Erik Dalton’s  
Freedom from Pain Institute  

Myoskeletal Alignment Techniques®  
For Pain Management

Sensory Receptors…  
Rebels Without a Pause?

Research conclusions from ongoing studies:
• Soft tissues (previously viewed as purely mechanical structures) are innervated and participate in active balancing of the spine.
• Specialized mechanoreceptors play major roles in myofascial unwinding AND also initiate aberrant feedback loops and muscle imbalance patterns due to injured articular structures.

SENSORY RECEPTORS

• Supply CNS input on stimuli such as pain, touch, sound, light, heat and cold
• Categorized by specific physiological duties such as nociceptors, mechano, chemo, thermo and electromagnetic receptors
• Transmit proprioceptive and nociceptive information
• Change sensory stimuli into action potentials so the CNS continually receives data on the overall body environment.

 Muscle Joint Reflexogenic Relationships

Is impaired muscle function the primary cause of joint dysfunction, or is the reverse true?
• McClain 1994:
  -- Receptors monitor capsular tension
  -- Receptors may initiate protective reflexes important in preventing joint degeneration.
• Grieve:
  -- Postural asymmetry joint blockage enhances fibroblastic activity resulting in periarticular tissue fibrosis.

Catch 22 Pain/Spasm/Pain Cycle

• Murphy:
  -- Added that changes in spinal joint soft-tissue fibrosis alters the normal instantaneous axis of rotation

How Joints Affect Muscles

• Joints influence muscle tone and therefore muscle function.
• The joint’s ability to alter muscle tone is mediated by articular receptors.
• In the joint capsule, the greatest number of receptors are found in regions subject to variation of tension during movement.
• Articular receptors can inhibit or facilitate muscle tone.
ARTICULAR RECEPTORS

- **Freeman and Wyke** categorized articular receptors into four types: Type I, II, III, and IV.
- Each is stimulated in a distinctive way and responds to stimulation differently.
- Type I and II mechanoreceptors act as physiological receptors/active during normal movement.
- Type III and IV receptors normally inactive/only stimulated at extremes of movement...may function under pathological conditions.

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**Ligament Innervation**

- Dense collagen bundles of Ruffini corpuscles suggest active monitoring of mechanical joint loading and provide static positional awareness for postural control.
- Jiang’s findings support concept of ligaments as part of neurologic feedback mechanisms for protection and stability of the spine.

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**Zygapophysial Joint Innervation**

- Belief in zygapophysial joint pain dates back to 1933 when Ghormley coined the term “facet syndrome”.
- Facet innervation is derived from the medial branch of the posterior primary division at the level of the joint and the levels above and below.
- **Jeffries 1988** suggested that this multilevel innervation is probably one reason why facet joint pain frequently has a broad referral pattern.

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**McLain’s Facet Studies**

- **McLain** dissected human cervical facet capsules from three normal subjects to determine the type, density, and distribution of mechanoreceptive nerve endings.
- Mechanoreceptors were found in 17 of 21 specimens.
- McLain concluded “the presence of mechanoreceptive and nociceptive nerve endings in cervical facet capsules proves that neural input from facets is important to proprioception and pain sensation in the cervical spine.”
**Whiplash and Facets**

- **Barnsley et al.** double-blind, controlled diagnostic blocks. Investigated cervical facets in 50 post-whiplash patients. Found facets were most common source of chronic neck pain.

- Bogduk, Hirsch et al, and Yamashita et al also reported on rich innervation of facet joints.

- They concurred that altered intersegmental and segmental joint motion and postural distortions create aberrant traffic in neuropathways.

- "Cross-talk" perpetuates aberrant reflex alterations, muscular and ligamentous alterations, inflammatory responses and resultant pain syndromes.

**Discogenic Pain**

- **Roofe (1940)** 1st evidence of annulus fibrosus nerve fibers.

- **Bogduk (1983)** nerve fibers in outer 1/3 of lumbar annulus fibrosus.

- **Farfan (1973)** type 4 nerve receptors penetrating nucleus, annulus and posterior longitudinal ligament.

- **Shinohara (1970)** nerve fibers penetrating degenerated discs nuclei.

- **Garfin (1995)** disc compression of normal nerve leads to paresthesias, sensory deficits and motor loss...pain is absent.

**Wilberger and the Silent Nerve Compression Syndrome**

- **Wilberger et al 176** lumbar myelographic herniated discs in 108 asymptomatic patients.

- Within 3 years, 64% developed lumbosacral radiculopathy.

- Wilberger hypothesizes that time was required for mechanical deformation to cause this "silent nerve compression syndrome".

**Radicular Pain**

**FASCIAL PLASTICITY**

- Therapist hands often palpate a myofascial unwinding as sustained pressure is applied to superficial and deep myofascial layers.

- **Juhan** attributed alteration in connective tissue resilience to what is commonly called thixotropy or the "gel-to-sol" phenomenon.

- **Currier and Nelson** significantly more force, time and heat must be generated in order to establish permanent connective tissue deformation.

- **Oshman** added piezoelectricity as a possible explanation for fascial creep.
Robert Schleip’s Observations on Fascial Plasticity

- Schleip concurred: these mechanisms may be a viable explanation for long-term tissue changes but questioned their effectiveness for short-term tissue release experienced in clinic.
- Schleip studies on anesthetized patients: in the absence of neural connection, short-term fascial plasticity is lost.
- Schleip, “Pacinian receptors are likely to be stimulated by high-velocity thrust manipulations as well as vibratory techniques, whereas the Ruffini endings may be activated by slow and deep ‘melting quality’ soft tissue techniques.”

Golgi tendon organs

- Golgi tendon organs (GTO’s) arranged in a series respond to slow stretch by resetting a muscle’s length, inhibiting its synergistic stabilizers and facilitating its antagonist.
- Jami 1992 - passive myofascial stretching does not stimulate GTO’s.

Nociceptors as Pain-Generators

- Nociceptor mechanical, thermal and chemical stimuli.
  - Nociceptor and chemoreceptor activation:
    1. Nerve fibers depolarized by joint capsule mechanical stresses
    2. Thermal extremes
    3. Inflammatory chemical agents such as histamines, prostaglandins, bradykinins, potassium ions, and lactic acid.
- Nociceptors can quickly become major generators of both myofascial and spinal-pain syndromes.

Postural Control

- Soft tissues within and surrounding spinal articulations are densely populated with sensory receptors.
- Macro or microtrauma may create joint misalignment and postural distortions.
- Injured articular structures initiate and facilitate spinal reflex pathways which increase contractility in paraspinal musculature.
Nociceptors and Posture

- Long-term CNS agitation by irritated nociceptors causes the brain to twist and torque the body in an effort to avoid pain.
- Regrettably, the brain has the ability to memorize these aberrant postural patterns.

Nociceptors and Posture

- Dysfunctional patterns that persist long after the painful stimulus has been removed are referred to as “neuroplasticity”
- “reflex entrainment”
- or “spinal learning.”

Transversospinalis

- Muscles are the body’s primary movers and must respond quickly to changes from neural structures.
- When tight muscles pull unevenly on the body’s bony framework, the joint’s axis of rotation and center of gravity changes.
- Prolonged joint misalignment (loss of joint play) agitates sensory receptors in spinal joint capsules, ligaments, discs, and transversospinalis muscles.

Transversospinalis

- Almost always pulls insertion points toward origins when at work. As the TP are pulled toward the SP, localized rotation and sidebending occur.

Transversospinalis

- Particularly stressed are mechanoreceptors embedded in overstretched capsules and the part of the joint bearing excessive weight.

GATING

- Joint dysfunction results in muscle dysfunction by changing gamma bias of spindle cells.
- Joint injury, degeneration, inflammation, or muscle guarding causes fewer mechanoreceptive fibers.
- As we age we lose mechanoreceptors = can’t gate. Because nociceptors are free nerve endings they are not as affected.
- This explains why a minor trauma can cause much pain or a major trauma can cause only minor pain.
Co-activating Nociceptors

- Nociception originating from muscle = passive massage, joint = dynamic stimulation produces more sensory gating.

- Lederman (1997) found that successful nociceptive gating requires that the stimulus be pain free or that the gating movements take place within a pain free range.

Joint Techniques to Lower Pain-Generating Stimuli

- Spinal soft tissue manipulations that initiate passive joint movements result in mechanoreceptive stimulation.

- This technique creates presynaptic inhibition of the nociceptive afferent to diminish or abolish the perception of pain.
- *Sandoz*—restoring normal joint structure/function helps normalize mechanoreceptive and nociceptive input.

Cutaneous vs. Articular Receptors

- Massage primarily stimulates cutaneous receptors. Active or passive movements primarily stimulate articular receptors = less joint pain.
- Active client participation better gates articular nociceptors.
- Active (rather than passive) positioning improves proprioception since muscles are allowed to play a larger role.

Passive Cutaneous Massage Release
**Active Articular Release**

**Muscle Inhibition or Atrophy?**
- **Janda 1988** “Although muscle weakness has usually been considered a result of decreased activity, inhibition may be an integral part of many, if not all, forms of weakness.”
- **Hurley (1997)** - muscle weakness - two factors:
  1. Decreased number of extramuscular muscle fibers
  2. A failure to activate all muscle fibers
- A decreased number or size of extramuscular fibers may be termed *atrophy*, whereas failure to activate all muscle fibers may be termed *inhibition*.

**Muscle Imbalance Patterns**
- **Janda’s Upper and Lower Crossed Syndromes** - 2 of most common aberrant postural patterns.
- Exposed to same stressors certain muscles become tight and facilitated, others weak and inhibited.
- Abnormal afferent information:
  - Painful or noxious stimuli
  - CNS malregulation
  - Psychological (emotional) stressors
  - Poor posture
  - Excessive physical demands
  - Joint blockage
  - Habitual movement patterns

**Upper Crossed Syndrome**
- Are the weak lower shoulder stabilizers solely responsible for the aberrant forward head posture seen in the upper crossed syndrome?

**Upper /Lower Crossed Syndromes**
- **Porterfield and DeRosa** - forward posture factors other than scapular retractors stretch weakness:
  - Weakness and lengthening of abdominal muscles allow the chest to fall causing an anterior upper trunk weight shift.
  - As gravitational exposure pulls upper trunk forward on the rib cage, the scapulae externally rotate and protract - forcing clavicle to drop on the first rib.
  - The clavicular head of pectoralis major and hypertonic latissimus dorsi internally rotate the humerus forcing the neck and head to follow.

**Nociceptive Reflexes and Somatic Dysfunction**
- **Somatic Dysfunction Model** - restriction in mobility, autonomic, visceral, and immunologic changes produced by pain-related sensory neurons and their reflexes.
- Nociceptive muscular guarding reactions and autonomic activation from stressed/damaged myoneural or visceral tissue.
- **Guarding** - abnormal myoneural position and decreased ROM.
- Local inflammatory responses and autonomic reflexes reinforce nociceptive activity, maintaining restriction.
- Nociceptive autonomic reflexes - visceral/immunologic changes.
- Abnormal guarding in muscles, joints, related tissues - changes in connective tissues, solidifying the abnormal position.
- Stretching tissues into normal range of motion may re-stimulate nociceptors, reinforcing the somatic dysfunction.
CONCLUSION

• Patients benefit by restoring balance/function to all soft tissue structures.
• A model for using receptor techniques to correct aberrant postural patterns is helpful in the clinical setting.
• Impaired Neuromusculoskeletal functions can cause stress, pain and altered performance of internal organs, hormonal systems and psycho-immunological functions.
• Working with the sensory receptor system, trained therapists can determine if problems are primarily within muscles, fasciae or joint-related tissues or if the problem exists elsewhere.
• With assessment and treatment training, a therapist can more efficiently determine dysfunction sites and improve structure.
• This leads to higher functioning in the self-regulating and self-protecting mechanisms of the body.