



CAPM NEWSLETTER

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PRE-AMBLE AND DISCLOSURES—DELAYED RECOVERY POST (NECK) TRAUMA PART ONE

BY GLORIA GILBERT, PT, MSC, CAPM EDITOR

Your editor has been attending meetings of IASP, CPS and other Regional Forums on ‘Pain’ and Pain Management for more than 30 years. During that time period (in fact since 1967!), I have continued to practice physiotherapy—either in a hospital out-patient department or in a community setting. Since 1982, I have owned a private physiotherapy clinic in London called The Downtown Clinic. The Clinic name has a second title - ‘Physiotherapy & Health Counselling’. Even in 1982, I was aware that physiotherapy services alone could not assist all our patients.

Because I owned my own clinic, I was not encumbered by ‘time’ (an element many health practitioners have little of). I was therefore able to spend the time needed to both assess, treat and most importantly LISTEN to (prolonged at times!) to what my patients were telling me—i.e. how the pain ‘felt’ and how the pain and resultant disability had impacted their lives and also how hard they were trying to get back to their normal lives—but could not!

In order to HEAR and understand our patients’ concerns more distinctly, we developed a vocabulary in our Clinic to talk to our patients about what and how they were feeling—i.e. trying to separate physical from emotional from ‘other’ feelings. It provided us with a vehicle to problem solve more effectively. And yet, the majority of my colleagues continue to discuss PAIN patients and PAIN treatment and PAIN personalities as if there is a commonality in all patients with ‘chronic pain symptoms’. My years of experience including reading and reviewing the literature would suggest that the majority of post-traumatic patients are trying hard to get back to their normal routine. If they were healthy, active, people a minute before the trauma, why would that ‘suddenly’ change?—i.e. personality, coping style etc. *(It is acknowledged that as a time passes after the trauma and the patient is NOT able to resume their pre-accident lifestyle—anxiety, worry and sometimes a clinical depression may make rehabilitation from a physical event more challenging).*

This newsletter will attempt to review some of the issues affecting delayed recovery post trauma. Many of the articles cited include references; others have reviewed the literature and provide clinical insights. Much information on the subject is very current. The newsletter is therefore far from a stand-alone document. Yet, it is anticipated that it will provide the reader with sufficient information and resources to further investigate the physiologic and anatomic mechanisms at play when assisting your patient with post traumatic pain.

This newsletter will help in better managing the patient with delayed or slow recovery post trauma. The format of this newsletter is somewhat different than previously published newsletters. As material was reviewed and provided, it became evident that the topic Post Traumatic Pain may have to be continued in future editions.

Your input about the relevancy of this topic to your areas of practice will make the decision! Please ensure that you inform your editor if you wish this topic to be continued.

For those of us who treat people who have sustained injuries in motor vehicle accidents (MVA), we are often faced with OPINIONS of other health professionals who may not agree with an ongoing treatment approach. This newsletter will NOT address that other ‘clinical problem’ at this time.

Information in this newsletter is based on published peer-reviewed journals and documents, as well as some clinical questions.

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EDITORIAL

BY GLORIA GILBERT, PT, MSC, CAPM EDITOR

The only extensive review articles on 'delayed/chronic post traumatic (neck or headache) pain' are found in the whiplash (MVA) literature. It is therefore this literature-base that has provided us not only with the long term sequelae of 'neck pain' but also with the possible association between Traumatic Brain Injury and Chronic Post Traumatic (neck) pain.

Dr. Eleni Hapidou, psychologist at Chedoke Hospital in Hamilton has submitted the first of what may be a series of articles on Post-Traumatic Stress Disorder (PTSD). Before we can discuss 'Chronic pain and PTSD', it is necessary to understand and appreciate the mechanisms of the development of PTSD.

Dr. David Rosenbloom, Pharm D and Professor of Medicine at McMaster University has added his opinion on SSRIs and their possible side effects.

Submitted by Dr. Howie Vernon, DC, PhD are a list of questionnaires (pain, disability, psychological impact) that can assist us with better assessment, and direction for treatment.

An article on cranio-sacral therapy written by Yonina (Nina) Chernick, RMT, CTS-D has been included as well. This health provider continues to integrate what may be referred to as complementary but may one day be seen as more main-stream treatment into individualized rehabilitation plans. Medication alone cannot treat trauma. The patient with post traumatic neck (as well as bodily pain) and symptoms of mild traumatic brain injury may benefit from 'different than the usual' treatment—including cranio-sacral that can assist with lessening the intensity of the musculo-skeletal pain and also managing more 'alignment' and 'fuzzy' symptoms of a head injury. (the term fuzzy will be elaborated on in the newsletter.)

Unlike his usual editorial comment about progress and administration concerns of the CAPM, Dr. Eldon Tunks, psychiatrist and President of CAPM has submitted an article on 'Central sensitization'.

As your editor, I have taken the liberty of reminding us about developing a pain vocabulary to dialogue more efficiently with our patients. As members of this Academy, we must be ever vigilant of 'looking outside the box' of what may be seen as traditional medical care and ensure that we are both investigating and providing appropriate treatment for our patients. As noted in the last newsletter, most patients do not want to be medicalized—the over-use of medication may mask the actual symptoms for which treatment is available. In closing, the editorial in the Winter 2013 Academy newsletter

was titled : ***Should we worry about some pain management trends?*** i.e. the fact that more and more **pain clinics** appear to be in the 'business' of pain management and not all have either a multi-disciplinary focus or even a physician available 24/7! A comment from a senior anesthesiologist has noted that ***"my fear is that they have not found the solution and are about to discover the problem"***. That sentence sums up the dilemma quite well. Chronic pain management (post trauma or otherwise) is generally challenging, time-consuming, and frustrating (for both practitioner and client). Only with appropriate goal setting and monitoring will success occur. Hopefully these essential ingredients will be factored into the new pain clinics.

REMINISCENCES OF A PAIN EXPERT

GLORIA GILBERT, PT, MSC

Dr. Harold Merskey, emeritus professor of Psychiatry, UWO (now called Western University) and founding member of the International Association for the Study of Pain (IASP) was instrumental in establishing the definition of pain used by IASP.

In 1994, with Dr Nikolas Bogduk, the word pain was defined as ***'An unpleasant sensory and emotional experience associated with actual or potential tissue damage or described in terms of such damage'***.

Recently, I asked Dr. Merskey for his current 'thoughts about pain'. It was interesting that the development of his approach to managing pain came from an initial psychological basis only. He told me that he had ***'started to do research on pain in response to the suggestion that pain could result from purely psychological causes. I had not previously thought of it but was aware that headache can follow from emotional stress and be felt more keenly when we are unhappy—although the same may be true of almost any unpleasant physical or emotional event'***. Dr Merskey continues with ***'after publishing a book on pain with Graham Spear in 1967 on Psychological Aspects of Pain, I returned to my then main interests which were Neuropsychiatry and Psychology. However, relatively few psychiatrists were writing on pain and patients started coming to me for help with diagnosis and treatment and suggestions of a psychological process causing their pain. That sequence is mentioned to emphasize that my initial investigations started into a purported primary psychogenic focus of clinical pain, however for practical purposes, and also for good theoretical reasons, we must continue to see pain as primarily due to physical causes that can—like almost all chronic illness—produce significant emotional change needing care'***.

Dr. Merskey cautions not to diagnose a patient with Post Traumatic Psychogenic Pain but certainly a secondary Post Traumatic Stress Disorder following trauma is possible.

PERSISTENT POST-TRAUMATIC NECK PAIN GLORIA GILBERT, PT, MSC

The literature on post-traumatic neck pain is primarily available through articles written about Whiplash or Whiplash–Associated Disorders (WAD)—the latter a term developed by The Quebec Task Force (QTF) (whose conclusions over time were acknowledged to be based on poor research but which have continued to be used to this day!).

Although not all neck trauma is resultant from a motor vehicle accident, it is that literature that we must use (since we have no other series) to discuss patterns of recovery. The reader must keep in mind that a slip and fall, an object hitting the head (from a shelf, from a window bracket, a bicycle or other work or sports-related accidents can also cause neck trauma and prolonged symptoms. The QTF and early articles after that time stated that approximately 75% of people who sustain neck injury (in a work related, not necessarily a MVA) will have recovered within 2 years. Little information was provided about the mechanism of injury or whether or not the person could resume all their pre-accident functional activities (home, work, leisure, social). The QTF document was flawed because ‘recovery’ was defined as when the client no longer had a work disability claim—not when they returned to work.

Many articles about ‘neck pain 2 years post trauma’ list the percentages of between 10-20%, i.e. people who will be left with chronic (persistent) neck pain.

Even when a multi-disciplinary model of care was followed—the same percentages of ‘disability’ continued 2 years post trauma. This health practitioner has hypothesizing that it is possible that some of those people may have ALSO sustained a MTBI or TBI—a Mild Traumatic Brain Injury (which has gone undiagnosed and untreated) **In a Clinical Review of Whiplash** published in Pain in 1994, Barnsley, Lord & Bogduk reported that structures explaining chronic neck pain included-

- Injuries to the zygo-pophyseal joints (facet joints)
- Intervertebral discs
- The upper cervical and anterior longitudinal ligaments
- The cervical musculature
- The temporo-mandibular joint

The authors also noted that symptoms other than pain may occur through damage to the sympathetic trunk, inner ear and esophagus. Symptoms of dizziness or disequilibrium occurring in association with other auditory or vestibular symptoms were also reported—as well as the fact that dizziness often occurred in the absence of clinically apparent vestibular or neurological dysfunction.

Remember—this was in 1994, and the authors acknowledged that their current diagnostic tools may not be specific enough to discern possible damage to the cervical and proprioceptor structures in the head and neck (as well as other structural changes). Many review articles have been written since 1994—on chronic pain—on TBI. Some authors have begun to link the words together.

Our investigative techniques have improved. The Ontario Neuro-trauma Foundation has published Guidelines (see next article). Yet we continue to be confounded by symptoms of dizziness, balance, nausea, vestibular and visual complaints as well as memory and concentration problems, without appreciating the fact that they COULD be related to the initial (head) trauma-whiplash or whiplash associated disorder.

The Sports World will be instrumental in assisting us to glean more information about head trauma (concussion, traumatic brain injury) and hopefully how to treat and manage symptoms. Hockey player Sydney Crosby truly is a hero to this clinician. The sportsperson with a ‘concussion’ (another INVISIBLE disability like pain) has been able to change the way amateur as well as professional sports personnel manage their ‘injured’ players.



Perfect summer day is when the sun is shining, the breeze is blowing, the birds are singing, and the lawn mower is broken.

~James Dent

MILD TRAUMATIC BRAIN INJURY (MTBI)

GLORIA GILBERT, PT, MSC

The diagnosis of a MTBI is usually made by administering a Glasgow Coma Scale (GCS) or the Ranchos Los Amigos Scale (RLAS) for cognitive functioning in the first 48 hours post-trauma. However, there are many reasons why people with post-traumatic neck pain may NOT have been diagnosed with a head injury:

- No report of loss of consciousness.
- No direct head trauma.
- Lack of recognition of unusual behavior, extent of injury, signs of concussion.
- No documentation of outcome measure (GCS, RLAS) at the time of trauma and/ or initial post-trauma management.
- No standard follow-up of the trauma victim.
- Lack of findings on imaging or
- Focus on the more critical acute injuries which may have MASKED the symptoms of a possible brain injury.

The definition of TBI used by the American Congress of Rehabilitation Medicine (ACRM) is 'a traumatically induced physiologic disruption of brain function' manifested by one of the following:

1. Any period of loss of consciousness.
2. Any loss of memory for events immediately before or after the accident.
3. Any alteration in the mental state at the time of the accident.
4. Focal neurological deficits which may or may not be transient.

We therefore have many challenges ahead, to both Identify & Diagnose a TBI since:

- There is an inconsistency in the definition and classification of TBI.
- People with mild TBI may not present to the hospital due to effects of the TBI such as poor judgment, inability to think clearly, make decisions, problems remembering what happened, whether they hit their head or were unconscious.
- Those who do go to the ER may be discharged without adequate documentation.
- Differences remain in diagnostic tools and admission criteria.
- X-rays shows skull fracture only, CT scan, MRI or functional MRI may need to be considered.
- Outcome measures (GCS, RLAS) are often not administered at the time of trauma (physician may be more concerned about whether there has been a neck fracture).
- Inconsistent documentation by first responders.
- No standardized follow-up of people involved in MVAs.

A FEW REFERENCES

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ALSO CHECK OUT THESE WEBSITES

Brainline.org CONCUSSION/MTBI

Pub Med Health: [CONCUSSION..www.ncbi.nlm.nih.gov/pubmedhealth/PMH00001802/](https://pubmedhealth.ncbi.nlm.nih.gov/pubmedhealth/PMH00001802/)

MayoClinic.com. CONCUSSION www.mayoclinic.com/health/concussionHEALTH/DS00320

Mild Head Injury and Posttraumatic Headache.

[www. neuropsychologycentral.com](http://www.neuropsychologycentral.com)

Whiplash: A Medical Doctor's Review of the Literature. Headaches/ Neckaches from "Whiplash Injuries". Hass David C. Hosted by SUNY Upstate Medical University.

ONTARIO NEUROTRAUMA FOUNDATION—www.ont.org

It is recommended that the reader review the website of the Ontario Neurotrauma Foundation. There are many important linked and related documents.

However, it is essential to review the 156 page document entitled “**Guidelines for Mild Traumatic Brain Injury (MTBI) Treatment and Persistent Symptoms**”

The Guidelines Development Team, included researchers and clinicians from across Ontario and Quebec. Published in 2011 (and soon to be updated), the Ontario Neurotrauma Foundation (ONF) initiated this project to create a set of guidelines that could be used by healthcare professionals to implement evidence-based, best practice care of individuals who incur a MTBI and experience persistent symptoms. It is interesting to note that one of the major concerns of the Committee was related to treatment and management of the person who does not experience spontaneous recovery. Clinical questions included—Can a management plan be developed to screen for and identify patients that are at high risk for persisting symptoms and once identified, can a management plan be developed to treat the symptoms commonly associated with the disorder?

The Guidelines (to name but a few) include sections on Diagnosis/ Assessment of MTBI, Post-traumatic headaches, Persistent Balance Disorders, Persistent Vestibular Disorders, Persistent Mental Health Concerns and Persistent Cognitive Difficulties.

The Guidelines will continue to be updated but remains an essential tool for all clinicians treating these complex patients.

DEVELOPING A VOCABULARY TO TALK TO YOUR PATIENT ABOUT PAIN (And Their Feelings) GLORIA GILBERT, PT, MSC

The editor has discussed this subject in previous editions of the Academy newsletter. However, given that we are trying to make a distinction between chronic pain and chronic post-traumatic

pain symptoms, some emphasis and clarification may be helpful.

It is timely to remember that the definition of pain (IASP, Merskey and Bogduk, 1994) is subjective, an experience that can be described in many ways. So let us use that definition to assist us in better delineating our patients concerns.

1. Discuss the word PAIN with your patient— remind them that you cannot feel their pain or experience their problems the way they do, so after a brief introduction, you want to encourage them to SEPARATE the different sensations that they are feeling.
2. Can you give me some PHYSICAL WORDS to describe WHAT you are feeling? Aching, burning, stabbing, shooting, cramping, knife-like, hot, cold, vice-like, throbbing, etc.
3. Can you now tell me HOW that pain (sensation) makes you FEEL? Anxious, worried, angry, depressed, overwhelmed, frightened, etc.
4. Are there ANY OTHER WORDS you can use to describe HOW or WHAT you are feeling? Dizzy, nauseous, headachy, light-headed, off balance, wobbly, clumsy, etc.
5. Consider asking your patient questions that will provide additional ‘functional’ information.
 - Do you have trouble reading, i.e. are you reading as much as you did before the accident?
 - Does computer work or watching television bother you?
 - Do loud noises, light, or movement of people around you make you uncomfortable? i.e. Are you avoiding going to the grocery store?
 - Do you find yourself tilting/listing to one side when you walk? Are you walking into walls or door-frames?

The primary health provider may therefore have to ask the question—Can he/she provide treatment/management for those symptoms? Should the patient be referred to another health provider for further assessment of problems? Would additional diagnostic tests be helpful?

BOOK REVIEWS

1. GrrrOUGH

This useful book was reviewed in a past edition of the newsletter. It is worth repeating that although this book was written for children (GrrrOUGH! Pain is like a Grouchy Bear’), the illustrations and content make it suitable for adults as well. The illustrations are designed to ‘echo emotions’, to trigger discussion and to discuss how to cope with pain. I have used this as an educational tool for clients when English is not the first language—a difficult subject to talk about in any language PAIN.

GrrrOUGH (ISBN 978-1-897508-84-8). Contact General Store Publishing House at 1-800-465-6072 (www.gsph.com) or author Cathryn Morgan, author (and former pain patient)

2. COPING WITH MILD TRAUMATIC BRAIN

INJURY: Diane Roberts Stoler and Barbara Albers Hill. *A Guide to Living with the Challenge Association with Concussion/ Brain Injury.* Avery, a member of Penguin Press, ISBN.978-0-89529-791-4

This comprehensive book written by an educator who sustained a head injury (post cerebral bleed) is a great resource for both clinician and patient. I am surprised that I only came across this book recently—it was first published in 1998.

Dr. Stoler has worked with experts in the field to provide a comprehensive and yet subjective appreciation of the effects of Traumatic (?Mild) Brain Injury including Physical Aspects (fatigue, headaches dizziness, vision, hearing, sensory and metabolic disturbances, muscular and motor problems, sexuality, seizure); Mental Aspects (attention and concentration, memory, reasoning, planning and understanding, speech and language etc.); Emotional Aspects (mood and behavior, psychiatric disorders, grieving etc.) and Recovering (rehabilitation, financial issues, living with someone with a MTBI etc.)

The book is available on Dr Roberts- Stoler website. I have otherwise been able to find several ‘used copies of this book. Although Dr Stoler wrote this is 1998, it remain pertinent to our understanding today of TBI. In fact perhaps it is time to integrate information from all health sources (imaging, speech and language, psychology physiotherapy, occupational therapy, neurology etc.) to really begin to really appreciate the CLINICAL presentation of a patient with MTBI.



ASSESSMENT TOOLS IN PAIN MANAGEMENT

DR. HOWARD VERNON, DC, PHD; hvernon@cmcc.ca

Instrument	Attribute	Scoring
Visual Analogue Scale	Pain intensity	0-10, 0-100
Verbal Rating Scale	Pain intensity	0-5, 0-10, 0-100
Pain Diagram	Pain location	Qualitative evaluation for area and consistency Quantitative scoring for body area with body grid
McGill Pain Questionnaire	Pain qualities	(1) <i>pain rating index</i> , based on two types of numerical values that can be assigned to each word descriptor (2) <i>number of words chosen</i> = 0-78 (3) <i>present pain intensity</i> based on a 1-5 intensity scale.
Oswestry Low Back Pain Disability Index	Low back pain disability	0-50
Roland-Morris LBP Scale	Low back pain disability	0-24
Pain Disability Index	Generic pain-related disability	0-70
Disability Rating Index	Generic pain-related disability	0-50
Patient-Specific Functional Scale	Generic pain-related disability	0-30
Neck Disability Index	Neck pain-related disability	0-50
Whiplash Disability Questionnaire	Whiplash-related disability	0-130
DASH	Upper limb-related disability	Disability/symptoms section = 30-150 Sport/Music or Work section = 4-20
SPADI	Shoulder-related disability	Pain = 0-50 Disability = 0-80
Lower Extremity Functional Scale	Leg-related pain and disability	0-80
SF-36	Quality of life	Eight scales = 2 composite scores: physical health and mental health 0-100 for each (on-line scoring)
SF-12	Quality of Life	0-100 for each score (on-line scoring)
Tampa Scale for Kinesiophobia	Fear avoidance beliefs	17-68
Fear Avoidance Beliefs Questionnaire	Fear avoidance beliefs	Physical scale: 0-24 Work scale: 0-42
Waddell Non-organic Signs	Non-organic signs and symptoms	Presence or absence of sign/symptom = 0-9
Cervical non-organic simulation signs	Neck pain-related simulation signs	Presence or absence of sign/symptom = 0-4
Beck Depression Scale	Depression	0-63
Zung Self-rating Depression Scale	Depression	20-80
Zung Self-rating Anxiety Scale	Anxiety	20-80

ABOUT OUR MEMBERS

DR. KEVIN ROD



On May 24, 2013, the Honourable Jason Kenney, Minister of Citizenship, Immigration and Multiculturalism presented Dr. Kevin Rod with a congratulatory certificate. Dr. Rod was recently selected by the University of Toronto's Department of Family and Community Medicine (DFCM) as the recipient of the prestigious Excellence

for New Teachers Award for 2012-2013. Dr. Rod is also a leader in the Iranian Canadian community.

Congratulations from the CAPM as well Kevin!

CAPM COURSE & AGM—SUNDAY SEPTEMBER 8

The CAPM is holding its next Course Day on Sunday September 8, 2013, along with its annual AGM, in Hamilton at the HGH Auditorium. All members are invited to attend the AGM, complimentary. The course will be fee-based, however CAPM members will be provided with a discounted fee to attend the course. The program is being finalized and will be distributed to all members and other groups and posted on the website once ready. Topics such as:

1. **Weaning patients from strong opioids;**
2. **Peer review in "out of hospital" practice pain clinics;**
3. **Relaxation and stress control;**
4. **Pain in the Elderly**

And other hot topics will be the focus of the day. **Watch for more information as it becomes available.**

POST TRAUMATIC STRESS DISORDER: OVERVIEW OF DIAGNOSIS AND TREATMENT—First article in a series on PTSD

DR. ELENI HAPIDOU, PHD, PSYCH, PSYCHOLOGIST

Post Traumatic Stress Disorder (PTSD) affects approximately 1 in 10 Canadians (Canadian Mental Health Association, 2009). It is triggered by exposure to a traumatic event and is classified as an anxiety disorder in the DSM-IV-TR (American Psychiatric Association (APA), 2000). PTSD develops in about 25% of those exposed to a traumatic stressor (Green, 1994).

PTSD has been known by different names over the years. The DSM-I, published in 1952, included a diagnosis of "gross stress reaction" among soldiers in combat with no prior history of a mental disorder. This diagnosis was removed from the DSM-II but was included in the DSM-III, published in 1980 (Scott, 1990). PTSD had also been referred to as "war neurosis," "combat fatigue" and "shell shock". However, these descriptions were associated with "weakness, cowardice, and malingering" in soldiers who were supposedly trying to avoid doing their duty in war (Scott, 1990).

Several specific criteria are required for the diagnosis of PTSD, including the occurrence of a traumatic precipitating event (APA, 2000). PTSD is often associated with war veterans in the media, but numerous different traumatic events such as natural disasters, child abuse, rape, or a serious accident can cause PTSD. A diagnosis of a life threatening illness, such as HIV/AIDS can also be traumatic enough to generate PTSD symptomatology due to anxiety about the future, social stigma, and losses which can be caused by the disease (Poole, Matheson, & Cox, 2007). However, in these cases it is more commonly developed among those with a history of PTSD preceding their HIV/AIDS diagnosis (Poole et al., 2007).

SYMPTOMS OF PTSD

Common symptoms of PTSD and diagnostic criteria for the disorder are re-experience of the trauma, avoidance behaviors in relation to reminders of the event, and a state of hyper-arousal. A person with PTSD will likely re-experience the traumatic event in nightmares, flashbacks, or hallucinations (APA, 2000). Individuals may also become agitated with stimuli or situations associated with or remind him/her of the event, and as such, will avoid these types of stimuli. A person suffering from PTSD may also have memory loss involving the traumatic event (APA, 2000). Additionally, s/he may also have forms of hyper-arousal in which s/he may be easily irritated, have difficulty sleeping, or trouble concentrating. Finally, the disorder will present for a period of longer than one month and will likely cause significant disruptions to social, occupational, and other important areas of one's life (APA, 2000).

Co-morbid conditions such as substance abuse, generalized anxiety or specific phobias, and depression may develop along with PTSD (Karatzias & Chouliara, 2009). Boscarino (1997) found that PTSD is also associated with poor physical health and medical problems including circulatory, digestive, musculo-skeletal, metabolic, and nervous-system disorders. After controlling for smoking, age, and substance abuse, the cause of many illnesses was attributed to severe stress exposure and the long-term effects of physiological arousal. Karatzias and Chouliara (2008) attempted to explain the role of cognitive appraisals in PTSD symptoms and their relation to poor physical health. In response to a traumatic event, PTSD symptoms may lead to negative cognitive appraisals about the body or PTSD symptoms. Negative appraisals may involve thoughts about what happened to the body during trauma, as well as negative appraisals regarding the PTSD symptoms the individual is experiencing. When these cognitive appraisals are linked with negative emotional responses such as despair or frustration, this may lead to physiological arousal as well as negative health behaviors. Without treatment, these behaviors will eventually lead to poor physical health (Karatzias & Chouliara, 2008).

DIFFERENTIAL DIAGNOSIS

Adjustment disorders, acute stress disorders, obsessive-compulsive disorders and malingering PTSD may share a lot of elements with PTSD (APA, 2000). Prevention of misdiagnosis is best done through proper adherence to identifying diagnostic criteria. This occurs with a thorough interview and comprehensive psychological evaluation.

POST TRAUMATIC STRESS DISORDER: OVERVIEW OF DIAGNOSIS AND TREATMENT

Continued

When ruling out adjustment disorder, the clinician must look at the severity of the event. In order to be diagnosed with PTSD, the patient must have undergone or witnessed a traumatic event of extreme, life-threatening nature (APA, 2000). With adjustment disorder, however, the event could be of any severity; not necessarily life-threatening (APA, 2000). Mental illness following a serious automobile accident, for instance, may be diagnosed as PTSD. A similar illness following a divorce, on the other hand, would most likely be an adjustment disorder.

Acute stress disorder (ASD) patients also exhibit similar signs and symptoms as individuals with PTSD. ASD, however, manifests within four weeks of the traumatic event and resolves itself within that time as well (APA, 2000). If symptoms carry on for longer the diagnosis is changed from ASD to PTSD (APA, 2000).

Obsessive-compulsive disorder is ruled out simply through the identification of a traumatic event. OCD does not develop in response to an event (APA, 2000). One of the most difficult tasks of a mental health diagnostician is to distinguish malingering from mental illness; PTSD is no exception to this. Patients often have much to gain through being diagnosed with PTSD, such as pardon or reduced sentence from the courts or financial compensation from their employers (Knoll & Resnick, 2006).

DIAGNOSTIC CRITERIA

The diagnostic criteria for PTSD are very specific and with many conditions. First, the individual experiencing the mental disturbance must have been a victim or have witnessed a traumatic or life threatening event (APA, 2000). PTSD is unique in this aspect as there are no other disorders in the DSM which specify the occurrence of a traumatic event in order for the condition to be diagnosed. As mentioned earlier, a PTSD sufferer will likely re-experience the trauma, have avoidance behaviors in relation to reminders of the event, and experience hyper-arousal. Fear, helplessness or horror should also be expressed in reaction to the event (APA, 2000).

Assessment methods

Two self-reported patient questionnaires often used in psychological evaluations include the Symptoms Check List (SCL-90) and the Impact of Events Scale (IES). The SCL-90 is a 90-question assessment developed by Derogatis, Lipman, & Covy (1974) designed to look at nine categories of symptoms: somatization, obsessive-compulsive, interpersonal sensitivity, depression, anxiety, hostility, phobic anxiety, paranoid ideation, and psychoticism (Holi, 2003). Patients are asked to rate the applicability of each statement to themselves on the questionnaire using a 0 ("not at all") to 4 (extremely) scale (Derogatis et al., 1974). The questionnaire can be given to anyone with a 6th grade reading level (Holi, 2003). The SCL-90 serves many purposes. It helps with the initial evaluation of patients at the first consult to objectively measure self-perceived symptom assessment. Additionally, it can serve to measuring patient progress during and after treatment to monitor change. The SCL-90 can also be used in clinical trials to measure change in symptoms as well, especially in cases of depression and anxiety (Holi, 2003).

The IES was developed by Horowitz, Wilner, and Alvarez in 1979, and gives a subjective response to a traumatic event as perceived by the patient (Weiss & Marmar, 1996). The exact same 0 to 4 scale from the SCL-90 is used in the IES as well, as patients judge the relevance of each of the 22 questions (Weiss & Marmar, 1996). This can aid the clinician in distinguishing between PTSD and adjustment disorder as allows the severity and extreme nature of the event to be determined.

Interview Model: CAPS-1

The Clinician Assessment of PTSD Scale (CAPS-1) was developed by Blake in 1990 as a model used to determine frequency and intensity of symptoms. CAPS-1 looks at signs and symptoms relating to 30 different areas, ranging from recollections of the traumatic event to homicidality and guilt (Blake et al., 1995). CAPS-1 is considered to be the most valid and reliable in comparison to several other PTSD psychiatric interview models, as demonstrated in a review by Blake et al. (1995). A "report card" of sorts grading each interview model to a proposed set of standards has ranked CAPS-1 the highest with near straight A's for each standard. It should be noted that a concern with regards to this scoring is the potential for bias, as the author of this comparative review is the same individual who helped develop the CAPS-1 model (Blake et al., 1995).

ETIOLOGY OF PTSD

As mentioned previously, a traumatic event is the main criteria for the future development of PTSD. However, even among those who suffer severe traumatizing events, the development of PTSD is seen in a minority of those individuals. Usual estimates of the PTSD incidence ranges from 5-35% of individuals exposed to a traumatic event (McKeever & Huff, 2003). As a result, it is not likely that experiencing trauma is the only causal factor in development of PTSD. A number of other factors have also been found to influence PTSD development. McKeever and Huff (2003) proposed a diathesis-stress model in order to delineate how pre-morbid risk factors and situational stressors may influence the development of PTSD in reaction to a traumatic event. Prior research has identified several variables which have been found significant in PTSD such as pre-morbid personality characteristics, childhood familial environments, social support, demographics, patterns of psychophysiological stress responses, and severity of trauma (McKeever & Huff, 2003). Additionally, it has been found that a previous exposure to trauma also increases the risk of developing PTSD after successional traumas (Breslau, Chilcoat, Kessler, & Davis, 1999). An interaction of these factors may predispose an individual to develop PTSD.

POST TRAUMATIC STRESS DISORDER: OVERVIEW OF DIAGNOSIS AND TREATMENT

Continued

A meta-analysis study conducted by Brewin, Andrews and Valentine (2000) found that some variables are more influential in the development of PTSD than others. Variables which influence responses during and after the trauma have the strongest effect on whether an individual will develop PTSD compared to pretrauma variables. However, retrospective reporting may bias how those with PTSD report pretrauma variables. The severity of the trauma is important as more severe traumas increase the likelihood that an individual will develop PTSD. A lack of social support and additional life stress after the trauma also are strong predictors of PTSD development (Brewin et al. 2000). Females have also been found to have a higher risk of developing PTSD in comparison to males. Overall, the occurrence of a traumatic event is the main causal factor in PTSD, but many other variables interact to cause the development of PTSD.

Military Combat

The risk of developing PTSD directly correlates with the level of war-related stress that the patient has been exposed to (Friedman, Schnurr, & McDonagh-Coyle, 1994). War-related stress includes battlefield activity, living as a prisoner of war, or combat stress reaction (formerly referred to as "shell shock") (Friedman et al., 1994). The risk of developing PTSD is also highly associated with pre-military and post-military factors. Pre-military factors include one's upbringing and childhood abuse, socioeconomic status, family history of PTSD, educational level, and personality characteristics (Friedman et al., 1994). Post-military factors include social support networks and coping abilities (Friedman et al., 1994). Several co-morbidities associated with PTSD can also occur such as depression, anxiety, substance abuse, and alcoholism, which are often also associated with social and marital problems (Friedman et al., 1994).

Specific populations at risk for further psychological damage include women who have experienced sexual harassment, minorities who have experienced racism, and individuals who have become handicapped as a result of war (Friedman et al., 1994). In this latter group, a downward spiral is often seen as exacerbation of the person's condition often makes PTSD last longer, which makes the experience of the injury perceived as worse. With that said, recovery can vary from having a full recovery to suffering from chronic or intermittent PTSD (Friedman et al., 1994).

Childhood Trauma

It is also possible to develop PTSD as a child, but some of the symptoms present slightly differently as compared to adults. Children may engage in repetitive play as a form of re-experiencing the trauma (APA, 2000). They may also experience generalized nightmares which do not necessarily represent the trauma (APA, 2000). Furthermore, the effect of trauma in childhood has been found to be a variable in the development of many other psychiatric disorders aside from PTSD. It should be noted that trauma in childhood as compared to trauma later in life poses no greater risk of developing PTSD (Breslau et al., 1999). However, childhood trauma increases the risk that PTSD will develop after a new trauma is experienced as an adult (Breslau et al., 1999).

Even though post-trauma events have a stronger influence on the development of PTSD than pre-trauma events, childhood abuse has been deemed an important factor in the development of PTSD. There is much evidence of childhood sexual or physical abuse contributing to the development of PTSD in adults. For example, it was found that women who had experienced childhood physical abuse were nearly five times as likely to have also experienced PTSD at some point in their lives (Duncan, Saunders, Kilpatrick, Hanson, & Resnick, 1996). McFarlane suggested that "A history of abuse predisposes an individual not only to placement in more traumatic situations, but also to the maladaptive cognitive patterns theoretically associated with the development of PTSD" (1990, p. 10). Maladaptive cognitive patterns developing as a result of child abuse can also contribute to the development of PTSD in adulthood. Abuse experienced by children is often seen as uncontrollable and as a result they may eventually develop a learned helplessness cognitive pattern. This pattern has been associated with the development of PTSD (McKeever & Huff, 2003).

Shame and anger directly resulting from childhood abuse have also been found to be associated with development of PTSD as an adult. Previous research has shown that shame relates childhood abuse to adult psychopathology such as bulimia nervosa and depression (Andrews, 1995, 1997). Andrews, Brewin, Rose, and Kirk (2000) investigated the role of shame and anger and their role in linking childhood abuse and PTSD. Individuals who had recently experienced a violent crime discussed the crime, but also their abusive experiences before the age of seventeen. It was found that both shame and anger stemming from childhood abuse contributed to PTSD symptomatology. However, shame seemed to be the more dominant feeling in the long term and contributed to the future course of PTSD (Andrews, et al., 2000). Again, a significant association between childhood abuse and PTSD symptoms was found. Though it has been found that post-trauma events and stressors have a strong effect on the development of PTSD, childhood abuse as a pre-trauma stressor can have significant effects on the development of PTSD as an adult.

TREATMENT OPTIONS

Types of Therapies

There are two different types of therapies available to people suffering with PTSD: psychotherapies or pharmacotherapies.

Evidence for psychotherapies

Psychotherapies include cognitive behavioural therapy (CBT), eye movement desensitization and reprocessing (EMDR) and relaxation techniques. CBT is the most common method of treating PTSD and is composed of both cognitive techniques (CT) that involve cognitive restructuring and behavioural techniques such as prolonged exposure therapy (ET) (Mendes, Mello, Ventura, Passarella, & Mari, 2008).

POST TRAUMATIC STRESS DISORDER: OVERVIEW OF DIAGNOSIS AND TREATMENT

Continued

CT is based on the idea that irrational thinking plays a role in generating anxiety and affective problems (Beck, Emery, & Greenberg, 1985). For the treatment of PTSD, CT involves cognitive restructuring which aims to reduce symptoms by helping patients identify and challenge negative dysfunctional thoughts and beliefs related to the traumatic event (i.e. overestimating danger) and replace them with functional, realistic cognitions (Mendes et al., 2008). This often involves the patient actively gathering and comparing logical evidence for and against their negative thoughts, which leads to reduced emotional distress (Livanou, 2001).

ET involves inducing PTSD symptoms through repeated exposure to symptoms-evoking objects or situation (imaginary or real) until the symptoms become gradually reduced through habituation (Mendes et al., 2008). This technique claims to enable the organization of traumatic memories which consequently reduces anxiety and other symptoms. It is usually performed either by asking the individual to repeatedly listen to a detailed present-tense account of the traumatic event, or physically exposing subjects to specific cues associated with the traumatic event (i.e. re-exposure to car travel follow a car accident) (Livanou, 2001). The level of distress of the patient during these sessions is continuously monitored.

EMDR was first introduced by Shapiro (1989) in a PTSD study where patients were asked to focus on traumatic images or memories while following a therapist's index finger moving quickly across their visual field. It seemed to reduce distress in patients after repeated exposures. While the repeated lateral eye movements were originally thought to be critical for this treatment, current research claims that other bilateral rhythmical cues (i.e. handtaps, auditory signals) may be just as effective (Shapiro, 1994). Shapiro (1989) originally claimed that one session of EMDR was sufficient to yield significant improvements in PTSD symptoms, which has been responded with much controversy. In addition, its effectiveness has been questioned due to the lack of controlled studies with high quality methodology (Livanou, 2001).

In a systematic Cochrane Review, Bisson and Andrew (2007) evaluated the efficacy of psychotherapy techniques. Cochrane Reviews is a regularly updated collection of evidence-based medicine databases, and this one was last updated in 2007. They reviewed 33 RCTs, completed before October 2004, on the effectiveness of trauma-focused cognitive behavioral therapy (TFCBT), EMDR, stress management, group TFCBT, and other therapies (supportive therapy, non-directive counseling, psychodynamic therapy, hypnotherapy) in the treatment of PTSD in comparison to waitlist/usual care controls. The authors concluded that TFCBT, SM, group TFCBT, and EMDR were more effective than wait-list and other therapies and that there was no significant difference between TFCBT, SM, and EMDR. However, these results must be interpreted with caution as there was significantly large unexplained heterogeneity and potential publication bias in this study (Appendix J).

In another systematic review by Shepherd, Stein, & Milne (2000), 16 RCTs were reviewed in order to compare EMDR and other psychotherapies. The results of this review indicated that for most of the studies, EMDR was effective in reducing symptoms up to 3 months post-treatment. Some studies found it to be superior to exposure therapies and relaxation techniques as well as wait-list controls. Interestingly, two studies concluded that treatment involving eye movement was more effective than those without, while three studies found no difference in effectiveness. However, it is important to keep in mind that although this study is in the form of a systematic review, which is at the top of the hierarchy of evidence, the studies that they reviewed were small in sample size and of limited methodological quality.

While EMDR has been found to be effective over no treatment controls, it may be that elements of EMDR that overlap with CT and ET account for this efficacy. Focusing on traumatic memories is, on its own, a form of ET. As mentioned in Shepherd et al.'s review, it is uncertain whether the lateral eye movements or other bilateral stimulations involved in EMDR add any new therapeutic effects to the process (Shepherd et al., 2000). Furthermore, the EMDR studies reviewed by Bisson and Andrew (2007) were of small sample size and the number of EMDR sessions varied between studies.

Mendes et al. (2008) evaluated 23 clinical trials from 1980 to 2005 and compared the effectiveness of CBT and other psychotherapies of PTSD. They found remission to be more significant in those treated with CBT than EMDR ($p = 0.01$) or supportive therapies ($p = 0.002$). However, CBT was comparable to ET ($p = 0.64$) and CT ($p = 0.98$) alone with regards to efficacy and compliance.

Recent literature suggests that CBT, ET and CT are all effective psychotherapeutic treatments for PTSD, with no difference in efficacy detected. There is substantial support for these types of therapies, proclaimed by many studies of high methodological quality (Livanou, 2001). On the other hand, it is uncertain whether EMDR is an effective method of treatment for PTSD as studies that have examined it vary in methodology and have relatively small sample sizes. More research is required to be conducted in the future, with larger sample sizes and longer follow-up periods before any conclusions can be made with certainty.

Future research should address potential differences in treatment between genders, age groups, severity of symptoms, and presence of co-morbidities. In addition, studies should continue to control the amount of bias present in their studies and the studies they review.

Evidence for pharmacotherapies

In addition to psychological symptoms, PTSD is characterized by specific psychobiological imbalances as well. This provides reasoning for the use of pharmacological interventions for restoring balance and treating the disorder.

POST TRAUMATIC STRESS DISORDER: OVERVIEW OF DIAGNOSIS AND TREATMENT

Continued

Earlier pharmacotherapies for PTSD were antidepressants, such as tricyclic antidepressants (TCAs) and monoamine oxidase inhibitors (MAOIs). Their efficacy has been investigated in relatively few studies with good methodology, yielding inconsistent results with respect to reducing symptoms of PTSD. In addition, these classes of drugs have also been associated with adverse side effects including cardiovascular complications, and other safety concerns, such as an ease of overdosing (Asnis, Kohn, Henderson, & Brown, 2004).

Stein et al. conducted a Cochrane Review in 2006 that evaluated the effectiveness of pharmacological therapies in treating PTSD. They concluded that pharmacotherapies can be considered effective, with SSRIs being the best option. Similar to CBTs in psychotherapies, SSRI trials make up most of the evidence in this treatment group and have been conducted with large sample sizes, longer follow-up time, and better overall methodology, including double-blinded placebo-controlled trials (Asnis et al., 2004). Thus, it is not surprising that SSRIs are considered first-line medications in treating PTSD. Currently, the SSRIs that have been approved by the US Food and Drug Association for treatment of PTSD are sertraline and paroxetine (Asnis et al. 2004). Interestingly, this systematic review and associated meta-analysis also found that war veterans are more resistant to pharmacotherapy than other patient groups with respect to symptom severity. It would be interesting to investigate in future studies whether various subgroups of patients with PTSD respond differently to treatment methods. Some characteristics that would be interesting to look at are gender, age, severity of PTSD, and cause of PTSD.

The National Institute of Clinical Evidence (NICE) recommends that trauma-focused psychological treatment should be considered first-line treatment over pharmacological therapy (NHS, 2005). With any study, it is important to look for methodological limitations such as lack of a control group, lack of clear descriptions of inclusion criteria, and small sample size. In addition, variations between studies in methodology make it difficult for systematic reviews to combine results in an accurate manner. The NICE recommend that trials of adequate power and high methodology be conducted in comparing the efficacy and cost-effectiveness between trauma-focused psychotherapy and pharmacological therapy (NHS, 2005).

Recently Identified Side Effect of SSRIs

In terms of SSRI use, whether in PTSD or other conditions, there are a number of recently and still largely unknown side effects that have emerged. Many practitioners will be aware of the need to taper SSRIs and SNRIs because abrupt withdrawal can lead to a variety of unpleasant and even painful side effects (1). Symptoms include headache, dizziness and a feeling like lightning in the head. Nausea and vomiting also occur. It occurs within a week of discontinuing the antidepressant.

The serotonin syndrome with features of increased sympathetic nervous system features (increased blood pressure and heart rate), sweating and central nervous system involvement (confusion, stroke) can occur (2). In particular, increased dose or interaction with drugs also affecting the release of serotonin (e.g. meperidine) can also precipitate this potentially life-threatening syndrome.

Recently, there have been 200 case reports of SSRIs causing violent behaviour with even small amounts of alcohol. Fifty per cent of the patients involved in these incidents have no memory for the events (3). This is not addressed in the product monograph. It is of interest that these classes of drugs can be responsible for both suicidal and homicidal behaviours without alcohol but it is largely unknown that 90% of school shootings are associated with the use of these drugs or other mood altering drugs (4).

There are also a number of case reports of alcoholism and other compulsive behaviours associated with the use of these drugs (5). Finally, there have been a number of case reports of these classes of drugs causing tardive dyskinesia (6).

The reason for updating readers on these issues is not to discourage the use of these drugs but to alert prescribers to be aware of the potential for these largely unknown adverse effects and to monitor their patients more closely.

CONCLUSION

Recently, PTSD has been extensively covered in the media with regards to soldiers returning from wars such as the one in Afghanistan. Due to its prominence in North American culture it is important to understand the disease with regards to its definition, diagnosis, and treatment. By understanding the diagnostic criteria as well as being able to recognize PTSD in the midst of other anxiety disorders and psychiatric illnesses, clinicians will be able to better improve their practices. This improvement will bring about a more deep and comprehensive understanding of the patient, and enhance the clinician-patient relationship. Such a fostered relationship will ultimately lead to effective treatment decisions and optimal patient outcomes.

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IN SUBSEQUENT EDITIONS OF OUR CAPM NEWSLETTER, WE WILL DISCUSS PTSD AND CHRONIC PAIN

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CENTRAL PAIN MECHANISMS UNDERLIE MANY CLINICAL PAIN SYNDROMES

DR. ELDON TUNKS, MD, FRCPC

There has been a tendency to regard chronic pain symptoms with suspicion, especially those involving widespread pain, symptoms not conforming to neurological explanation, or with prolonged disability. Often findings on the physical examination, such as "Waddell's signs", have been cited as evidence that the pain problem is imaginary or even deliberately exaggerated or faked. As studies of neurobiology of central pain mechanisms have become more sophisticated, however, new evidence is being brought to light that the nervous system can change the way that pain symptoms and signs appear, sometimes in ways that previously were regarded non-neurological and non-organic.

Consider for example the example of a man in his late 70s who had had 5 failed lumbar surgeries for low back and legs pain, who subsequently ended up on morphine SR that initially helped his pain for a few years, but eventually pain breaking through, leading to further increases of opioids with temporary relief after each increase, and finally he came in to clinic taking approximately 1.6 g of sustained release morphine per day, complaining of pain from head to foot, unable to stand upright, unable to sleep, depressed and with total body tenderness and pain. Eventually the pain clinic doctor reduced his opioids to 20% of what they had been on referral, and miraculously his pain management was much better, he could sleep, still had signs of nerve root fibrosis, pain still in low back but less widespread, and depression much improved.

In an influential seminal review, Angst and Clark (2006) noted that in animal literature some mechanisms of hyperalgesia are linked to the mu-opioid receptor for analgesia that is subsequently followed by an hyperalgesic phase. The Kappa opioid receptor may be more prone to induce analgesic or hyperalgesic phase. A somewhat contradictory mechanism also identified was the capacity of a very small mu agonist effect to induce hyperalgesia, followed by analgesia as the mu agonist effect was increased. The mechanisms may include sensitization of peripheral receptors, opening calcium channels on astrocytes in the white matter of the spinal cord releasing glutamate and other transmitters which recruit NMDA receptors and lower pain threshold. Also mesencephalic pathways efferent to the spinal cord might be recruited to increase pain transmission. In humans, some parallel evidence came from opioid-induced hyperalgesia in con-

text of maintenance opioid therapy, or withdrawal syndrome provoked by opioid abstinence. Patient data included aggravation of postoperative pain with acute or chronic opioid administration, and increased sensitivity to experimental pain in methadone maintenance.

Clinically it may be difficult to distinguish between tolerance versus opioid-induced hyperalgesia. The difference lies in the hyperalgesia gradually getting worse as opioid is increased, accompanied by diffuse tenderness, allodynia, widespread hyperalgesia, increased pain despite increased opioid.

A more recent review (Wolf, 2011) amplified these observations with characteristics of central sensitization. Perceived pain reflects increased excitability of central nociceptive circuits. These increases in synaptic function may be triggered and maintained by nociceptive input. Increased sensitivity of the pain system results in innocuous inputs being amplified into painful sensations, and pain may become prolonged and spread widely within and beyond the original nociceptive location. Peripheral nociceptors and mechanoreceptors connect anatomically to CNS. The peripheral nociceptors can become sensitized to noxious stimuli within the area of injury (primary hyperalgesia), and also post-synaptic potentiation of peripheral neurons and dorsal horn neuron windup may occur in response to repeated noxious stimuli, and long-term synaptic potentiation in the CNS through synaptic plasticity. For most central circuits, synaptic input is sub-threshold or restrained by inhibitory input or by state of low membrane excitability, but activity-dependent changes in microglia, astrocytes, gap junctions, membrane excitability, gene transcription, all might amplify pain intensity, duration, and spatial extent. For example, local injection of capsaicin results in a small area of primary hyperalgesia followed by longer-lasting and more widespread secondary hyperalgesia with tactile allodynia and pinprick hyperalgesia, and this secondary hyperalgesia requires CNS input and activated TRPV1 receptors (transient receptor channel receptors). At the level of the skin, interactions between nociceptive and low threshold fibers mediate some hyperalgesic responses. Note intradermal capsaicin which activates TRPV1 receptors can induce contralateral hyperalgesia and allodynia. Other studies have shown that central sensitization can be accompanied by changes in reflexes, excitability at the brain cortex and circulatory

changes in the cortex and brainstem.

Studies in humans functional imaging (fMRI) reviewed by Apkarian et al 2011, noted that anticipation and early detection of the pain stimulus engages the nucleus accumbens, mid-cingulate and anterior insula, and pain relief engages the brainstem especially perioperative gray, and the nucleus accumbens calculates the reward value of pain relief after painful stimulus cessation. High-intensity chronic back pain engages the medial prefrontal cortex extending to the anterior cingulate, while experimentally applied thermal pain did not engage these areas. When chronic back pain patients were given an experimental thermal stimulus, the nucleus accumbens signaled loss of reward and the chronic back pain patients reported that during the stimulus their own spontaneous low back pain was diminished. Mapping brain activity across multiple chronic pain conditions reveals different networks for post-herpetic neuralgia, osteoarthritis, or chronic pelvic pain. During resting state, brain activity in chronic pain is distorted with higher frequencies in the insula and anterior cingulate in chronic pain patients. The magnitude of activity in spontaneous chronic low back pain and post-herpetic neuralgia was greater in the amygdala and prefrontal cortex – areas typically associated with mood.

EXAMPLES OF CLINICAL SIGNIFICANCE OF THESE CENTRAL SENSITIZATION MECHANISMS (REVIEWED BY WOLF, 2011)

Sustained nociceptor stimulation of myofascial trigger points can induce more widespread central sensitization, and topical cutaneous capsaicin can increase myofascial trigger point sensitivity in the same segment, explaining why patient subject to myofascial pain or fibromyalgia might experience aggravation of their widespread pains after a painful event.

Nociceptive stimuli from viscera also can induce central sensitization resulting in hypersensitivity the upper esophagus and allodynia in the chest wall – for example reflux disease and esophageal motility problem mimicking heart attack. Furthermore sensitization of the esophagus can increase thermal and mechanical pain sensitivity in the rectum. There are cases in which patients with abdominal pain and proctalgia experience relief of their abdominal pain and rectal pain with an infusion of local lidocaine to the rectum.

CENTRAL PAIN MECHANISMS UNDERLIE MANY CLINICAL PAIN SYNDROMES

Continued

Central sensitization can underlie many common clinical phenomena such as migration of appendicitis pain to McBurney's point, or referral of angina pectoris from the retrosternal to the left pectoralis minor and then down the left arm.

There is enhanced sensitivity to noxious stimuli at the joints and remote areas in patients with juvenile rheumatoid arthritis, with and without active disease.

In osteoarthritis, pressure pain thresholds at joints in remote areas and increased temporal summation and degree of pain did not correlate with radiological findings, compared to controls. Patients with high preoperative pain and low pain thresholds have higher risk of persistent pain after total knee replacement. Osteoarthritis patients have punctate hyperalgesia in areas of referred pain, associated with brainstem activation demonstrated on fMRI.

Temporomandibular joint disorders is associated with generalized pain sensitivity after isometric contraction of the orofacial muscles. There is widespread bilateral mechanical and thermal pain oversensitivity in women with TMJ dysfunction, compared to controls.

In fibromyalgia patients, there is widespread reduction in thermal and mechanical pain thresholds and laser evoked potentials. In fibromyalgia there is referred pain, temporal summation, muscular hyperalgesia and muscle pain that are attenuated by ketamine. Also in fibromyalgia there is widespread temporal summation of pain which can be maintained by low-frequency stimuli. In twin studies of fibromyalgia there is greater than chance co-occurrence of chronic widespread pain, chronic fatigue, joint pain, irritable bowel syndrome, tension headache, temporomandibular joint disease, panic attacks, PTSD, and depression.

In chronic pain from whiplash there is lowered widespread pain threshold – injection of anesthetic to trigger points increase the range of motion and improve the pain threshold.

In tennis elbow there is widespread bilateral mechanical pain hypersensitivity.

With radiating low back pain with disc herniation there is generalized deep tissue hyperalgesia

outside the neurologically predicted area.

In migraine, cutaneous allodynia also develops in 79% of patients during migraine, including beyond the area of referred pain, and spontaneous body pain and allodynia can proceed migraine attacks. During either migraine or tension headache there is reduced threshold to pressure, pinprick, and reflexes. A

longitudinal prospective study of tension headache found that subjects had normal thresholds before developing the chronic tension headache and the threshold decreased in those eventually developing the chronic tension headache. In cluster headache, the nociceptive flexion reflex threshold was reduced on the symptomatic body side.

In complex regional pain syndrome type I (CRPS I), thermal hyperalgesia on the same side as the diseased limb and contralateral hypersensitivity in the absence of contralateral inflammatory change have been demonstrated, and ketamine reduces CRPS pain.

In chronic pancreatitis there is generalized deep pressure hyperalgesia and a greater degree of widespread secondary hyperalgesia evoked by repetitive experimental stimulation, and that is reduced after splanchnic denervation (celiac block).

In pelvic pain such as chronic prostatitis there is increased pain sensitivity in the perineum, and vulvodynia enhanced responsiveness to capsaicin and secondary hyperalgesia, and vulvodynia is associated more often with fibromyalgia.

WHAT WE CAN LEARN FROM THIS

The central nervous system may respond to persistent and troublesome clinical pain by changes in the way the nervous system functions and this will affect the way the pain is manifest, with patterns of pain which may apparently contradict usual expectations for neurologically consistent findings, and changes occur at multiple levels from the periphery up through the spinal cord and brain. There are central nervous system mechanisms involved in many chronic pain conditions. Central sensitization is normal in that it underlies many common clinical presentations and even some acute pain conditions such as the onset of CRPS. Clinical assessment should include

identification of severe pain that is not apparently predicted by anatomical injury, noting presence of hyperalgesia or allodynia, sensory summation with repetitive stimulation, persistence of pain after stimulation, radiation patterns outside the expected region, associated pain syndromes such as CRPS, phantom limb syndrome, post-stroke thalamic syndrome, and patterns of widespread tenderness and pain. Rather than dismissing these features as nonorganic, it is prudent to look further for central sensitization features that can be addressed with the appropriate methods.

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CRANIOSACRAL THERAPY YONIA (NINA) CHERNICK

CranioSacral Therapy is a manual input which involves facilitating the position of connective tissue using, predominantly, an indirect technique. The name is derived from the significance of the central and autonomic nervous systems and their meningeal wrappers in the overall function of the body. The technique has its roots in Osteopathic Medicine, one of whose main tenets is that structure and function are inter-related.¹ Dr Andrew Taylor Still, a Civil War era physician, developed the practice of Osteopathic Medicine from a crisis of conscience and a desire to find treatment options which were holistic and patient-centred.

This model serves us well when we have the opportunity to work with patients who have suffered traumatic injury, whether to the body, brain or both.

To understand the benefits to patients with soft tissue injury, we can look first to the fascial system of the body. In general, the orientation of fascia is longitudinal, with areas of transversely oriented tissue providing stability throughout the body, particularly at joints. Trauma involves force entering the tissue. This force, if great enough or the point of contact is already trophically challenged, affects the local connective tissues.

CRANIOSACRAL THERAPY

Continued

The collagen and elastin mix of the tissue responds with shortening, thickening and generalized trophic changes over time.

The second component of soft tissue injury requiring equal attention is the Autonomic Nervous System. Autonomic dysfunction or imbalance is evident in many patients post-trauma. Presentations include problems with sleep, anorexia and localized trophic changes. Trophic changes that may present include excessive sweating, erythema and localized oedema.

The medium of CranioSacral Therapy is connective tissue. This means that restriction or limitation in tissue can be addressed throughout the body by accessing the level or layer affected by tension. CranioSacral Therapy stimulates a strong parasympathetic state which contributes to a decrease in overall tone. The intensity of pain, as reported by the patient, decreases. This is immediately helpful as well as facilitative in attaining more of a balanced autonomic response.

Although this form of therapy has proven extremely effective anecdotally, as it fits into the CAM model it does not conform well to standard methods of analysis. The assessment is based on tissue presentation, not symptom-reporting; the focus is to find the primary lesion or cause of the dysfunction. As a therapy for patients who are already experiencing pain, it is excellent, as it facilitates tissue changes which increase comfort and result in optimal functional change.

Case Study: An active 55 year old school teacher presented with Bell's Palsy and chronic left-sided neck and shoulder pain, which had recently flared up. His history included a traumatic bicycle injury sustained at the age of 15, with no follow-up treatment until the presentation of neck and shoulder pain began during adulthood. This patient attended several forms of therapy and was extremely compliant in regards to attendance and self-care, but experienced no symptom change. He was treated using CranioSacral Therapy and had a change in facial innervation after one treatment. This was evidenced by his delight at "having wrinkles on that side of my face again". Treatment continued with a focus on the upper cervical spine as well as at the left thoracic outlet and, posteriorly, at the cervico-thoracic junction. Soft tissue addressed included levator scapula, upper trapezius and scalenes. Neurological focus included the right Facial nerve, Sub-occipital nerve, Spinal Accessory nerve and Dorsalscapular nerve. This patient's facial and oral function have continued to improve. A change in the intensity, character and quality of the chronic neck/shoulder pain has been reported. He has returned to work, sports and activities of daily living.

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NEWSLETTER ARTICLES

With the focus of the Newsletter changing to "topic-driven", the Editor will be reaching out to members requesting specific articles on various topics. At any time you would like to see an article highlighted, and if you have an article to go with that topic, we would be happy to accept it. Please contact the office at 905-404-9545 or CAPMOFFICE@eventsinsync.com.

Thank you.

ATTACHMENTS

Along with this Newsletter there two attachments entitled "**CranioSacral Article, 2013**" and also the "**Appendices for the Post Traumatic Stress Syndrome Article**".