Joint Session with ACOFP, AOASM and AAO: Motor Control, Pain, Somatic Dysfunction, Core Stability

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Motor Control, Pain, Somatic Dysfunction, Core Stability

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How do we move?
Decision to move (Executive or Cognitive Stage)

Movement (Feed-back or Muscle Stage)

Access Motor Program (Effector or CNS Stage)

Sensory feedback

Higher brain functions

Spinal Processing
Movement Patterns

- Movements are coded throughout the nervous system.
  - They are not coded in a single place such as the motor cortex, cerebellum, thalamus, or spinal cord.
- Movement appears to be a series of learned and activated behaviors.
  - We learn by making mistakes.
- Just as we can learn an adaptive movement pattern, we can learn a maladaptive pattern.
  - The difference between adaptive and maladaptive is often a matter of timing.
Organization of CNS

- Organized into relatively autonomous functional systems, for example:
  - Discrete systems for 5 special senses
  - Different classes for movement, such as:
    - Eye, hand, upper extremity, lower extremity
  - Language
- But all must function cooperatively together.

Transformation of sensory information

- Information is either attenuated or amplified at every synaptic relay, depending on the state of arousal.
- Nearly all sensory information is organized topographically, at each level.
- This forms a sensory map of information from the receptor surface.
- This is not just represented spatially, but also by density of receptors.
Hierarchical organization

- Sensory and motor information processing is hierarchically arranged.
- Within a system, some areas of the cortex are designated primary, secondary and tertiary.
  - “Primary” motor cortex is so named because it contains neurons that activate somatic motor neurons in the spinal cord.
    - This is the final site of cortical processing for motor commands.
  - Primary sensory areas receive most of their information from thalamus, which receives from peripheral receptors.
    - The primary sensory areas are the initial site of cortical processing for sensory information.

Peripheral nervous system anatomically distinct

- PNS supplies the CNS with a continuous stream of information concerning both the external and internal environment of the body.
- Somatic and autonomic divisions

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<tr>
<th>CNS</th>
<th>PNS</th>
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<td>Neurons</td>
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<td>1. impulse formation</td>
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<td>2. impulse conduction</td>
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<td>3. information processing</td>
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<td>Choroid plexus cells</td>
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Somatic division

- Includes sensory neurons from skin, muscles and joints.
- Cell bodies lie in the dorsal root and cranial ganglia.
- Receptors associated with these cells provide information about muscle and limb position, touch, pressure from the skin, and nociception.

Autonomic division

- Mediates visceral sensation, motor control of the viscera, vascular system/flow, and exocrine glands.
- Contains sympathetic, parasympathetic and enteric systems.
- These all are reflected to, and play a roll in, emotion and motivation.
Facilitated Segment

- Korr reasoned that the effects of OMT must be mediated by the nervous system.
  - Nothing else has the ability to react so quickly
  - Hypothesized, based on information at that time, that it was mediated by the muscle spindle.

Facilitated Segment

- Korr, Denslow, Patterson: found that it did not just involve the muscle spindle, but the entire segmental region, to include the sympathetics, and that these changes were maintained over time.
  - Proving neurological basis of SD
  - Facilitation (sensitization): long term changes in the relationship of inhibitory/excitatory interneurons to favor certain firing patterns.
  - This is what maintains the somatic dysfunction.
Sensitization and Nociception

- By the 1980’s it became clear that more yet was involved to explain what was happening either in regard to SD or OMM.
- It was well documented that the certain firing patterns could be inhibited or excited by interneurons, and reasonably accepted as part of the phenomena of SD.
  - “Sensitization” was determined to be a better term.
- Van Buskirk introduced the idea that “nociceptors” were involved in the maintenance of SD.
  - One of the criticisms was that his model was too complicated.

Nociceptors

- Nerve endings sensitive to noxious or potentially noxious (mechanical/chemical) stimuli
- Clinically, three areas of pain perception to be considered:
  - Skin: superficial somatic pain
  - Locomotor: deep somatic pain
  - Viscera
- Of these, only skin is adapted to localize pain exactly to the site of injury.
  - All others refer pain based up embryological enervation from which they originated.
Nociception vs. Pain

- **Nociception** is the activation, transduction and conditioning of neural activity from various nociceptors in the body.

- **Pain** is the conscious perception of inputs to the cortex which results in what is called pain.

- There may or may not be a close correlation between the two.

- Many factors influence the perception of nociception.

What is “pain?”

- Pain: "an unpleasant sensory and emotional experience associated with actual or potential tissue damage or described in terms of such damage."

  *Merskey H, Pain terms: a list with definitions and notes on usage. Recommended by the IASP Subcommittee on Taxonomy. Pain, 1979;6:249

- It is not a primary sensation such as taste, smell, touch, vision, hearing.

- It is an emotional state such as love, hate, or sorrow.

  - This is one reason it is so difficult to explain one's pain to another person.

- However, unlike (?) other affective states, pain is typically felt in some particular part of the body.
Types of Pain

- Adaptive: acute and chronic inflammatory and deep pain
  - A\textsuperscript{TM} myelinated (10m/s)
    - Bright pain causing action to avoid situation.
  - C: unmyelinated (1m/s)
    - Dull, burning, causing inactivity, worry, rest so tissues can be repaired.
    - Major culprit in chronic pain progression

- Non-adaptive: neuropathic pain
  - Manifestation of neural damage or change
  - Serves NO adaptive value
  - Very difficult to measure, but often causes immeasurable harm to the individual

Afferent Nociceptive System

- Fibers enter spinal cord, divide into short ascending and descending branches, terminating at various groups of relay neurons in dorsal horn spinal grey matter, mostly basal spinal nuclei.
- Afferents then cross cord to turn upwards on contralateral side, connecting basal spinal nucleus with thalamus (hence "spinothalamic" tract).
Spinothalamic Tract

- Most fibers in tract do not ascend directly without interruption, but instead synapse with brainstem reticular system, while others re-enter spinal grey matter to synapse with internuncial neurons.
- Eventually most get to thalamic relays, but after many synapses.
- Thalamic neurons then ascend to cerebral cortex.
- There are four thalamocortical projections (that we know of).

Thalamocortical Projections

- **1st projection**: superior paracentral region, contributing “perceptual component” of pain.
- **2nd projection**: medial and anterior thalamus to the frontal lobes, the “emotional component.”
- **3rd projection**: medial thalamic to cortex of ipsilateral temporal lobe, for recent and long term memory.
- **A 4th projection**: relates some thalamic nuclei to subjacent hypothalamic nuclei. This is probably the source of the visceral reflex effects and hormonal changes associated with chronic pain.
“Pain”

- Therefore nociception is distributed to at least 4 parts of the brain, each contributing to a specific component of “pain.”
- Intensity of pain is not determined by intensity of peripheral stimulation, but largely depends on peripheral and central modulation.
- Over time, changes occur in the spinal cord (and probably all levels), altering neural processing: sensitization, long term sensitization, fixation, and finally cell death and neural sprouting.

Sensitization

- Occurs rapidly, within seconds
- Due to activation of interneurons by nociceptive inputs and from mechanoreceptors (A\textsuperscript{\textregistered} fibers)
- Usually goes away rapidly once the input ceases.
Long-term Sensitization

- Occurs with more prolonged inputs
- Last for hours or longer
- Is due in part to NMDA (N-methyl D-aspartate, plays a role in synaptic plasticity) activation in internurons by substance P
- Very complex process
  - Might want to check *Science*, 304, 7 May 2004, 811 and 884

Fixation

- Occurs with inputs lasting 30-60m, depending on intensity.
- Lasts for days.
- Can be enough to cause spontaneous activity in interneuron pool.
- Product of early gene up-regulation, cell changes, etc.
Loss of inhibitory interneurons

- With sufficient nociceptive input, inhibitory interneurons die.
  - Paradoxically, opioids can do the same thing, resulting in increasing hyperalgesia (opioid tolerance).
- Shift balance toward excitation and effectively amplifies any nociceptive input.
- This may be permanent.

Sprouting

- With nerve damage to c-fibers, central terminalis die away in lamina II.
- Sprouts from Aδ fibers in lamina II and Aβ from lamina I grow to synapse onto the interneurons formally activated by c-fibers.
- Now, movement or light nociceptive inputs can cause activation of pain pathways.
- This can be very debilitating.
Glial cells

- Several types of glial cells in the central nervous system.
- Microglia are involved in some types of neuropathic pain.
- Under some circumstances they are activated and release compounds that sensitize neurons in nociceptive pathways.
- The role of glial cells in the CNS deserves its own presentation. What we have learned in the last several years alone is astounding.

Concluding the spinal cord

- All these changes are potential amplifiers of input to any signals coming into the cord and underlie many processes such as hyperalgesia, even allodynia and referred pain.
- Substance P is necessary for most long term processes and alterations in interneuron excitability.
Brainstem and cortex

- Nociceptive inputs go to many upper brain areas.
  - Important, nucleus paragigantocellularis (PGI), near hypothalamus.
  - PGI outflows to locus ceruleus (LC).
    - PGI activates sympathetics
    - LC activates brain.
    - Basis of increased arousal and vigilance with pain.
    - Inputs from here and other areas to limbic system.
    - Essentially driven toward depressed state.

Cortical alterations

- Altered cortical inhibitory cells in prefrontal cortex.
  - Prefrontal cortex is wired to limbic system.
- Cell death
- Close correlate to spinal cord changes.
Psychological Implications

• Prefrontal cortex contains inhibitory interneurons to limbic system and is heavily interconnected to it.
• Limbic system is primary area for emotions.
• Emotions control what affect is being expressed.
• This is the core of a person’s “I.”
• This is the final perception of pain.
• The psychological aspect can be changed, maybe permanently, by cortical changes.
• New research shows some regeneration of neurons is possible under some circumstances.

Spiritual implications?

• Frontal lobes and spirituality
• Book of Job
  • Implications of the story
  • Meaning of the story
• How does spirituality interact with “I”?
• How does a person’s world view affect pain? How is a person’s world view affected by pain?
Conditioned response

- Expectation of pain influences perception of pain.
  - People who expect more pain have more pain.
  - People who expect less pain have less pain.
- This is akin to conditioning a stimulus response.
  - Note that we are discussing pain not nociception.
  - Nociception is not changed, only the perception of pain.

But is there a connection?

- Recently shown that there are direct neurological connections between adrenal medulla and the motor cortex.
- Importantly there is greater linkage (somatic mapping) of core musculature to adrenal medulla.
- Additionally, direct linkages demonstrated between motor and sensory cortex suggesting more integration than previously recognized.
Origin and pathways for top-down influence over the adrenal medulla by the cerebral cortex.

Richard P. Dum et al. PNAS 2016;113:9922-9927

Mind–body circuits.

Richard P. Dum et al. PNAS 2016;113:9922-9927
Significance

- “How does the ‘mind’ (brain) influence the ‘body’ (internal organs)? We identified key areas in the primate cerebral cortex that are linked through multi-synaptic connections to the adrenal medulla. The most substantial influence originates from a broad network of motor areas that are involved in all aspects of skeletomotor control from response selection to motor preparation and movement execution. A smaller influence originates from network in medial prefrontal cortex that is involved in regulation of cognition and emotion. Thus, cortical areas involved in the control of movement cognition, and affect are potential sources of central commands to influence sympathetic arousal. These results provide an anatomical basis for psychosomatic illness where mental states can alter organ function.”

So, it **is** all connected!

Directly
Implications

- Motor control, visceral function, sympathetic activation, cognition and affective behaviors are all linked directly to one another.

- Somatic dysfunction is a form of abnormal motor control for which the body must compensate. It is neurologically mediated and a manifestation of the entire central nervous system. It is not limited to any single structure or single aspect of the nervous system.

- The condition of the musculoskeletal system is an essential component of health and disease.

How do we affect this?

- Firstly, it is important to treat somatic dysfunction.

- In some instances we must do more to affect the motor programing.
  
  - Pilates, tai chi chuan, and yoga are all examples of motor retraining programs that can be effective for many people.
  
  - Sometimes the underlying pathology interferes with a patient’s ability to perform these activities, and she/he may need more targeted therapy addressing the abnormal motor patterning.
Upper and Lower Cross Syndromes…

…are common patterns of abnormal motor control. They can be easily identified, relatively quickly assessed, and treated. This will be the focus of our following laboratory section.

I would be remiss…

• …if I did not mention a recent Cochrane review published in *Spine*, August 2016, “Motor control exercise for non-specific low back pain.”

• “Conclusion. MCE is probably more effective than a minimal intervention for reducing pain, but probably does not have an important effect on disability, in patients with chronic LBP. There was no clinically important difference between MCE and other forms of exercises or manual therapy for acute and chronic LBP.”

• I rather disagree…