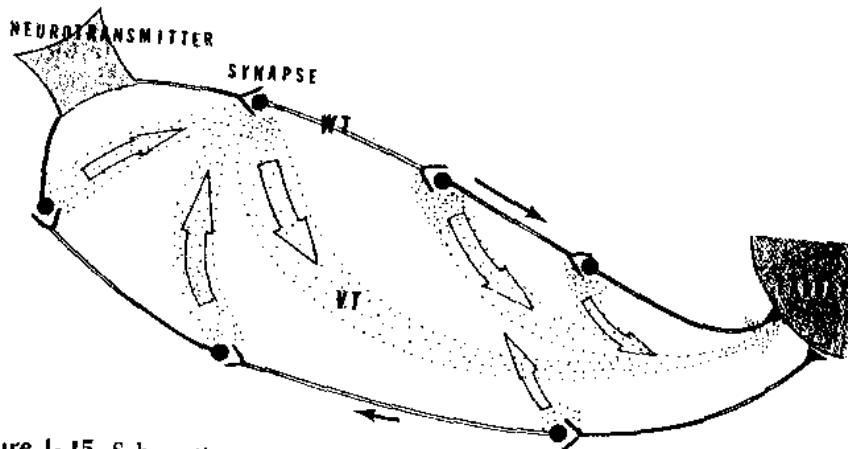
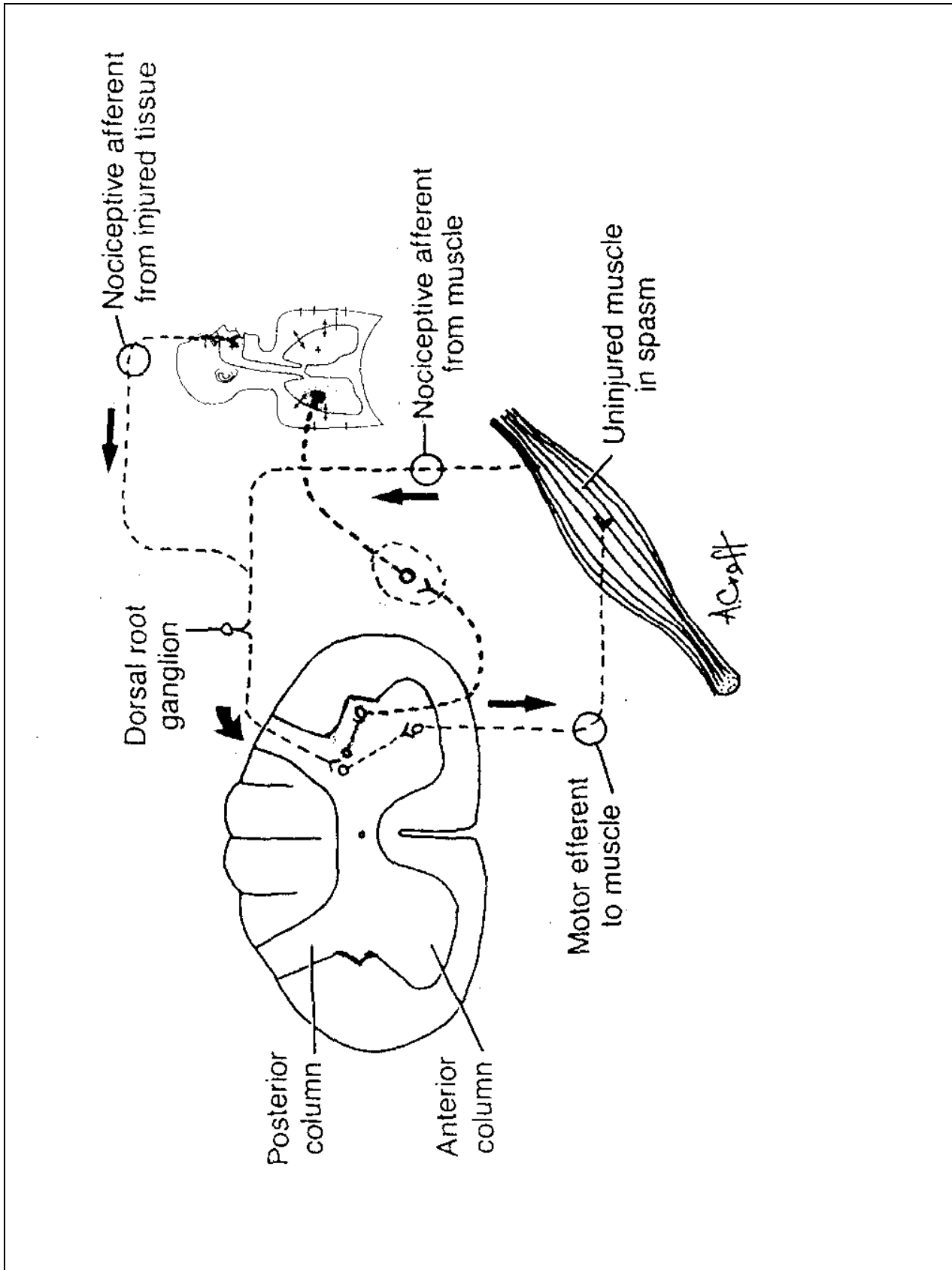
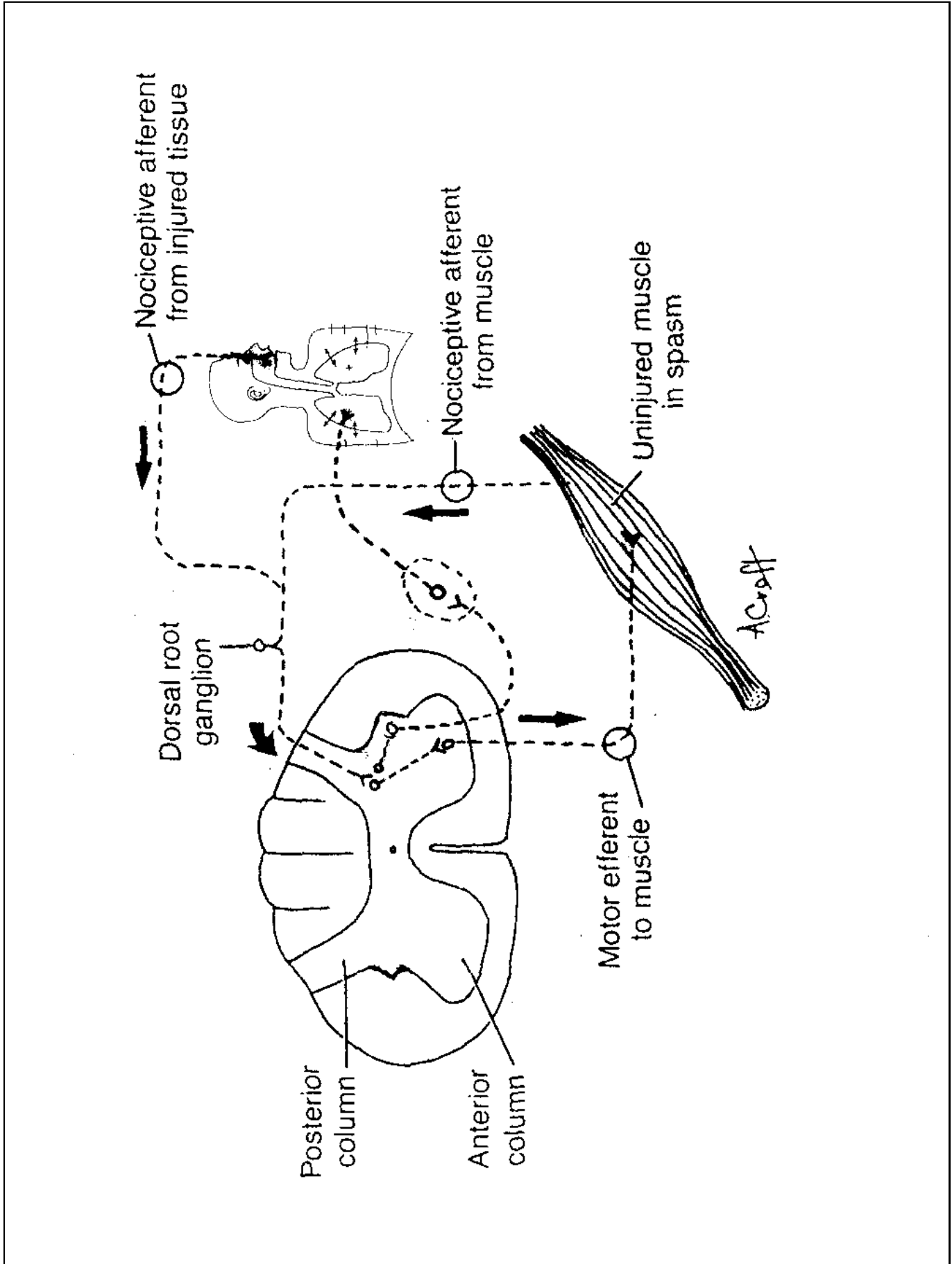


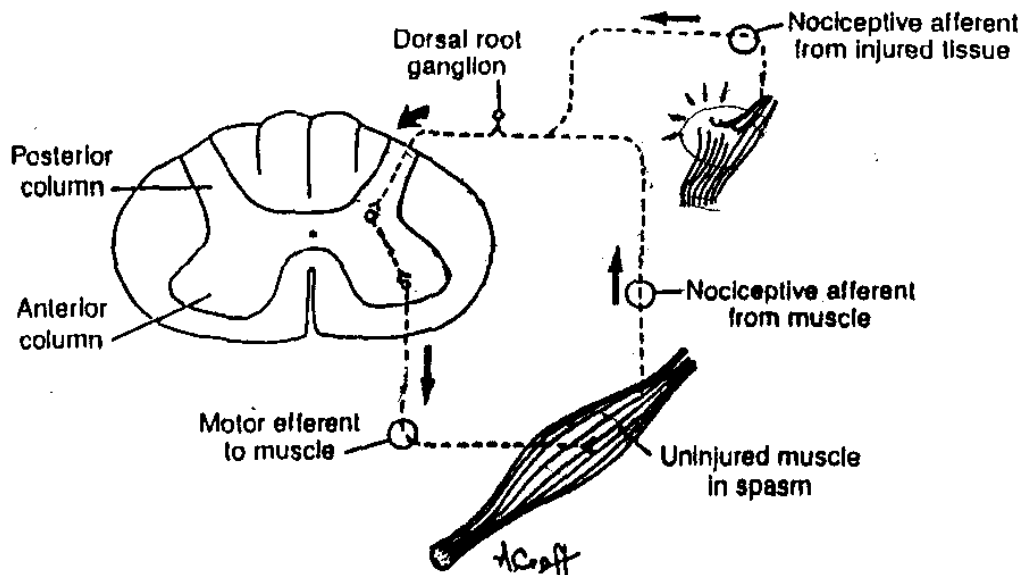
Figure 1-15. Schematic wiring transmission versus volume transmission of neurotransmission.

Direct neurotransmission resembling a direct wire transmission (WT) is depicted as going directly to its specific receptor (dark arrows) albeit via synapses.

At each synapse there allegedly leak chemical neurotransmitters that enter the interstitial spaces and chemically find their receptors. This transmission goes in afferent and efferent directions (white arrows).







**Figure 8.18.** The vicious circle of pain originates in injured tissue such as ligament or muscle and is perpetuated by reflex arc via segmental spinal cord connections, resulting in muscle spasm in segmentally related yet uninjured muscle distal to the lesion.

So, you have a facet jamming (inflammation occurring) and synovium is building up. 2 ways to stimulate nociceptors (chemically and mechanically)

A-fibers → acute pain, and subsides

- C-fibers → dull ache pain (most prevalent...90%)

Facet Joints → its innervated (mostly nociceptive fibers) and the nerve comes into the dorsal horn like the above fibers. After it enters it is releasing glutamate and stimulates nerves around it including the nerve it meant to fire on. This is why referred pain is present. The Brain thinks that something is going on in the extremity since all the nerves are firing, or are appearing to be firing.

### From Neuroanatomy of pain Mechanisms (ch 8)

Wiring transmission vs. Volume transmission

Wiring → Direct nerve to synapse

Volume → anything can happen when the light is switched (however today a segmental response can be elicited...yet the sclerotome is not as well defined as the dermatomes) with volume so much neurotransmitter

**Muscle → Muscle → lungs → respiratory system → muscle**

Pain from torn muscle → to interneuron → efferent fiber to muscle → muscle goes into spasm (SA-SE)

Supraspinatous tendonitis can cause the rhomboids to spasm (**this is a segmental relationship**) (SA – SE) in this reflex you have healthy nerves

Now the supraspinatous tendon is healed, yet rhomboids in spasm are still in spasm (secondary problem)

From the secondary spasm there is nociception taken from that muscle into the cord. This then stimulates the lungs and it receives additional input which irritates the airway. (SA-VE)

If this was happening to the heart, then beta blockers would be given beta blockers to block the norepinephrine that is released on the heart after the heart attack.

So now the airway is irritated and the upper respiratory irritation is causing additional efferent stimulation to the muscle that caused it all.(VA-SE)

**CES → central excitatory state (this is what all of this stuff is called  
No Neuropathy, and all neurological signs are NEGATIVE**

### **C-FIBERS ARE THE SOURCE OF AFFERENTATION**

Excitations of the systems that release nor-epinephrine create inflammation in the areas and leads to pathologies. The subluxation excites the nerves which cause the problem.

For 20 plus years Dr. Korr studied “sustained sympathicotonia as a factor in disease” ...a condition sympatheticonia, which is considered prevalent in many states, including chronic pain states. (this was brought up in a study after Dr. Korr)

Know about **sympatheticonia**

These areas of hypersympathetic area correlated well in a *segmental* distribution, with musculoskeletal strain, trauma, deep and superficial tenderness, electromyographic activity of paraspinal muscles, etc.

The diversity of effects of stimulating various peripheral sympathetic pathways is not in the influences of the sympathetic neurons, but in the responses of the innervated tissues and organs. The responses are as varied as the tissues and organs which are innervated---virtually every tissue in the body

Sympathetics innervate every tissue in the body?...well in physio you would say only glands and smooth muscle and cardiac, but in Dr. Christy's findings he has found different. That every tissue has some affect from sympathetic innervation, not a direct connection though (neurotransmitters floating around).

Why have sympathetics going to all tissues? **Sympathetics do not bring a new quality but will increase function**

- Do you want increased skeletal muscle contraction? Spasm
- Do you want pancreas constantly stimulated? (too much insulin)

It is the sustained sympathetic hyperactivity that causes the problems.

**Research article** → Here today and not gone tomorrow: The curse of chronic pain...

- Many chronic pain conditions have no apparent ongoing peripheral injury once a precipitating injury is healed
- Despite recovery from an initial painful condition, pain starts to be generated from within the CNS, a process now known as “central sensitization”

**Central Sensitization** → excited state of the nervous system

Dr. Joel Pickar → PhD and DC

- Sympathetic efferent discharge is thought to sensitize primary afferent nerve endings
  - Sympathetics leaving the spine to go to tissues and increase the afferent fibers firing back to the spine
  - **Nor-epinephrine is an Alpha-adrenergic**
  - Annulo spiral is primary afferent fiber
  - Sensitizes the spindle → becomes spastic → increased tone

Article → 1978

Osteopathic lesion has many aspects.

Nor-epinephrine causes the excited function (sympathetics)

- Hyperesthesia → especially of the muscles and vertebrae
- Hyperirritability → reflected in altered muscular activity and in altered states of muscular contraction
- Altered visceral and other autonomic functions.

Cardiac sympathetic afferent sensitivity is enhanced in heart failure. From heart to cord → cord to heart → nor-epinephrine released

On the basis of animal studies that have shown acupuncture to be **sympathoinhibitory**.

**BETA-BLOCKERS** → patients with heart failure were divided with high and low plasma norepinephrine (produced in adrenal cortex and sympathetic nerves) these higher levels were found to increase severity of heart failure.

- Hypothesis that the observed increase in adrenergic drive was in fact not adaptive, but rather mal adaptive for the failing heart.
- Alpha/beta – adrenergic receptor blocker carvedilol
- We thought that the nor epinephrine release was healthy since the heart was needing to pump more blood and in naturally increased the heart rate, yet this is only increasing the time to heart failure.



**Afferentation** – (VA or SA) fibers go to the dorsal horn → goes to the interneuron → efferent fibers with a Ach or nicotinic interaction → then to organ where NE is released

Catecholamine

- NE - nor epinephrine (nor adrenalin) = adrenergic → alpha, must use alpha receptors
- E - epinephrine (adrenalin) = adrenergic → alpha/beta, can use either receptor

Smooth muscles of the airway are beta. Sympathetics will cause bronchodilation...you are talking about beta (E). So what about the norepinephrine being released near by? This is sensitizing and enhancing tissue function.

### **Effects of the subluxation (looking at it at the side of exciting the NS)**

Nociception becomes the afferentation that causes the problem.

Somatic Afferents → A delta fibers

Visceral Afferents → C fibers, make it into cord over autonomics (motor)

Vagal afferents as well as vagal efferent fibers.

So where are the autonomic cell fibers? In the DRG

Nodose Ganglia (this ganglia contains the cell bodies of the vagal afferent fibers)

- the vagal afferent are irritant receptors (like in the lung if you get water down the trachea. The immediate vagal response would be coughing.
- Vagal afferents stimulate the vagal efferents. → cough, increased mucous, broncho constriction (asthma)
- Norepinephrine sensitizes primary afferent fibers to (bring the fiber closer to threshold and making firing easier)
- Vagus nerve innervates the lungs. So if you were to increase vagal output to the lungs the tissues in the air way would become sensitive

What causes an increase in Norepinephrine?

Where is the afferentation coming from?

**Glutamate** → excitatory NT released from the VA with A-delta fibers

**Substance P** → excitatory substance from C-fibers (this substance is released at both ends of the pain fiber)

- Substance P will also be released by all segmentally related C-fibers. This means that if you are having a heart problem then the NT will be released in all segmentally related lung tissue.
  - So a segmental linking from the heart to the lung
- Now prostaglandins are released into the tissue...This leads to inflammation
  - NEUROGENIC INFLAMMATION → generated by the release of substance P from the C-fibers
  - So the facet is irritated and I have acute pain from the A fibers, however most of the day I have a small ache and the muscles are slowly going into spasm as my cervical neck gets tighter. The C-fibers are also firing substance P. In the cord when substance P comes in it

says to fire all c-fibers segmentally. Norepinephrine is now being released and is sensitizing all primary afferents in the fiber.

- NorEpinephrine also does something when it lands on Mast cells → the mast cells degranulate and release histamine and SRS-A (slow reactive substance – anaphylaxis)
- Now the CNS is very excited as C-fibers are facilitated and SA & VA fibers are firing.

Overall Somatovisceral and Visceralvisceral reflexes are going on.

GABA → this is an inhibitor

- it works post synaptically
- blocks the tract cell and interneuron in the cord

**Enkephalon** → nature's opiate

- this works directly on the primary afferent fibers and prevents them from releasing the NT glutamate and substance P
- comes from the periaqueductal grey area to the DIP to the interneuron that releases the enkephalon
  - Mechano reception will stimulate the Peri Aqueductal grey

GTO, Merkel's disc, meissners, pacinian, spindle → these are all mechanoreceptors that are located with great density in the spine.

TENS unit → block pain and decrease sympathetic tone

Increased sympathetic tone can kill someone slowly and make them sick

## **Research**

CES → Central excitatory State

Visceral or somatic afferentation → cord → interneuron → efferent neuron

You also have sensory fibers coming in too. Now we look at new information. The efferent branch may not be needed at all. Substance P will actually feed backwards on the nerve.

Glutamate is NT for A-delta fibers

Substance P is NT for C fibers

## **Article from pharmaceutical therapy**

Substance P discussion

- it is a neuro peptide
- pain transmitter of the dorsal horn (concentrated there)
- Integral to the nociception input
- Very involved in pain
- Classified as a tachykinin → this can be used in place of substance P

- Neurokinin is the same, but only used in mammals
- When highly concentrated it is considered to be an excited state and it regulates the excitability of dorsal horn nociceptive neurons
- Vomiting reflex, cardiovascular tone, smooth muscle contraction (all of these effected by substance P)
- Intense peripheral stimulation (lig, facet) may induced release of substance P into the Dorsal horn, causing central hyper excitability and an **increased** sensitivity to pain ( so hyperesthesia is the result of central excitation)

### **Osteopathic relationships to the spine**

Dorsal root reflexes →

- Goes in and comes back out → does not go any where up or down the cord
- SA – VA is what were talking about
- Involving conduction of neural activity along primary afferent fibers, have been demonstrated to initiate the release of proinflammatory peptides (substance P) from the peripheral terminals into the local tissues. These reflexes are initiated by activity in primary nociceptive fibers at adjacent spinal segments. (antidromic release) (backwards)

### **Article**

Infected, using a virus, and SA fiber in a rat. Then the segmentally related bladder became inflamed. This means that there is a neurogenic effect. The virus was not found in the bladder, but only found in the SA nerves. So, as the SA fiber was infected with rabies, the bladder became inflamed. So what does this mean?

Joe Pickar → facet loading → to see if facet loading would affect internal organs by reflex effect. To see if lumbar input could affect organs.

- Muscles irritated at L2-L4 on rats. This affected the splenic and renal Sympathetic discharge and BP.
- The results indicate that there can be a SV reflex. The afferent goes to the cord and comes out the efferent to affect the organs.

### **Dorsal root reflex or Axon reflex picture**

1. Nerve Activation
2. Substance P released
3. Vasodilatation
4. Cholinergic Activation

### **Spinal cord stimulation Article for Chronic Angina**

- The effect of SC stimulation involves a mutual interaction of decreased pain and also decreased sympathetic tone. We hypothesize that the spinal cord stimulation will give benefits by lowering pain and sympathetic tone.
- The stimulation of the fibers has an inhibitory effect on the nociception of A-delta and C-fibers. Spinal cord stimulation has shown a decrease in glutamate

and aspirate, while an increase in GABA occurs. Also there has been speculation that opioid receptors exist in the presynaptic terminals of sympathetic neurons innervating the heart. Activation of these beta-endorphins may also cause a decreased release of **norepinephrine**.

- By stimulating mechanoreceptors this was all done. Electrical stimulation by TENS was found to give peripheral sympathetic inhibition.

#### Article

- Tachykinins such as Substance P (SP) and NKA (neurokinin A)
- Although now a days, asthma is recognized as an inflammatory disorder if the airways, neural mechanisms remain very important
- Therefore it has been suggested that modulation of axon reflexes could be of potential benefit in asthma treatment
- Substance P degranulates mast cells and eosinophiles. Tachykinins also enhance eosinophil chemotaxis

#### **Why do mast cells degranulate? Why is immunoglobulin E produced instead of A in asthmatics?**

Substance P is released from C-fibers

Glutamate is released from A-delta fibers

**Pretnazone** → it prevents the mast cell from degranulating. (works well with poison ivy and works well with cardiac muscle)

#### **PICTURE → neurogenic inflammation (acute inflammation)**

Coming from sensory nerve

- the C-fiber is releasing substance P and then causes the mast cell to degranulate and it will release all of the inflammatory substances
- Also substance P will cause the macrophage to degranulate too. TNF (tumor necrosis factor) is within the macrophage.

Coming from Postganglionic Sympathetic Nerve Terminal

- NE (norepinephrine) also causes degranulation of the mast cell and macrophages.

Catecholamines (adrenergics)

- norepinephrine,
  - Alpha predominately
- Epinephrine (adrenalin)
  - Alpha, beta
- Dopamine

The beta blockers today actually block alpha and beta.

#### **Article**

These findings suggest that release of NE increases sensitivity to heat in skin sensitized by capsaicin. In addition, neurogenic inflammation appears to increase access to the receptors that facilitate thermal hypalgesia.

*So, with NE being release you have alpha-adenergetic receptors firing and neurogenic inflammation.*

Patients with low levels of NE have a better prognosis than those with high levels.

**Recurrent Meningeal Nerve (sinuvertebral nerve)**→ (*innervates Dura, ligaments, periosteum, disc*) this stimulates fibers that will cause afferentation into the cord. **PIC**

- there are NO A fibers
- C-fibers Present → this means it can fire constantly and release substance P
- Perfect source of chronic sensitization
- Sympathetic fibers present
- Branches off posterior primary ramus and goes back into the vertebra

Asthma → inflammation of the airways

- effected by the
  - Dorsal Horn → scleratome
  - Lateral horn → sympathetic atonia
  - Anterior horn → spasm

Sinovertebral nerve stimulation (disc, ligament) → afferentation along the C fiber to dorsal horn → through the interneuron in the lateral horn to the anterior horn → the white rami (myelinated) → acetyl Co-A released onto Gray rami → to the lungs where NE (alpha-adrenergic) is released (however NE does not cause muscle contraction!!!!) this is because the NE does not release the right chemicals to stimulate the receptor sites (beta receptors) of the bronchioles

- NE stimulates Mast Cells (inflammatory cells) that release granules of histamine which is a MAJOR bronchoconstrictor
- Histamine has receptor sites called (ALPHA). And NE releases alpha.
  - Histamine can make a nerve fiber and works as a NT
- Increased release of SRS-A (anaphylactic granule)
- Macrophage will also degranulate in the presence of NE since it has an ALPHA receptor → leukotriene TNF released (aka trypsin) → tumor necrosis factor is released from the macrophage and effects the muscles of the airways
- NE also increases the sensory nerve fibers (J fibers → responsible in the feeling of the shortness of breath, dyspnea) (SENSITIZES them bringing them closer to threshold)
  - Since the body now is getting inflamed Substance P will be released (this is a proinflammatory substance) at the lung and at the Dorsal horn
    - **All of this is called the dorsal reflex** a VV reflex

With an adjustment you would increase GABA to reduce the nociception

3-22-05

**Spindle → maintains muscle tone**

- maintain tone in muscles as the muscle changes length
- this is whether they are shortening or lengthening
- They do NOT increase tone (an increase in tone is spasticity)
- Maintain a steady stream of afferentation throughout the motion
- Maintain stability in muscle
- Stretch will cause the spindle to fire

Dorsal excitation → scleratome pain

Lateral horn → Sympathetic atonia

Ventral horn → spasm

**Ventral horn**

The nerve looks as if it is an Alpha motor neuron, but it is really not. A different nerve (gamma MN) controls the tone by going to the spindle.

Then the spindle sends a message

Spindles found in skeletal muscle. With out tone there is no stability.

They are found in between the muscle cells

**Mechanics of the spindle**

Nuclear Chain → tonic portion of the spindle (responsible for tone)

Nuclear Bag → phasic portion (responsible for DTR)

Quick stretch will stimulate the spindle which will increase muscle tone

Long stretch will stimulate the Nuclear chain (tonic)

**GTO → muscle contraction fires this**

**Muscle spindles are fired by lengthening it**

**Annulospiral** (aka 1a fiber) enters the dorsal horn and does one unique thing (only fiber in the body that is monosynaptic...does not go through the interneuronal pool...this means it is the most important and primitive level of proprioception). It connects directly onto the alpha motor neuron.

- the purpose of the spindle is to keep the muscle from getting too long. This then causes the muscles to contract to take the pressure off of the spindle. So when the contraction takes place the spindle shortens and decreases the activity of it. (but what will bring it back to life if you decrease the activity of the spindle then you could loose tone...)
- Fucimotor system (refers to spindle) – inside the spindle (Nuclear chains and bags or **intrafusals muscle fibers**)

- Actin and myosin are present on these and are innervated by the gamma motor neuron. Gamma innervates the nuclear ends of the nuclear chains.
- GAMMA restores internal stretch and brings the fiber back up to baseline firing. (**what made gamma fire?**) Attaches at the ends of the spindle.
- Extra pyramidal pathways control tone through the fusimotor system (gamma system) (fire gamma → tone goes up) (inhibit gamma → tone goes down)
  - Spinocerebellar pathway → cerebellum → basal ganglia → extra pyramidal → Gamma motor neuron

**Barry Wyke** → considered the father of articular neurology (neurology surrounding articular joints)

\* classified type I, II, III, IV

\* Type I, II, and IV are found in the facets

\* Type I, II are Mechanoreceptors

\* Type IV are nociceptors

Found that stretching the capsule you would fire nociceptors and would activate the fusimotor system

Mechanoreceptors → pacinian corpuscles and merckles disc (inhibit incoming pain and cause muscles to relax)

Spindle readjusts → base line firing is where the spindle needs to be and Gamma does this. This is necessary for joint stability. (aka. Returns to null position)

### **Barry Wyke**

Postulate that facets innervation was (type IV) free nerve ending. His theory is that a capsule that is placed under stress (synovial lining would produce more fluid, synovial effusion). This would fire the pain receptors (type IV) fibers.

The expression from the nociceptors will enter the Dorsal horn, then fire onto propriospinal fibers (interneurons) and carry the message to the gamma motor neuron. This then exits and goes to a muscle and releases NT.

### **TEST 2**

1. What results from facilitation of the lateral horn? Sympathetic atonia (dr Korr)
2. What kind of reflex is this called? Somatovisceral or somatoautonomic (Somato autonomic response...visceral reaction to subluxation)
3. Where is the spill over to explain sclerotogenous pain? In the Dorsal Horn, which will stimulate the surrounding nerves to fire
4. What fibers create scleratome pain? C-fibers, fibers responsible fro this (NEVER A FIBERS) only C can fire long enough and hard enough. (Dr. Mooney and Robertson, wrote facet syndrome) (Dr. Finestein and Inman)
5. What is a more technical term for spillover? Volume transmission
6. Differentiate sclerotome from radicular pain! Radicular → neuropathic deficits

7. Relationship between nociception and Mechanoreception? Mechanoreception inhibits nociception (gate theory → melsak and wall)
8. When is the use of myotome and dermatome appropriate? Radiculopathy only (nerve root lesion)
9. What condition establishes a sclerotome pain pattern? Somatic lesions establish a sclerotome (soma to soma, not neuropathic or visceral) pain patten, they do **not** come from a visceral pain pattern
10. What two types are segemental? Scleratome and dermatomal pain patterns
11. What could cause parasympatheticotonia?
12. Explain how sympatheticotonia could contribute to the symptoms of asthma?
13. Give an example of parasympathetica atonia.
14. Give an example of sympathetic atonia.
15. What type of reflex does Dr. Barry Wyke discuss?
16. What type of reflex does Dr. Koor Discuss?
17. Name some Authors who discuss sclerotome pain.
18. What are the EMG?NCV findings associated with sclerotome pain?
19. Nerve “irritation” versus “compression.”
20. Know THOROUGHLY the four reflexes.