Dealing with pain and Traumatic Brain Injury

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Disclaimer

- I have no relevant financial relationships and do not intend to discuss the off-label/ investigative (unapproved) use of medical products/devices.
- The views, opinions and/or findings contained in this presentation are those of the author.
Traumatic Brain Injury

- Cost: 40 Billion dollars/year In the United States, traumatic brain injury (TBI) is most common cause of death and disability in those < 45 years old
- 1.5 million cases/year – half seen in emergency room or intensive care unit (F. Foot, L. Schwartz, Explore Sept/Oct 2012, 282-290, Vol 8, No. 5)
- Signature injury of Operation Iraqi Freedom/Operation Enduring Freedom - approximately 15% of all deployed troops
- TBI in the USA estimated 180-200 cases/100,000
- Around 600,000 New TBI occur every year- 10% of these injuries are fatal
- NIH survey estimates in USA 1.9 million suffer skull fracture or intracranial injury, ½ have suboptimal outcome.
Causes of TBI (CDC Data)

- Transportation (MVA) 48.9%
- Falls 25.8%
- Firearms 9.7%
- Other Assaults 7.5%
- Others 7.4%
- Unknown 0.6%
Severity Grades of TBI

- **Mild (Grade 1):**
  - altered or LOC < 30 min with normal CT or MRI, GCS 13-15, PTA < 24 hours.

- **Moderate (Grade 2):**
  - LOC < 6 hours with abnormal CT and/or MRI, GCS 9-12, PTA < 7 days.

- **Severe (Grade 3 & 4):**
  - LOC > 6 hours with abnormal CT and/or MRI, GCS < 9, PTA > 7 days.
Frequency of PCS Symptoms following a MTBI

- Poor concentration  71%
- Irritability  66%
- Tired a lot more  64%
- **Depression**  63%
- Memory problems  59%
- **Headaches**  59%
- Anxiety  58%
- Trouble thinking  57%
- Dizziness  52%
- Blurry or double vision  45%
- Sensitivity to bright light  40%
Referrals (Team work)

- Audiologist
- Kinesiotherapist
- Neuro-ophthalmologist
- Occupational therapist
- Recreational therapist
- Speech and language pathologist
- Case manager
- Neurologist
- Neuropsychologist (psychologist)
- Physiatrist
- Psychiatrist
- Social worker (counselor)
- Vocational rehabilitation counselor
Interplay of cognitive and emotional problems

Psychogenic/Psychiatry symptoms
- Denial
- Anger and irritability
- Depression
- Rigid compulsive/hypervigilant
- Emotional lability
- Social withdrawal
- Sense of featurelessness
- Thought disorder
- Personality and conduct disorder

Neurogenic symptoms
- Anasognosia (lack of awareness of impairment)
- Frustration, catastrophic reaction, reduce information
- Lack of initiative, impaired emotional expressiveness (Aprosodias), lower crying threshold, fatigue
- Distractibility, inability to deal with more than one task at a time, dependence on external controls.
- Lability of emotional expressiveness (not the underlying feeling state)
- Lack of initiative
- Impaired planning
- Aphasia, anomia, or confusion
- Impulsivity, social disinhibition
Challenges in TBI

- No two mild TBIs are the same
- Co-morbidity common – post-traumatic stress disorder, depression, sleep problems, headaches, mood problems
- Pain conditions common
- Medication side effects/interactions, balance cognitive side effects
- Self-image, peer pressure, secondary gain issues
- Effects on family and relationships
- Pain significantly interferes with neuropsychological functioning and behavioral, emotional factors interfere with treatment decisions
- TBI and pain do NOT simply act as independent factors
- Pain increases level of disability
- Severe TBI: verbal communication deficits, extent of cognitive functioning impairment, “over-focus” perseveration, sources of information, insurance
Pain and Traumatic Brain Injury

- **Acute Pain:**
  - ”Normal sensation triggered by the nervous system to alert you to possible injury.”

- **Chronic Pain:**
  - ”Pain persists, signals keep firing in the nervous system for weeks, months, even years”
Pain and TBI

- 70-90% Mild TBI have been found to report pain, especially headaches.
- One year post-injury 72.6% post acute rehab had pain, with 47.2% mild pain and 25.4% moderate to severe pain.
- Risk factors: female, lower functional status, depression
- 22% of patients with moderate to severe TBI identified some form of pain.

Hoffman and colleagues, 2005 AAPMR. Uomoto & Esselman AAPMR1993
International association for the Study of Pain

“Pain is an unpleasant sensory and emotional experience associated with actual or potential tissue damage or describe in terms of such damage”

Pain

- Tissue injury triggers an inflammatory cascade that will alter nociceptive function.
- Plasticity and learning play a role in pain.
- Synaptic potentiation is facilitated by repetitive noxious stimulation and at the level of the brain, environmental influences alter the response to noxious stimulation.
- The brain can generate pain in the absence of input from the peripheral nociceptors or the spinal cord, e.g., phantom limb pain.
- Therefore, a brain pattern generating mechanism or Neuromatrix has been proposed.
Components of Pain

- **Nociception**: detection of tissue damage by specialized transducers attached to A delta and C fibers. Aspirin can prevent inflammation and local and regional anesthesia can prevent nociception.

- **Perception of Pain**: triggered by noxious stimulus. It can be generated by lesion in the peripheral or central nervous system e.g. diabetic neuropathy, spinal cord injury or stroke. Pain can occur without nociception. The intensity of chronic pain has no relation to the extent of tissue injury or other pathology.

- **Suffering**: negative response induced by pain and by fear, anxiety, stress, loss of loved objects and other psychological states. Cassell: ”Suffering occurs when the physical and psychological integrity of the person is threatened”.

- **Pain Behaviors**: results from pain and suffering and the things the person do or does not do. Examples: ”ouch”, grimacing, limping, lying down, recourse to health care, refusing to work, etc.

Assessing the patient who has pain

- Onset of duration
- Location, distribution
- Quality
- Intensity
- Aggravating, relieving factors
- Associated features or secondary, signs, symptoms
- Treatment response
- Associated factors:
  - Mood, emotional distress
  - Functional activities
Pain Assessment Scales

Verbal Pain Intensity Scale
No pain  Mild  Moderate  Severe  Very severe  Worst possible pain

Visual Analog Scale
No pain  Worst possible pain

0–10 Numeric Pain Intensity Scale
No pain  Moderate pain  Worst possible pain

“Faces” Scale
0  1  2  3  4  5

References:
Nociceptive vs Neuropathic Pain

Nociceptive Pain
Caused by activity in neural pathways in response to potentially tissue-damaging stimuli

Mixed Type
Caused by a combination of both primary injury or secondary effects

Neuropathic Pain
Initiated or caused by primary lesion or dysfunction in the nervous system

- Postoperative pain
- Mechanical low back pain
- Sports/exercise injuries
- Arthritis
- Sickle cell crisis
- Neurpathetic low back pain
- Neuropathic low back pain
- Distal polyneuropathy (e.g., diabetic, HIV)
- CRPS*
- Trigeminal neuralgia
- Central post-stroke pain

*Complex regional pain syndrome

The National Initiative on Pain Control, 2002
Histamine, serotonin, bradykinin, prostaglandins, ATP, H+, NGF, TNF alpha, endothelins, interleukins
Pain treatment options: TCA, anticonvulsants, Na+ channel blockers, NMDA receptor antagonists, opioids
Molecular Events of Pain Peripheral

**Transduction**
- TRPV1, TRPV2, TRPV3, TRPM8
- ASCI, DRASIC
- MDEG, TREK-1
- BK$_1$, BK$_2$
- P2K$_3$

**Peripheral sensitization**
- NGF, TrkA
- TRPV1
- Na, 1,8
- PKA, PKC isoforms, CalMK IV
- Erk1/2, p38, JNK
- IL-1β, cPLA$_2$, COX2, EP1, EP3, EP4
- TNFα

**Membrane excitability of primary afferents**
- Na$_v$ 1.8, Na$_v$ 1.9
- K$^+$ channel

**Synaptic transmission Presynaptic**
- VGCC
- Adenosine-R
- (mGlu-R)

Molecular Events of Pain Central

**Synaptic transmission Postsynaptic**
- AMPA/kainate-R, NMDA-R, mGlu-R
- NK1
- Na\_\textsubscript{v} 1.3
- K\textsuperscript{+} channels

**Central inhibition**
- GABA, GABA\textsubscript{A}-R, GABA\textsubscript{B}-R
- Glycine-R
- NE, 5-HT
- Opioid receptors
- CB1

**Signal transduction**
- PKA, PC isoforms
- ERK, p38, JNK

**Gene expression**
- C-fos, c-jun, CREB
- DREAM

# Acute vs. Chronic

<table>
<thead>
<tr>
<th>Features</th>
<th>Acute Pain</th>
<th>Chronic Pain</th>
</tr>
</thead>
<tbody>
<tr>
<td>Etiology</td>
<td>Generally known</td>
<td>Generally unknown</td>
</tr>
<tr>
<td>Duration of pain</td>
<td>Short, well characterized</td>
<td>Persist after healing &gt; 3months</td>
</tr>
<tr>
<td>Treatment approach</td>
<td>Underlying disease</td>
<td>Underlying disease and pain disorder</td>
</tr>
</tbody>
</table>

The National Initiative on Pain Control, 2002
## Effects of chronic pain

<table>
<thead>
<tr>
<th>Physical functioning</th>
<th>Psychological morbidity</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Ability to perform ADLs</td>
<td>• Depression</td>
</tr>
<tr>
<td>• Sleep disturbances</td>
<td>• Anxiety</td>
</tr>
<tr>
<td></td>
<td>• Anger</td>
</tr>
<tr>
<td></td>
<td>• Loss of self-stem</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Social consequences</th>
<th>Society consequences</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Relationship with family and friends</td>
<td>• Health care cost</td>
</tr>
<tr>
<td>• Intimacy, sexual activity</td>
<td>• Disability</td>
</tr>
<tr>
<td>• Social isolation</td>
<td>• Loss of workdays</td>
</tr>
</tbody>
</table>

The National Initiative on Pain Control, 2002
## Descriptions of neuropathic pain

<table>
<thead>
<tr>
<th>Sensations</th>
<th>Cardinal signs and symptoms</th>
</tr>
</thead>
<tbody>
<tr>
<td>Burning</td>
<td>Allodynia: pain from stimulus that does not normally evokes pain</td>
</tr>
<tr>
<td>Paresthesias</td>
<td>Thermal</td>
</tr>
<tr>
<td>Paroxismal</td>
<td>Mechanical</td>
</tr>
<tr>
<td>Lancinating</td>
<td>Hypeperalgesia: exaggerated response to a normally painful stimulus</td>
</tr>
<tr>
<td>Raw skin</td>
<td></td>
</tr>
<tr>
<td>Shooting</td>
<td></td>
</tr>
<tr>
<td>Deep, dull, bone-like ache</td>
<td></td>
</tr>
</tbody>
</table>

The National Initiative on Pain Control, 2002
Pathophysiology of Neuropathic pain

- “Pain arising as a direct consequence of a lesion or disease affecting the somatosensory system”
- Chemical excitation of nociceptors
- Recruitment of nerves outside of the site of injury
- Excitotoxicity
- Sodium channels
- Ectopic discharge
- Deafferentation
- Sympathetic involvement
- Antidromic neurogenic inflammation
- Central sensitization
- Maintain by peripheral input
Diagnostic Studies and Limitations

- **Studies**
  - Xrays, CTs, MRIs,
  - EMG
  - NCS
  - Quantitative sensory testing
  - Epidermal skin biopsy

  Insensitive in acute injury (EMG/NCS)
  Normal studies does not rule out neuropathic pain
  Does not assess small fiber (which mostly involved in neuropathy)
Pharmacological Management of Neuropathic Pain

- **First line medications:**
  - Clinical trials for PHN, DNP and HIV neuropathy, Chemo induced PN
- **Antidepressants with both norepinephrine and serotonin reuptake inhibition:**
  - Tricyclic antidepressants: nortriptyline or desipramine: SE, limitations such as cardiac disease
  - Duloxetine and venlafaxine: 2-4 weeks
- **Calcium Channel α ligands (gabapentin and pregabalin)**
  - Dose dependent dizziness and sedation and renal dosage reduction
  - **Topical lidocaine 5%**
- **Second line Medications:**
  - **Tramadol:** weak opioid μ receptor (inhibits reuptake of serotonin and norepinephrine. SE: lower seizure threshold and CI with SSRIs
  - **Opioid Analgesics:** morphine and oxycodone
- **Third line medications:**
  - antidepressants (SSRIs), antiepileptics, topicals

Recommendations for pharmacological management of neuropathic pain, Mayo clinic 2010;85 S3-S14
Pharmacological Management of Neuropathic Pain

- Recent clinical trials
  - Botulinum Toxin: studies shown in PHN, DPN, significant reduction of pain and allodynia vs. placebo
  - High concentration capsaicin patch: improvement with prolonged use over 8-week
  - Lacosamide: antiepileptic, significant pain reduction in DPN

Recommendations for pharmacological management of neuropathic pain, Mayo clinic 2010;85 S3-S14
Pharmacological Management of Neuropathic Pain

- Combination Therapy likely to be best tolerated in refractory cases
- Gabapentin and ER-oxycodone resulted in lower doses of each and better pain relief in PHN and DPN
- ER-oxycodone and pregabalin better tolerated and improvement in QoL
- Nortriptyline and gabapentin
- Pregabalin and topical 5% lidocaine
- Failed back surgery syndrome: spinal cord stimulator

Recommendations for pharmacological management of neuropathic pain, Mayo clinic 2010;85 S3-S14
General Principles of Opioid therapy for Neuropathic pain

- Opioids should be slowly titrated
- Fixed dose regimes over prn preferred
- Document treatment plan and outcome
- Recommend written agreement when using opioids
- Most side effects are manageable
- Opioid are effective in treatment of neuropathic pain
Algorithm for Medication Selection in Chronic Pain

**Nociceptive Pain**
- Short-Term NSAIDs, Cox-II Inhibitors, Opioids
  - Secondary Sleep Disturbance
  - Persist After Adequate Analgesia
    - Evaluate Risks
      - Antihistamine, Zolpidem, Low Dose Benzodiazepine
      - Low Dose Tricyclic at Bedtime: Trazodone

**Neuropathic Pain**
- Persist After Adequate Analgesia
  - Evaluate Risks
    - Lidocaine Patch, Gabapentin, Topiramate, Lamotrigine, Tizanidine, Opioids

**Pain Condition + Major Depression**
- Secondary Persist After Adequate Analgesia
  - Evaluate Risks
    - Titrate Nortriptyline, Desipramine, Others
      - SSRI Trial
        - Venlafaxine, Duloxetine

**Primary**
- Assess Medical Risks
Pain and TBI: spasticity

- Spasticity in severe TBI is associated with both rigidity and dystonic-type posturing.
- Risk for early contractures
- MSK exam to r/ou MSK co-morbidities, occult fractures, peripheral nerve injuries and heterotopic ossification
- Xray, bone scan
- Stepped treatment and combined methods (table)
- Medications:
  - Acetaminophen
  - NSAIDs
  - Transdermal lidocaine
  - Clonidine patch
  - Narcotics analgesics (morphine)
  - Spasticity- meds: Dantrolene sodium, Baclofen, Tizanidine, Diazepam.
Pain and TBI: spasticity

- Physical modalities (stretching, splinting, casting, electrical stim, ice, heat, acupuncture, massage)
- Local neural block or chemodenervation
- Intrathecal baclofen infusion pump
- Oral medications
- Surgical intervention: tendon lengthening, Capsulotomy, neurectomy, rhizotomy
Pain and mild TBI

- Headaches:
  - Poorly characterized
  - Migraine and tension type PTHA are most frequent
  - 37% tension-type, 27% migraine, 18% cervicogenic PTHA (Packard)
- Headaches and psychological factors
- Premorbid headaches
Pain and mild TBI

- ICDH-2 Diagnostic Criteria
- A. Headache, no typical characteristics known, fulfilling criteria C and D
- B. Head trauma, with all of the following
  - a. Either no loss of consciousness or LOC of < 30 min
  - b. GCS > or equal 13
  - c. Symptoms and/or signs diagnostic of concussion
- C. Headache develops within 7 days after head trauma
- D. One or other of the following
  - a. Headaches resolves within 3 months after head trauma
  - b. Headache persists but 3 months have not yet passed

Comment: Acute PTHA due to moderate or severe head injury develops 7 days after regaining consciousness and meets criteria: LOC > 30 min, GCS < 13, PTA > 48 hrs, positive imaging
# PTHA

<table>
<thead>
<tr>
<th>Migraines</th>
<th>Tension Type</th>
</tr>
</thead>
<tbody>
<tr>
<td>Moderate to severe</td>
<td>Mild to moderate</td>
</tr>
<tr>
<td>Throbbing, pulsating, pounding</td>
<td>Vicelike, tight, squeezing</td>
</tr>
<tr>
<td>Activity, bending over worsens</td>
<td>No worsening with movement</td>
</tr>
<tr>
<td>Unilateral (60%)</td>
<td>Bilateral</td>
</tr>
<tr>
<td>Nausea and/or vomiting</td>
<td>No nausea or vomiting</td>
</tr>
<tr>
<td>Photophobia and phonophobia</td>
<td>Photophobia or phonophobia rarely</td>
</tr>
</tbody>
</table>

## Headaches

- **Sinus:** pain is usually behind the forehead and/or cheekbones
- **Cluster:** pain is in and around one eye
- **Tension:** pain is like a band squeezing the head, pain, nausea and visual changes are typical of classic form
- **Migraine:**
Treatment PTHA

- **Acute**
  - Restore sleep cycle, NSAIDs, caffeine, triptans
  - Avoid opioids if possible

- **Preventive**
  - Considered with HA frequency is more than 4-6 days per month
  - Amitriptyline good for PTH and depression and sleep disorder
  - Valproic acid, gabapentin, SSRIs for mood disorder, anxiety related issues
  - Tizanidine for somatic pain or myofascial pain
  - Botox (onabotulimun toxin A)
  - Children and adolescent should be treated if possible with behavioral therapy – may use Bblockers or Ca-channel blockers
Treatment of PTHA

- Guidelines:
  - Start each preventive at a low dose and increase slowly
  - Adequate trial at an effective dose is usually 6-8 weeks
  - Perform through ROS and review of current medications
  - Always discuss contraception with at-risk patients (e.g., valproic acid, topiramate)
  - Choose a drug based on patient preference and lifestyle (avoid Bblockers in athletes)
  - Choose medication to treat comorbidities effectively
CEFALY

Cefaly delivers transcutaneous electrical nerve stimulation (TENS) to the trigeminal nerve to either ease the pain during an attack, or in the longer term, help minimize their frequency.

For treatment during a migraine, Cefaly uses high-frequency neurostimulation, which limits the pain signals from the nerve center. For preventative use, intended for regular sufferers, Cefaly uses low-frequency stimulation to change the migraine’s trigger threshold, making it harder to reach and the headaches less painful, or causing them to disappear entirely.

According to the company, users can expect to feel a light sensation when wearing the headband, though it says the dose of electromagnetic waves is weaker than you receive when watching television.

For preventative use, Cefaly is intended to be worn for 20-minute sessions. Pressing a button will begin the session, with the intensity and tingling gradually increasing as time progresses. The idea is to build up a tolerance to the sensation and, in effect, the migraine threshold in your brain, though if the sensations do become too much, pressing the button again will reduce the intensity.

Researchers have been studying the efficacy of Cefaly since 2011 through a series of clinical studies, 54.4% of the subjects tested reported satisfaction with the treatment provided by Cefaly.
**The Cerena Transcranial Magnetic Stimulator**

- This pulse stimulates the brain's occipital cortex, which may stop or ease migraine pain.
- 38% of people using the stimulator said they were pain-free two hours later, compared to 17% of patients who did not use the device. A full day after the onset of migraine, nearly 34% of device users said they were pain-free, compared to 10% of people who hadn't used the device.
- "The Cerena TMS is another tool in the battle to relieve migraines"
- Side effects are rare, but may include in isolated cases sinusitis, language difficulties and vertigo
- The new device is approved only for use by those aged 18 or older, and should not be used by people with suspected or diagnosed epilepsy or a family history of seizures. It should not be used by anyone with any metal device implanted in the head, neck or upper body, pacemaker or deep brain stimulator.
- The stimulator, manufactured by eNeura Therapeutics of Sunnyvale, Calif., is not meant to be used more than once every 24 hours.
Injections

- Botox protocol
- Greater Occipital block
Myofascial trigger point
Trigger point

- Trigger point are tender muscles
- Injections:
  - Corticosteroids
  - Saline
  - Local anesthetics
  - Dextrose
  - Botulinum toxin
Myofascial trigger point

- Treatment for myofascial pain syndrome involves a multi-faceted program designed to break up the trigger point and relieve areas of muscle soreness.

- Includes:
  - Integrating several types of physical therapy
  - Ultrasound, electrical stimulation, and myofascial release
  - May also include massage therapy, chiropractic/osteopathic manipulation, & nutritional supplementation
  - Trigger point injections have also proven to be very effective in reducing spasms and relaxing muscles
Neck pain

- Neck pain usually results from strains and sprains.
- Muscle strains and ligament strains are the most common causes.
- Injuries, arthritis, a ruptured or herniated disk, meningitis, and fibromyalgia are other causes.
- Other disorders that cause only neck pain are:
  - Atlantoaxial subluxation: the first and second vertebrae are misaligned.
  - Cervical spondylosis: In the neck, the vertebrae and the disks between them degenerate. As a result, the nerves that emerge through the vertebrae may be pinched. Sometimes the spinal canal is narrowed (cervical spinal stenosis) and the spinal cord is compressed.
  - Temporomandibular joint disorders: Problems occur in the joint of the jawbone. Women are more commonly affected, usually during their early 20s or their 40s.
  - Spasmodic torticollis: The neck muscles contract, causing the head to tilt and rotate into abnormal positions.
  - Artery dissection, a blockage or tumor in the esophagus, infections (such as a bone infection), and inflammation of the esophagus or thyroid gland are other disorders that can cause neck pain.
What is Complementary and Alternative Medicine (CAM)?

- Involves Medical doctors, doctors of osteopathy, physician assistants, nurse practitioners
- Most common – breathing exercises, herbal medicine prescribing, meditation, functional medicine
- Referrals to other providers for acupuncture, massage, yoga, meditation
- Separate from conventional medicine; complete systems of theory and practice
- Homeopathy, naturopathy, Chinese medicine, Ayurveda
- Biologically-based Practices
- Use substances found in nature Dietary supplements, herbal preparations, diet therapy
- Mind-Body Medicine
- Techniques to enhance the mind’s ability to affect bodily function and symptoms
- Meditation, yoga, prayer, mental healing, creative arts, biofeedback, hypnosis
- Manipulative and Body-based Practices
- Based on manipulation or prescribed movement
- Chiropractic, massage,
- Energy Medicine Use energy fields – either extant in body or by adding some form of electromagnetic (EM) energy
- Therapeutic touch, Reiki, qi gong, pulsed EM field therapy, cranial electrotherapy stimulation, laser/light therapy, transcranial direct current stimulation (tDCS), repetitive transcranial magnetic stimulation (rTMS)

CAM Defined

- National Center for Complementary and Alternative Medicine defines CAM as:
  - Health care systems, practices, and products that are not generally considered part of conventional medicine
  - Conventional medicine (also called Western or allopathic medicine)
  - The boundaries between CAM and conventional medicine are not absolute
  - Specific CAM practices may, over time, become widely accepted

- Treatments/modalities
  - Movement Therapies
  - Yoga • Tai chi • Qi gong
  - Transitional Aquatics
  - Mindfulness Activities – may be incorporated
  - Breathing, progressive relaxation, qi gong and tai chi

- Integrative Medicine: Combines conventional medicine and CAM
  - Mind and body • Family • Is there anything that it doesn’t?
### Alternative Care for Symptoms of TBI

<table>
<thead>
<tr>
<th>A</th>
<th>Good evidence + benefits outweigh harms</th>
<th>Self-education and training (biofeedback), for stress, headaches and sleep; acupuncture for pain; omega-3s for anxiety/depression</th>
</tr>
</thead>
<tbody>
<tr>
<td>B</td>
<td>Fair evidence + benefits outweigh harm</td>
<td>Stress management – biofeedback and mindfulness training for sleep, mood, cognition and PTSD; acupuncture for headaches; CES for headaches, insomnia, depression; magnesium for headaches; melatonin for insomnia</td>
</tr>
<tr>
<td>C</td>
<td>Fair evidence but benefits not greater than possible harm</td>
<td>St. John’s Wort for depression, other herbals</td>
</tr>
<tr>
<td>D</td>
<td>Evidence for ineffectiveness or harm definitely outweigh risks</td>
<td></td>
</tr>
<tr>
<td>I</td>
<td>Insufficient evidence</td>
<td>Healing touch, Reiki, manipulative treatments</td>
</tr>
</tbody>
</table>
Neuropsychology

- Ideally done prior to enrollment in a Pain management program
- 1. Can the person with TBI focus long enough to participate?
- 2. Can the person recall the behavioral and physical exercises?
- 3. Can the person follow an exercise sequence?
- 4. Can the person follow the logic of the treatment plan?
- 5. Are there any significant emotional factors that need to be treated prior?
- 6. Is there hx of abuse alcohol or drugs? (90% in chronic pain patients)

Assist with:
  - Understanding obstacles (family and patient)
  - Outline steps for compensating for cognitive limitations
  - Set up appropriate incentives to enhance participation
  - Outline Cognitive, Motivational and Emotional components of the pain
  - Returning to work
Neuropsychology

- Psychologist or Neuropsychologist to treat pain as a learned behavior:
  - Relaxation training
  - Hypnosis
  - Stress management
  - Attention-diversion strategies
  - Biofeedback
Brain Imaging Techniques

**PET**
- Requires relatively long pain stimulation periods (40 – 60s).
- Different functional states (e.g., pain and rest) are always acquired in separate scans.
- Maximum number of scans that can be acquired is limited by radioactivity dose restraints.
- Usually requires multi-patient study designs.
- Potential to map neurotransmitter systems and drug uptake in vivo and molecular imaging.
- Provides a solution in cases where fMRI cannot be accomplished because of contraindications.

**fMRI**
- Offers better temporal and spatial resolution than PET.
- Pain stimuli do not need to be applied over along period.
- The control state and the active pain condition are done in the same run.
- Better suited than PET for studying cognitive effects on pain processing.
- Unlimited amount of repetitions within a single patient, allowing single participant, and follow-up studies.
- Offers less comfort to the patient (noise, body constrained in the magnet bone).
- Requires expensive fMRI-compatible stimulation and monitoring equipment.

**MEG**
- Allows mapping of the sequential activation of brain structures in pain processing.
- Provides a direct measure of neuronal activity.
- The most ecological technique with the highest comfort and least distress for participants.
- Allows conclusions from single trial and single participant studies (great clinical potential).

Questions?

<table>
<thead>
<tr>
<th>Number</th>
<th>Description</th>
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</thead>
<tbody>
<tr>
<td>0</td>
<td>No Hurt</td>
</tr>
<tr>
<td>1</td>
<td>Hurts Little Bit</td>
</tr>
<tr>
<td>2</td>
<td>Hurts Little More</td>
</tr>
<tr>
<td>3</td>
<td>Hurts Even More</td>
</tr>
<tr>
<td>4</td>
<td>Hurts Whole Lot</td>
</tr>
<tr>
<td>5</td>
<td>Hurts Worst</td>
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