Combined Sections Meeting: Value versus Obligation

As I am writing this the American Physical Therapy Association (APTA) is preparing to hold the Combined Sections Meeting (CSM) in Anaheim, California during the week of February 15th. This is an annual meeting in which those APTA members who are also members of one of the many sections of the APTA come together to learn, network, meet and socialize. I must admit that I look forward to this meeting each year and if you have never attended then I would suggest that you are missing a lot and that you should plan on attending next year. Besides some great programming and educational opportunities, this is where many new relationships are made and even some business gets done. I greatly enjoy connecting with colleagues that I only get to see once a year in some cases and renewing relationships with others that I see more often.

As this is one of the two primary national conferences held by the APTA on a yearly basis we must ask ourselves if we have an obligation to attend at least one of the two conferences. As professionals we should be active members of our national and state organizations even if we do not always agree with the national agenda or decisions that are made on the national level. Without membership and active participation we really have no basis for complaining or wishing that things were different. Attending one of the two national

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CSM
continued from page 1

conferences helps to keep us involved and keeps us informed.

Each year when I attend CSM I enjoy seeing the great attendance by physical therapy students. Many of them will present posters (I have 3 student groups doing so this year) or platform presentations and it is great to see their enthusiasm and involvement. Often many of the most popular educational sessions are jammed packed with students and practicing therapists and this is also great to see. Most of the time when I attend a session I am pleased with the presentation. This is also time to set agendas for research with colleagues and network with them to gain ideas, share experiences and just renew relationships. I usually leave a bit tired from a lack of sleep but also renewed in my enthusiasm for a profession that I have found to be rewarding on so many levels.

Going to CSM causes me to reflect on my own career and realize that we have many good days ahead of us in this profession. If you attended this year great, I hope that you had a great experience and if you did not make it this year then I hope to see you there next year, if you see me stop and say hello.

Adding Psychosocial Factors Does Not Improve Predictive Models in Patients With Spinal Pain Enough to Warrant Extensive Screening for Them at Baseline.


OBJECTIVE: A prospective, multi-center chiropractic practice-based cohort study in Belgium and the Netherlands to determine whether certain psychosocial factors provide added value to predict recovery.

METHODS: 917 patients, of which 326 with neck pain and 591 with low back pain, completed self-administered questionnaires at baseline and at 3, 6 and 12 months. They provided information on several demographic, biomedical and psychosocial variables. We used lasting perceived recovery as outcome, i.e. recovery at all follow-up moments from 3 months on. 27 Potential predictors of outcome were used to build the predictive model. We conducted stepwise, backward GEE regression models to take into account the clustering of patients within practices. To assess the added value of the psychosocial variables, we compared two model fit indices.

RESULTS: After adding psychosocial variables, predictors in the final model for neck pain included occupational status, BMI, duration of complaints, previous treatment and patient expectation (model fit marginally improved - AUC: from 0.684 to 0.695, % correctly predicted: from 65.0 to 66.1%), and in the final model for low-back pain the selected predictors included country of treatment, age, duration of complaint, previous imaging and somatization (AUC: from 0.669 to 0.715, % correctly predicted: from 68.6 to 69.5%). Only a minority of chiropractic patients scored high on psychological variables.

CONCLUSION: Psychosocial variables have little added value in predicting outcome in patients presenting to the chiropractor with NP or LBP. We therefore advise chiropractors not to screen extensively for them at baseline. Identification of the small subgroup with high psychosocial scores and high risk for chronicity needs further investigation.
Peripheral inflammation causes mechanical pain behavior and increased action potential firing. However, most studies examine inflammatory pain at acute, rather than chronic time points, despite the greater burden of chronic pain on patient populations, especially aged individuals. Furthermore, there is disagreement in the field about whether primary afferents contribute to chronic pain. Therefore, we sought to evaluate the contribution of nociceptor activity to the generation of pain behaviors during the acute and chronic phases of inflammation in both young and aged mice.

We found that both young (2 months old) and aged (>18 months old) mice exhibited prominent pain behaviors during both acute (2 day) and chronic (8 week) inflammation. However, young mice exhibited greater behavioral sensitization to mechanical stimuli than their aged counterparts. Teased fiber recordings in young animals revealed a twofold mechanical sensitization in C fibers during acute inflammation, but an unexpected twofold reduction in firing during chronic inflammation. Responsiveness to capsaicin and mechanical responsiveness of A-mechanonociceptor (AM) fibers were also reduced chronically. Importantly, this lack of sensitization in afferent firing during chronic inflammation occurred even as these inflamed mice exhibited continued behavioral sensitization. Interestingly, C fibers from inflamed aged animals showed no change in mechanical firing compared with controls during either the acute or chronic inflammatory phases, despite strong behavioral sensitization to mechanical stimuli at these time points.

These results reveal the following two important findings: (1) nociceptor sensitization to mechanical stimulation depends on age and the chronicity of injury; and (2) maintenance of chronic inflammatory pain does not rely on enhanced peripheral drive.

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### Some words hurt more than others: Semantic activation of pain concepts in memory and subsequent experiences of pain.

Theory suggests that as activation of pain concepts in memory increases, so too does subsequent pain perception. Previously, researchers have found that activating pain concepts in memory increases pain perception of subsequent painful stimuli, relative to neutral information. However, they have not attempted to quantify the nature of the association between information studied and ensuing pain perception.

We subliminally presented words that had either a low or high degree of association to the word ‘pain’, although this was only partially successful and some words were consciously perceived. Participants then received randomized laser heat stimuli, delivered at one of three intensity levels (low, moderate, high), and we measured the effect of this on behavioral and electrophysiological measures of pain. Participants (N = 27) rated moderate and high intensity laser stimuli as more painful after viewing high relative to low associates of pain; these effects remained present when measures of mood, anxiety and physical symptom reporting were controlled for.

Similar effects were observed physiologically, with higher stimulus preceding negativity after high relative to low associates and greater amplitudes for the N2 component of the laser-evoked potential (LEP) after presentation of high associates in the moderate and high laser intensity conditions. These data support activation-based models of the effects of memory on pain perception.

In response to recent publications from pain neuroimaging experiments, there has been a debate about the existence of a primary pain region in the brain. Yet, there are few meta-analyses providing assessments of the minimum cerebral denominators of pain. Here, we used a statistical meta-analysis method, called Activation Likelihood Estimation (ALE), to define 1) core brain regions activated by pain per se, irrelevant of pain modality, paradigm, or participants, and 2) ALE commonalities and differences between patients with chronic pain and healthy individuals.

A subtraction analysis of 138 independent datasets revealed that the minimum denominator for activation across pain modalities and paradigms included the right insula, secondary sensory cortex, and right anterior cingulate cortex (ACC). Common activations for healthy subjects and pain patients alike included the thalamus, ACC, insula, and cerebellum. A comparative analysis revealed that healthy individuals were more likely to activate the cingulum, thalamus and insula.

Our results point towards the central role of the insular cortex and ACC in pain processing, irrelevant of modality, body part, or clinical experience, thus furthering the importance of ACC and insular activation as key regions for the human experience of pain.

Experimental manipulations of pain catastrophizing influence pain levels in chronic pain patients and healthy volunteers. Pain. 2016 Feb 11

Pain catastrophizing (PC) has been related to pain levels in both patients experiencing acute or chronic pain and in healthy volunteers exposed to experimental pain. Still, it is unclear whether high levels of pain catastrophizing lead to high levels of pain or vice versa. We therefore tested if levels of pain catastrophizing could be increased and decreased in the same participant via hypnotic suggestions and whether the altered level of situation-specific pain catastrophizing was related to increased and decreased pain levels, respectively.

Using the spontaneous pain of twenty-two chronic tension-type headache patients and experimentally induced pain in twenty-two healthy volunteers, participants were tested in three randomized sessions where they received three types of hypnotic suggestions: Negative (based on the 13 items in the Pain Catastrophizing Scale (PCS)), Positive (coping-oriented reversion of the PCS) and Neutral (neutral sentence) hypnotic suggestions. The hypnotic suggestions significantly increased and decreased situation-specific PC in both patients and healthy volunteers (p < 0.001). Also, the levels of pain intensity and pain unpleasantness were significantly altered in both patients and healthy volunteers (p < 0.001). Furthermore, regression analyses showed that changes in pain catastrophizing predicted changes in pain in patients (R² 0.204-0.304; p < 0.045) and in healthy volunteers (R² 0.328-0.252; p < 0.018).

This is the first study to successfully manipulate PC in positive and negative directions in both chronic pain patients and healthy volunteers and to show that these manipulations significantly influence pain levels.

These findings may have important theoretical and clinical implications.
CHRONIC LOW BACK PAIN: 
WHAT YOU SEE IS WHAT YOU GET

JUNE 24-26 | MINNEAPOLIS, MN

ADDITIONAL SPEAKERS:

Tim Flynn PT, PhD
Louie Puenteura PT, PhD, DPT
John Childs PT, PhD
Steve Schmidt PT, M.Phys
Kory Zimney PT, DPT
Mark Pirtle PT, DPT
Megin Sabo John PT, DPT
Janette Ahrndt PT, MPT

Mikki Townshend PT, DPT, OCS
John Groves PT, DPT
Tim Irving DC, MS, LMT
Rasa Troup MS, RD, CSSD, LD
Franz Macedo DO
Hollis Krug MD
Ensor Transfeldt MD

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• Snacks and beverages during breaks
• Vendor Fair
• Door prizes and swag bags
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• Saturday afternoon reception
• Conference T-Shirt
• Morning fitness class
• Detailed handouts of each presentation
• Up to 18 CEUs for Physical Therapists
Patients with depression often report pain. Evidence regarding altered pain sensitivity in depressed patients remains, however, inconclusive. In a large cross-sectional study we investigated the association between depression and pain sensitivity with regard to two different dimensions of pain sensitivity, as well as the impact of somatic co-factors, symptom severity and subtype of depression.

In 735 patients with a current episode of major depression and 456 never-depressed controls, pain thresholds (pressure pain thresholds, PPTs) were measured at the index finger pad and self-rated suprathreshold pain intensity ratings were obtained using the Pain Sensitivity Questionnaire (PSQ-minor subscore), an instrument that assesses pain intensity in daily life situations. Additionally, lifestyle factors, medical and psychiatric conditions were assessed. Unadjusted, patients with depression had lower PPTs and higher PSQ-minor scores indicating increased pain sensitivity.

After adjusting for potential mediators, such as poor sleep quality and physical inactivity, patients did not differ from controls regarding PPTs, but still had significantly higher PSQ-minor ratings. Among patients with depression, severity of anxiety symptoms predicted higher PSQ-minor scores. In conclusion, we found a differential effect of depression on the two pain sensitivity dimensions: Decreased experimentally obtained pain thresholds were explained by depression-associated somatic factors whereas increased self-rated suprathreshold pain intensity ratings were associated with increased anxiety symptoms.
What Every Patient Needs

A Clinical Pearl by Adriaan Louw, PT, PhD, CSMT

In all the years I’ve been exposed to pain science, this issue has been front and center. Given to us by Louis Gifford, this applies to every healthcare provider on the planet, regardless of where they practice.

Patients are interested in the answer to four questions:

1. What is wrong with me?
2. How long will it take?
3. What can I (the patient) do for it?
4. What can you (the healthcare provider) do for it?

Again, this applies to all patients - be it a knee replacement, cancer patient, fibromyalgia, back pain, and so forth. The healthcare provider will have a long-lasting effect and needs to use his or her knowledge and experience to satisfy these questions. Furthermore, these four key issues need to be incorporated into all pamphlets, brochures, etc. So, let’s review all four and give a few guidelines.
BACKGROUND: Lateral epicondylalgia (LE) is a musculotendinous condition characterized by persistent pain, sensorimotor dysfunction and motor cortex reorganization. Although there is evidence linking cortical reorganization with clinical symptoms in LE, the mechanisms underpinning these changes are unknown. Here we investigated activity in motor cortical (M1) intracortical inhibitory and facilitatory networks in individuals with chronic LE and healthy controls.

METHODS: Surface electromyography was recorded bilaterally from the extensor carpi radialis brevis (ECRB) muscle of 14 LE (4 men, 41.5 ± 9.9 years) and 14 control participants (4 men, 42.1 ± 11.1 years). Transcranial magnetic stimulation of M1 was used to evaluate resting and active motor threshold, corticomotor output, short- (SICI) and long-latency intracortical inhibition (LICI) and intracortical facilitation (ICF) of both hemispheres.

RESULTS: In individuals with LE, SICI (p = 0.005), ICF (p = 0.026) and LICI (p = 0.046) were less in the M1 contralateral to the affected ECRB muscle compared with healthy controls. Motor cortical threshold (rest: p = 0.57, active: p = 0.97) and corticomotor output (p = 0.15) were similar between groups. No differences were observed between individuals with LE and healthy controls for the M1 contralateral to the unaffected ECRB muscle.

CONCLUSIONS: These data provide evidence of less intracortical inhibition mediated by both GABAA and GABAB receptors, and less intracortical facilitation in the M1 contralateral to the affected ECRB in individuals with LE compared with healthy controls. Similar changes were not present in the M1 contralateral to the unaffected ECRB. These changes may provide the substrate for M1 reorganization in chronic LE and could provide a target for future therapy.

WHAT DOES THIS STUDY ADD: Lateral epicondylalgia (LE) is a common musculoskeletal condition characterized by elbow pain and sensorimotor dysfunction. The excitability and organization of the motor cortical representation of the wrist extensor muscles is altered in LE, but the mechanisms that underpin these changes are unknown. Evidence of less intracortical inhibition mediated by both GABAA and GABAB receptors, and less intracortical facilitation mediated by NMDA receptors, in the M1 contralateral to the affected extensor carpi radialis brevis muscle in chronic LE compared with healthy controls. Altered activity in intracortical networks may contribute to altered motor cortex organization in LE and could provide a potential target for future treatments.

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