Identifying Central Pain Mechanisms

2011 California Physical Therapy Association Annual Meeting
Richard Kring, PT, DMT, DPT, FAAOMPT

Segmental Facilitation

Facilitated Segment

- Korr: “barriers having been lowered.”
- Korr and Upledger:
  - Reduction in the stimulus threshold in a particular spinal cord segment. When becoming highly excitable, a smaller stimulus will trigger excessive impulse firing in the segment.

Hair triggered

- Nerve roots: “hair triggered”.
- Hyperactive motor root joins the sympathetic chain state of constant over-activity. further increasing facilitation.

Threshold Spill Over

Facilitated Segment

- Denslow:
  - Result of a sustained barrage of subthreshold impulses into the segment of the cord with the lesion.
  - A central excitatory state is established.

Segmental Facilitation

Location

- Areas of postural stress
- Sites of trauma
- Areas related to visceral problems.
- Once a segment is facilitated, all of the target structures (connective tissue, muscle, bone, blood vessels, skin, sweat glands, and internal organs) will be affected.
- Must interrupt the self perpetuating cycle.

Influence

- Influenced by:
  - psychic
• emotional
• traumatic
• bacterial
• physical.

11

• Pain is not what occurs at the periphery
• Pain: what the brain perceives: indisputably modifiable by emotions and beliefs
• Actual damage is neither necessary nor sufficient for the actual perception of pain
• Anger, depression, anxiety, fear, and other psychological variables can all increase the perception of both acute and chronic pain

12

Findings
• Palpable changes: muscle & subcutaneous
• Decrease in joint mobility
• Fibrositis: tender tissues
• Sympathetic: changes in tissue texture, sweat gland activity, and capillary blood supply to skin.
• Increase in muscle electrical activity

13

Treatment
• Focus on: relaxing the muscles
• Mobilization
• Reducing
  • stasis and edema
  • postural stress
  • the number of signals from higher centers in the CNS.
• Must break the cycle and reduce the number of signals from higher centers of the CNS.

14

Osteopathic Lesion / Somatic Dysfunction

15

Osteopathic Lesion
• Denslow: “Impaired or altered function of related components of the somatic system: musculoskeletal, arthrodial, and myofascial structures, and related vascular, lymphatic, and neural elements

16

Definitions cont.
• Korr: sustaining factor in disease
• Establishes and maintains a vicious cycle of irritation, inflammation, and pathological process
17  **Korr:**
- "chronic segmental facilitation, a localizing, channelizing, and predisposing influence in the bodily expression, mental or emotional imbalance"
- Cole: Instigates, augments, and maintains an alteration in function through reflex activity of the nervous system, including the ANS.

18  **Multiple factors influence the process:**
- Psychic
- Emotional
- Traumatic
- Bacterial
- and physical.
- Same as FS.

19  **Results**
- Greater tension on one side increasing proprioceptor firing. Afferent input will increase tension, increase reflexive activity, increase sensitivity to the gamma motor neuron and increased alpha motor neuron output.

20  **Myofascial Pain Syndrome**

21  **Clinical Presentation**
- Hyperesthesia of the muscles and vertebrae
- Hyperirritability with altered muscular activity
- Changes in tissue texture and local circulation
- Altered visceral function.

22  **Clinical Findings: OL**
- Denslow: Clinical evidence: hyperalgesia, abnormal skin texture, anatomic assymetry, and disturbance in range and ease of joint motion.
- Digital palpation: painful, thickened soft tissue, muscular rigidity.
- Hyperalgesia due to visceral, emotional, and other disturbances.

23  **Treatment**
- Manipulation:
- Induces muscle relaxation
Removing excitation
Easing tension on proprioceptors
Lowering the bombardment of impulses from that segment to the spinal cord

24 Cellulotenoperiosteomyalgic Syndrome

25 Maigne: Cellulotenoperiosteomyalgia
CTPM Segmental dysfunction altering the sensitivity and texture of soft tissues: detected by careful palpation of the cutaneous (cellulalgia), muscular (myalgia), and tenoperiosteal tissues.
These will effect the same tissues for a given segment.

26 Maigne: Findings
Subcutaneous swelling and induration in all or part of the dermatome: cellulalgic
Indurated myalgic cords (trigger points) localized in some muscles of the myotome
Hypersensitivity of the tenoperiosteal insertions of the scleratome on palpation.

27

28 A focusing factor, the irritation of the corresponding spinal nerve. Near constant bombardment of the spinal cord can bring about a state of facilitation of a neuron whose cellular body is localized to the same spinal cord segment and provoke motor and autonomic responses.

29 Neurodynamics

30 Neurodynamics: Background
Walsh2005 and Shacklock 2005
“Mechanical and physiologic properties of the peripheral nervous system that are dynamically interdependent, and correlates the effects of tension and excursion on the peripheral nervous system.”
“Alterations in neurodynamics will manifest as adverse neural tension. Changes in neural physiology and mobility may result in the development of the patient’s symptoms.”
Treatment to restore normal neural physiology
Neurodynamics and Neuropathy
- Adverse neural tension presents with neuropathic pain as a primary feature.
- Neuropathic pain: “pain initiated or caused by a primary lesion or dysfunction in the peripheral nervous system”

AIGS: Abnormal impulse generating site
- If a nerve is damaged by trauma or disease, it may become demyelinated resulting in an abnormal impulse generating site.
- Central sensitization - causative factor, may need to be treated to break cycle.

Neuropathic pain
- May be linked to stress via the sympathetic nervous system and antalgic posturing to relieve mechanical tension on the offending neural tissues.

Neural tension testing:
Findings
- Active and passive motion dysfunction
- Palpable neural hyperalgesia
- Local tender points in tissues innervated by the hypothesized segment.
  - Myalgia and cellulalgia
- Presence of local dysfunction.
  - Segmental dysfunction

Central Sensitization
Woolf 1993: (Melzak and Wall)
- CS is a response of the dorsal horn neurons to normal afferent input which is augmented or facilitated. This is the result of modification of the receptive field properties of dorsal horn neurons including:
  - Reduction in threshold
  - Increase in responsiveness
  - Recruitment of novel inputs: A-beta
  - Increase in size of the receptive field.
  - Spread of pain to uninjured sites.
    - Ji and Woolf
Cellular change from repeated input due to functional plasticity of the CNS.
Local anesthesia to the site of injury has no effect.
Recruitment of receptive fields outside of this region means a change within the CNS and not an increased sensitivity of the peripheral terminals of sensory fibers innervating the injured tissue – peripheral sensitization.
CS vs PS: PS is a source, not needed to maintain.

May become sensitized by: an emotional event or residual somatic symptoms of depression or anxiety rather than from a peripheral painful input.

Stress may sensitize emotional centers in the amygdala and limbic cortex, to precipitate anxiety and affective disorders that are amplified and endure long after the stress has abated.
Rationale for chronic pain clinics.

Guarding against the possibility of pain and anticipation of its occurrence activates cells in the rostroventral medulla that function to amplify incoming pain signals at the level of the Dorsal Horn.

The simple act of anticipating a pain and expecting it to be important are sufficient to trigger these “on cells”. In essence activating the amplifiers before the pain stimulus has begun!

Descending pathways
Stem from cortico-reticular system, locus ceruleus, hypothalamus, brain stem, and local spinal cord interneurons.
Ascending and descending pathways are interactive and function bidirectionally; both pathways have the properties of both facilitating and inhibiting pain, depending on the site of action.

(Negative affect activates the right cerebral hemisphere that potentiates further adverse stimuli, including pain, integrating the depression and pain in these patients.)

Nucleus Raphe
Magnus & Nucleus Gigantocellularis
Facilitate and maintain secondary hyperalgesia.
Also implicated in descending inhibition.
Reversible blockade (ropivicaine) of NRM or Gi prevents both primary and secondary cutaneous mechanical hyperalgesia.

44 NMDA receptors
- NMDA receptors of the rostral ventromedial medulla are involved in maintenance of both cutaneous and muscle hypersensitivity after muscle insult.
- NMDA receptors in nucleus reticularis gigantocellularis are associated with cutaneous hypersensitivity.
- Repeated muscle insult results in sensitization of the RVM and GiC by increased glutamate release, increased NMDAr responsiveness, or increasing expression of NMDA receptors.

45 Referred Pain
- Theore: Impulses from deep tissue injury producing sensitization of the referred area by axon reflex
- Misinterpretation of inputs whose axons also branch to the referred areas
- Convergence projection theory: axons from the injured and referred areas converge on the same cells in the spinal cord with a misinterpretation
- Facilitation of cells in the cord along with axons from the referred area.
- Modulation of somatosensory sensitivity at referred sites is mediated by central mechanisms.

46 Visceral

47 Visceral

48 Visceral

49 Visceral
- Korr: referred pain of the viscera to the soma in areas morphologically separate from the disease process though related segmentally in the nervous system.

- Cutaneous, muscular (rigidity and sustained contraction), and spinous process tenderness to the level of the viscus.

50 Visceral
- Input from irritation in the viscera renders the nerves to the same segment, hyperirritable, the “irritable focus”.

51 Visceral
- Secondary tissues of irritation may become a source of irritation: vicious cycle.
- Then there is no further dependence on afferent impulses due to sustained facilitation.
- Treatment: interruption of cycle
HEART (pericarditis)
- Sympathetic segment
- T1–4 (T5)

Electrical Skin
- Resistance
  - Characteristic electric skin resistance patterns 3 weeks prior to coronary occlusion (Korr 1949).
- Symptoms
  - Pressure on chest, squeezing, heaviness, burning indigestion, stabbing shortness of breath, anxiety
  - Dyspnea– shortness of breath
  - Pain sometimes down both arms.

Segmental Dysfunction
- Comparing segmental somatic dysfunction with significant accuracy for 50 patients with:
  - 1. Cardiovascular disease
  - 2. Pulmonary disease
  - 3. Gastrointestinal disease
  - 4. Disorders in the musculoskeletal system

Sympathetic Distribution

T4 and Coronary Disease
- A double blind study documenting palpatory T4 findings with coronary artery disease defined by cardiac catheterization.

Placebo:

Theories
- Expectancy theory: expectations influence response.
- Negative expectation: negative response: nocebo
- + or – attitude of provider affects pt’s belief = response
- Conditioning theory: past experiences results in conditioning
• Involves pre-frontal and limbic areas = stimulation produce physiological outcome.
• placebo results in opioid activation in certain brain regions.
• Anterior cingulate, orbitofrontal, insular cortex, nuc accumbens, amygdala, PAG.
• Cognitive information processing, analgesia, and reward expectations.

• Positive outcomes regardless of real or sham treatment
• “Yellow Emperor’s Inner Classic” – Huang Di Nei Jing
• Written in 1st century BCE
• “If a patient does not consent to therapy with positive engagement, the physician should not proceed as the therapy will not succeed”

• Tx is less effective when patient is unaware than when tx is given overtly. This difference can represent the placebo component of the treatment, even though no placebo is given.
• Highlights the importance of the patient / provider interaction.
• being told that a pain killer has been given has been shown to be as potent in pain relief as 6–8 mg of morphine.

Radiology
• Over-reliance on imaging findings derives, in part, from the persistence of obsolete concepts concerning nociceptive pain.
• A situation can be worsened by health care providers who attribute the pain to incidental findings on imaging that may bear only a modest relationship to the pain!

Pain from Noxious Stimuli
(Physiologic Pain)

Pain from Segmental Facilitation
(Pathophysiologic Pain
– Neuropathic Pain)
69 **Chronic Pain**
(Complex Regional Pain Syndrome)

70 **Testing for Segmental Facilitation**

71 **Signs and Symptoms of FS**
- Motor
- Sensory
- Subjective numbness
- Hyperactive immune system
- Visceral
- Psychological

73 **Clinical Testing for FS**
- Basic neurological evaluation
- Excessive sudomotor activity (sweating) (Sugenoya et al. 1990, Allen et al. 2004)
- Scratch test: histamine/sympathetic reaction (turns white)
- Scratch test: hyper-reactivity (central and peripheral)
- Pinch / Roll (central and peripheral)
- Interspinous Ligament Palpation
- Trigger points and somatic points in distribution
- Piloerection (hair follicles), altered color or temperature (Allen et al. 2004).
- Electrical skin conductance (segmental)
- Cold sensitivity / not heat (segmental)

74 **Nerve Root Pathology**

75 **Scratch Test:**
**Histamine**
• Symmetrical scratch along posterior spine
• Initial redness (histamine response of vasodilation)
• Sympathetic system causes vasoconstriction (turns white)
• Level of lesion has asymmetrical loss of redness

76 Scratch Test: Hyper-reactive
• Centrally: scratch anterolateral cervical spine from C1–C7 levels
• Peripherally: scratch around limb, crossing dermatome levels
• Positive is hyperalgesia in region

77 Pinch-Roll Test
• Test for segmental thickening and hypersensitivity of the skin and subcutaneous tissues (Maigne 1980).
• Skin taken between the thumb and fore finger of the examining hand and rolled.
• The area of painful, thickened skin found on the involved side differs from the contralateral region where the skin is normal in texture and sensitivity.
• Central anterolateral neck or lateral to spinous process.
• Peripheral around extremity, crossing dermatomes.

81 Suggested Treatment
for Segmental Facilitation

82 • Remember:
• The most recent evidence shows that pain is a creation of the nervous system and not just a gauge of nociceptor activation!
Soft Tissue Techniques:

Indications
- Pain
- Muscle guarding
- Decreased extensibility
- Decreased circulation
- Lymphatic congestion
- Scar tissue adhesions

Goals
- Decrease / inhibit pain
- Decrease / inhibit muscle guarding
- Increase extensibility
- Improve circulation
- Improve lymphatic drainage
- Remodeling of scar tissue

Articulation
- Vicenzino: mobilization proximal to the injured tissue decreasing hyperalgesia.
- Sterling: 22% increase in ppt pressure pain threshold, increased motor function, increased skin conduction and decreased temp.
- Length of effectiveness is 45 minutes.

Correlates with sympatho excitatory effect of tx.
- 9–15 min of mobilization: very effective, 3 minutes nearly no effect. must be site of pain or proximal. Not cutaneous.
- MT relieves pain through: 1. stimulate healing response in peripheral joints. 2. modify chemical environment of peripheral nociceptors. 3. activate segmental inhibitory mechanisms. 4. activates descending descending pain control. 5. positive psychological influences of the therapeutic intervention.

Similar effect as lateral column on PAG.
• Analgesia associated with sympatho excitation and motor facilitation. This analgesic response is not effected by naloxone at a similar dose to reverse morphine. This suggests a non-opioid form of analgesia, similar to that of produced by stimulation of the PAG.

88 □ Manipulation as Tx:
• Serotonergic fibers originate in the RVM
• Agonists to serotonin and Alpha2 –adrenergic receptors produces analgesia, decreased hyperalgesia and decreased dorsal horn activity.

89 □

90 □ Manipulation
Summary
• Joint manipulation produces a non-opioid form of analgesia.
• Mediated by spinal serotonergic and noradrenergic receptors utilizing descending inhibitory pathways from the RVM and Pons.

91 □ Manipulation
Approaches
• Robert Maigne:
  • Rotate away from pain (neurophysiology model)

92 □ Medical Exercise
Therapy (1965)
• “Through a systematic approach and with active participation by the patient, Medical Exercise Therapy aims to improve one or several functional properties by utilization of objectively graded activity.”

93 □ Exercise Principles are the Same in Athletes as in Patients, the Difference is in Dosage and Specificity

94 □ Tissue Training
Effects:
• Muscular Influence: Strength – Volume
• Vascular Influence: Endurance
Articular Influence:  R.O.M. – Shock Absorbance
Collagenous Influence:  Mobility – Stability – Elasticity
Neuro-Muscular Influence:  Coordination

95 Manipulation Above and Below the Level of Lesion
• Results suggest that a C7–T1 manipulation induced changes in pressure pain thresholds in both right and left C5–C6 zygapophyseal joints in healthy subjects.

96 Cervical Manipulation for Segmental Tone Inhibition
• Cervical patients with inhibition in their biceps, cervical ROM restricted laterally and increased pressure pain sensitivity.
• Manipulation at C5/6/7.
• Significant reduction in biceps inhibition, increase in biceps force.
• Cs ROM and pressure pain thresholds increased.

97 Cervical Manipulation for Segmental Tone Facilitation
• High-velocity low-amplitude thrust, right rotation C5/6, increases resting EMG activity of the biceps bilaterally, irrespective of whether or not cavitation occurs (Dunning J, et al. 2008)
• Cs manipulation of dysfunctional cervical joints may alter specific central facilitatory and inhibitory neural processing and cortical motor control of 2 upper limb muscles (Taylor HH, et al. 2008).

98 Reduce Water in Glass
• Treat the cause not the symptoms
• Tissue repair training
• Biomechanical issues
• Training deficits
• Posture / ergonomics
• Psychological
• Diet / nutritional / immune system

References not already sighted
References not already sited

• Cole WV. The osteopathic lesion syndrome. VII. the effects of the experimental osteopathic lesion, chemical irritants in the muscle at the level of the sixth thoracic segment, and chemical irritants in the liver. J Am Osteopath Assoc. 1949;49:135–141.
• Cole WV. The osteopathic lesion syndrome. X. the effects of an experimental vertebral articular strain on the sensory unit. J Am Osteopath Assoc. 1952;51:381–387

References


References

Woolf CJ. Central sensitization. Anesthesiology [Central sensitization]. 2007;106:864–867

New updates


Placebo References


**Sluka References used**

• Articulation:
  • Skyba DA, Radhakrishnan R, Rohlwing JJ, Wright A, Sluka KA. Joint manipulation reduces hyperalgesia by activation of monoamine receptors but not opioid or GABA receptors in the spinal cord. Pain. 2003;106:159–168.

**Sluka Continued**

• Hingne PM, Sluka KA, Blockade of NMDA receptors prevents analgesic tolerance to repeated transcutaneous electrical nerve stimulation (TENS) in rats. J Pain. 2008;9:217–225
• Tillu DV, Gebhart GF, Sluka KA. Descending facilitatory pathways from the RVM initiate and maintain bilateral hyperalgesia after muscle insult. Pain. 2008;136:331–339

107 Old School

Segmental Facilitation
• Segmental innervation of blood vessels described (Di Palma and Foster 1942)
• Segmental circulatory: skin temperature, erythema and photoelectric measurement of the vascular coloration of the skin (Wright et al. 1960)
• Segmental pattern for cutaneous tenderness and muscle rigidity (Korr et al. 1962)
• Segmental increase in sweating (Thomas and Korr 1951)
• Segmental spasm of erector spinae (Denslow et al. 1947).
• Inhibition tissue regeneration with hyperactivity of the sympathetic system (Cruickshank 1957).
• Influential impacts of segmental facilitation include neurogenic pulmonary edema, peptic ulcer, pancreatitis, arteriopathy, cardiovascular-renal syndromes, heart disease, hypertension, post-traumatic pain syndromes, hepatotoxicity and shock (Korr 1978).