**Too Hot To Handle**

Jessie Podolak, PT, DPT, TPS

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**Disclaimer...**

- Many VERY smart people have contributed to this
- I am “dumbing it down” so I can understand it
- Will all neuroscientists, neurobiologists and immunologists please leave...
- A LOT remains unknown about CRPS...

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**A Neuroscience CRPS definition...**

CRPS is a multiple system output, activated by the pain neuromatrix (brain map) in response to perceived threat

Adapted from Mosley GL, A pain neuromatrix approach to patients with chronic pain, Man Ther. Aug 2003;8(3):130-140.
Introduction: CRPS

- Pain in combination with sensory, autonomic, trophic and motor abnormalities.
  - CRPS-1: Nerve lesion cannot be identified
  - CRPS-2: Nerve lesion can be identified

Introduction

- Criticism of CRPS 1 and CRPS 2
  - Fracture or surgery - damage peripheral nerve, but usually Dx as CRPS 1
  - Nerve degeneration causes CRPS-1?
    - Other causes of neuropathic pain are frequently associated with a loss of C-fiber peripheral terminals, the specificity of these findings with respect to CRPS is questionable (Devigili).

Introduction

- Our understanding of CRPS has increased substantially in the past decade.
- Three major pathophysiological pathways:
  1. Aberrant inflammatory mechanisms
  2. Vasomotor dysfunction
  3. Maladaptive neuroplasticity.
- Between-individual variability
After minor or moderate tissue injury (i.e., a wrist fracture).
In the acute phase, the injured limb is usually extremely painful, red, warm (although sometimes it quickly becomes cold) and swollen (Veldman 1993).

- Allodynia (non-painful stimuli evoke pain)
- Hyperalgesia (painful stimuli evoke more intense pain than usual)
- Changes in sweating
- Changes in hair and nail growth
- Muscle weakness
- Mechanical and thermal hyperalgesia are frequently present
Clinical presentation & diagnosis

- Pain often spreads
- Voluntary motor control is reduced
- Hyperpathia (nociceptive stimuli evoke exaggerated levels of pain)
- Negative sensory signs
  - Hypoesthesia (reduced sense of touch)
  - Hypoalgesia (decreased sensitivity to painful stimuli)
  - Hypothermesthesia (abnormally decreased sensitivity to heat)

- Mixture of noxious sensations and sensory loss
- Over months: Warm limb becomes cold
- Dystonia
- Tremor
- Myoclonus
- Activity of the limb: exacerbates signs and symptoms
- Over time, clinical features spread proximally (but not distally) and can even emerge on the opposite or ipsilateral limb

Clinical presentation & diagnosis

- Diagnosis of CRPS:
  - Orlando criteria
  - International Association for the Study of Pain
  - Modified version called the Budapest criteria (panel)
  - Diagnosis according to the Budapest criteria - grouping of signs and symptoms into four distinct categories
• Unclear

  • 5 cases per 100,000 person-years in the USA
  • 26 per 100,000 person-years in the Netherlands.
  • Might expect that 20,000–80,000 new cases of CRPS would be identified per year in the USA.
  • Incidence increases with age until 70 years of age, and 3–4 times more women than men are affected.
  • The arm is affected in about 60% of cases and the leg in about 40%.
Epidemiology

• Resolution rate:
  • Ranging from 74% in the first year to 36% within 6 years.

Epidemiology

• Fractures (about 45%), sprain (about 18%), and elective surgery (about 12%) are the most frequently reported triggering events.

Epidemiology

• Spontaneous-onset CRPS - uncommon (<10% of cases).
  • Associated with substantial disability, loss of quality of life, and personal and societal economic burden.
Risk factors & prognostic determinants

• Lots of people have fractures, injuries and surgery and DO NOT develop CRPS

• Some individuals are more susceptible than others.

1. Psychological risk factors
   • Compelling evidence that patients with CRPS are more anxious and depressed than healthy control individuals.
   • Whether patients with CRPS are more anxious and depressed than patients with other chronic pain syndromes is unclear
1. Psychological factors

- No evidence of psychological risk factors for CRPS onset
  - A large population-based case control study reported no difference in psychological variables between those who developed CRPS after trauma
  - Recent prospective multi center cohort study - 600 consecutive patients with a fracture reported that none of the psychological factors predicted the development of CRPS-1.
  - The popular presumption that anxiety and depression predispose to CRPS is incorrect.

2. Immobilization of the injured limb

- Immobilization - risk factor for CRPS,
  - Topical application of capsaicin (induces neurogenic inflammation):
    - Mechano sensitivity
    - Thermo sensitivity,
    - Perceptual disturbances
  - Reported in people whose limb was subsequently immobilized for 24 h, but not in people whose limb was not immobilized (Moseley GL, unpublished).
  - The signs rapidly resolved once the limb was moved again.

3. Epidemiological findings

- Angiotensin-converting-enzyme inhibitors at the time of trauma and a history of migraine or asthma - associated with increased risk of developing CRPS.
  - Migraine - risk factor for CRPS
  - Both of these risk factors implicate inflammation: angiotensin-converting-enzyme inhibitors increase the availability of substance P and bradykinin, which are important mediators of inflammation, and migraine and asthma share an underlying mechanism of neurogenic inflammation with CRPS.
3. Epidemiological findings

- Fracture - more favorable course than soft tissue injury
- Gender does not seem to affect prognosis
- Women > men: risk for more severe form of CRPS
- Cold CRPS: Worse prognosis than warm CRPS

3. Epidemiological findings

- Longitudinal study
  - 1549 nearly consecutive patients who presented with wrist fracture and who were managed non-surgically were assessed within 1 week of their fracture and then followed up 4 months later (Moseley GL, unpublished).
- Mean pain intensity over the past 2 days (> 5/10): Red flag for CRPS

4. Genetic findings

- CRPS sometimes occurs in several family members and siblings of young-onset cases have an increased risk of developing the syndrome
  - Suggest potential genetic predisposition to CRPS
  - Need more research
Genetics?  

Genetically Coded…  

Environmentally Sculpted

Risk factors & prognostic determinants summary...

1. Anxiety/depression – no  
2. Immobilization after fracture – yes  
3. Neurogenic inflammation – yes  
4. High level of acute pain – yes  
5. Genetics – likely  
6. Gender, fracture, etc. – not compelling

Before we move on...

A neuroscience explanation of these risk factors is needed to start the understanding the vast neurobiological and neurophysiological processes that underpin the development and maintenance of CRPS...
Remember these?

Ion Channels

Ion Channels
DNA → mRNA → Proteins

Every 48 hours...

distribution of ion channels...
Removing myelin

- Mechanical
- Immune
- Chemical

Ion Channels

Recap...CRPS (peripheral neuropathic)

- Genetics/family – ion channel expression
- Stress/Anxiety
  - High level of initial pain – adrenaline ion channels
  - Fracture, surgery, sprain – adrenaline ion channels
- Temperature sensitization – ion channels
- Female/male - ? Hormonal/ion channels
- Immobilization – movement channels
- Extreme sensitization – nerve up-regulation
- Spreading pain – connected nervous system
Key pathophysiological changes

- Three major pathophysiological pathways:
  1. Aberrant inflammatory mechanisms
  2. Vasomotor dysfunction
  3. Maladaptive neuroplasticity.

1. Aberrant inflammatory mechanisms

Ever had inflammatory soup?
**Tissue Inflammation**

When tissues get injured, there is an inflammatory "soup" that ensues with release of various chemicals in/around the tissues by the damaged cell membranes: bradykinin, prostaglandin, serotonin, leukotrienes, etc. (Drummond 2010) The immune cells also release macrophages, cytokines and histamine via mast cells (Dickerson, Undem et al. 1998). This is sterile inflammation, normal and designed for protection and healing. With these processes there is stiffness > pain.

**Inflammation: More inflammatory cells**

- Minor tissue trauma increase cytokine signaling in the injured tissue
  - Cytokines and nerve growth factor (NGF) wake up nociceptors and increase nerve sensitivity

**Inflammation: Nerves go both ways**

- Activation of cutaneous nociceptors can induce retrograde depolarization of small-diameter primary afferents (axon reflex), causing the release of neuropeptides such as substance P and calcitonin-gene-related peptide (CGRP).
Neuropeptides promote vasodilation and protein extravasation (cells leaking from capillaries) in the tissue:
  - Neurogenic inflammation
  - Redness, warm and swelling

**Neurogenic Inflammation**

Via antidromic impulses (retrograde depolarization), nerves fire "backward" and facilitate increased release of substance P, noradrenaline, neurokines, calcitonin-gene-related peptides, nerve growth factor, etc. Substance P is vasoactive and contributes to mast cell degranulation, which release histamine. This in turn signals macrophages and enzymes to facilitate "mop up" of the chemicals and part of the healing process (Nordin, Nystrom et al. 1984). These chemical releases infuse the "soup" with more inflammatory cells, and activate nociceptors, thus increasing CNS barrage. Neurogenic inflammation is characterized by persistent swelling and inflammation and can be driven by thoughts.

**Neurogenic Inflammation increases**

- Serum concentrations of CGRP and substance P – higher in patients with CRPS than in healthy control individuals.
- Elevated CGRP release is probably responsible for the augmented flare response in patients with CRPS
Mechanism for neurogenic inflammation
- Substance P produces greater extravasation in both the affected and the unaffected limbs of patients with CRPS than in control individuals.

Facilitated cutaneous neuropeptide signaling contributes directly to the enhanced extravasation, limb edema, and increased cytokine expression that are present in CRPS.

Cytokine changes are associated with the extent of mechanical hyperalgesia
Mechanical hyperalgesia
- Hallmark of central sensitization
- Excitability of neurons in the spinal cord is increased

Inflammatory cytokines:
- Acts locally in the limb
- Sensitization of secondary nociceptive neurons in the spinal cord or by glial–neuronal interaction.
Glia in the spinal cord?

- Or neuroglia (Greek for "glue"), classically = cells that provide metabolic & structural support, but also:
  - Establish & maintain synapses
  - Regeneration and plasticity
  - Myelin formation/repair
  - Immune function
- Outnumber neurons >10 to 1


Response of microglia in the spinal cord after peripheral nerve injury

A cascade of immune changes start to occur…
Injury to a peripheral nerve and electrical stimulation of C-fibers each cause an increase in the permeability of the blood-spinal cord barrier and blood-brain barrier


Summary

• The changes in the spinal cord however, also track up to the brain and affect the blood-brain barrier

• Changes in blood-brain-barrier leads to changes in the body maps in the brain
  – Smudging occurs
  – With smudging, the brain has a hard time recognizing “self” from “non-self”

• This activates the immune system, especially cytokine
  (Costigan, Moss et al. 2009)

Summary

• With increased cytokine activity (Devor 2006)
  – Ion channel upregulation with increased widespread sensitization
  – Persistent swelling
  – Investigation and clinical awareness of old injuries “waking up”

• With the increased “smudging of the maps”
  – Nervous system is upregulated (allodynia)
  – Altered temperature
  – Neglect
2. Vasomotor dysfunction

- Substance P release
- Capillaries open
- Swelling
- Warmth

Which way do nerves fire?

- Antidromic impulses
  - Substance P release
  - Capillaries open
  - Swelling
  - Warmth
Antidromic impulses

- Substance P
  - Mast cells degranulation
  - Histamine
- Increased Enzyme activity

Sympathetic Sprouting and basket weaving

Causes and consequences of sympathetic basket formation in dorsal root ganglia

Matt S. Ramer, Stephen W.N. Thompson, Stephen B. McMahon

![Images of hands showing sympathetic sprouting and basket weaving]
Dorsal root ganglion – non-myelinated
Sympathetic axonal sprouting and weaving around DRG, releasing more adrenaline onto it
- 70% of the adrenal receptors in the body live in the DRG
Nerve fires: both ways

Vasomotor

- Common in CRPS
- **Affected limb:**
  - Usually warmer than the healthy limb early on
  - Colder than the healthy limb later
  - Temperature shift - activity in vasoconstrictor neurons
  - Three distinct patterns of temperature change:
    1. Warm type
    2. Intermediate type
    3. Cold type

Vasomotor

- **Warm type**
  - Affected limb warmer and skin perfusion values were higher than the contralateral limb (CPRS for a mean of 4 months).
  - Norepinephrine concentrations from the venous effluent above the painful area are lower in the affected limb than in the contralateral one.
**Vasomotor**

- **Intermediate type**
  - Temperature and perfusion either warmer or colder, depending on the amount of sympathetic activity (mean disease duration of 15 months).

- **Cold type**
  - Temperature and perfusion in the affected limb consistently lower than those in the contralateral limb (mean of 28 months).
  - Norepinephrine concentrations lower on the affected side.

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**Vasomotor**

- Inhibition of cutaneous sympathetic vasoconstrictor neurons
- Thermoregulatory impairment probably caused by functional changes in the spinal cord, brainstem, or brain that are triggered by the initial trauma.
Vasomotor

• There are suggestions that cutaneous sympathetic vasoconstrictor activity returns to normal as CRPS persists, even though the limb becomes cold and bluish.

Vasomotor

• Central disturbances – acute
• Neurovascular transmission – chronic

• About 20% of patients with CRPS have the cold type from the start. These patients not only differ in skin temperature but also in sensory symptoms and history.

Vasomotor

• The sympathetic nervous system, in addition to its effect on peripheral circulation in CRPS, might also contribute to pain.
• Nociceptors develop catecholamine sensitivity, probably as a result of decreased activity of cutaneous sympathetic vasoconstrictor neurons.
• Norepinephrine released by the sympathetic nerve fibers activate or sensitize the altered afferent neurons.
Easy huh?

Maybe you can start seeing why it is *COMPLEX* regional pain syndrome...

3. Maladaptive neuroplasticity

Which way do nerves fire?
In CRPS there is a structural and functional neuroplastic change that occurs...
1. Driven by the brain
2. To protect/survive

If you can answer this, you’re well on your way:

• What is 1 + 1?
Allodynia

- A-beta grows in C-fibers die back

Sensitized Mode 1-G

Sensitized Mode 1-H
The brain...

How Dangerous is this?

This is not dangerous

Inhibition Endogenous

How Dangerous is this?

This is dangerous

More information

Facilitation Neuronal adaptation
### End-Result

<table>
<thead>
<tr>
<th>Process</th>
<th>Consequence</th>
</tr>
</thead>
<tbody>
<tr>
<td>Death of the inhibitory neurons</td>
<td>Decreased gating from the periphery</td>
</tr>
<tr>
<td>C-fibers pull back; A-fibers grow in</td>
<td>Allodynia</td>
</tr>
<tr>
<td>Up-regulation of second-order neurons</td>
<td>Increased firing towards the brain</td>
</tr>
<tr>
<td>Inappropriate synapsing – other levels</td>
<td>Spreading pain</td>
</tr>
<tr>
<td>Inappropriate synapsing – other fibers</td>
<td>Sympathetic, immune, motor contributions</td>
</tr>
<tr>
<td>Inappropriate synapsing – other side</td>
<td>Bilateral “mirror” pains</td>
</tr>
<tr>
<td>Decreased endogenous mechanisms</td>
<td>Allodynia and Hyperalgesia</td>
</tr>
</tbody>
</table>

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### 3. Neuroplasticity

- The CNS undergoes **functional** and **structural changes** in people with persistent pain

  > Central Sensitization

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### Neuroplasticity

- Sensitized spinal nociceptive neurons become:
  - More responsive to peripheral input
  - Might even fire in the absence of such input

- Central sensitization can cause chronic pain, hyperalgesia, and allodynia, as well as the spreading hyperalgesic priming
Neuroplasticity

• In some patients a transient insult can lead to chronic pain
• A transient insult triggers long lasting changes in primary afferent nociceptors that prime them to become hyper-responsive to future mild insults that would normally not evoke pain in the unprimed state.

NEXT INJURY!!!!!

Neuroplasticity

• Impaired motor function
  • Common after most injuries but generally resolves
  • In CRPS susceptible patients develop marked movement disorders.
  • Dystonia - most prevalent movement disorder in CRPS
  • Characterized in the arm by persistent flexion postures of the fingers and wrist

Neuroplasticity

○ The risk of dystonia spreading to additional limbs in patients with CRPS increases with the number of limbs that are already dystonic.

○ This accelerated disease course is a typical characteristic of maladaptive neuronal plasticity.
Want the BAD NEWS?

- CRPS is a brain disorder
- We have only really covered the periphery to this point
- We have not even covered the brain yet
- This is where the MAJOR changes occur

Input to the brain...

- Beta fibers
- C fibers
- Interneuron
- Second Order Nociceptive Specific
- Second Order Wide Dynamic Ranging Neuron

Neuroplasticity

- Activation and up-regulation of glutamate receptors, which enhance signal transmission in the nociceptive circuitry from the spinal cord to the cerebral cortex
  - Glutamate – excitatory neurotransmitter
  - 70% of all brain synapses are glutamate synapses
The Brain’s Processing (Blobology 101)...Grandma

PREMOTOR/MOTOR CORTEX
organizes and prepares movements

CINGULATE CORTEX
regulation, valuation

PREFRONTAL CORTEX
problem solving, memory

AMYGДALA
fear, fear conditioning, addiction

SENSORY CORTEX
sensory discrimination

HYPOTHALAMUS/THALAMUS
stress responses, autonomic regulation, motivation

CEREBELLUM
movement, cognition

HIPPOCAMPUS
memory, spatial recognition, fear conditioning

SPINAL CORD
gating from the periphery

A TYPICAL PAIN NEUROTAG

Physical Therapy in Sport
A neuroscience approach to managing athletes with low back pain

Butler & Moseley 2003
A neuroscience approach to managing athletes with low back pain

Beliefs

Knowledge, logic

Denotes synaptic modulation

Physical Therapy in Sport

Beliefs

Knowledge, logic

Physical Therapy in Sport

Beliefs

Knowledge, logic

Denotes synaptic modulation
Living your pain

“Nerves that fire together, wire together”
Living your pain

“Nerves that fire together, wire together”

Neuroplasticity

- The CNS undergoes functional and structural changes in people with persistent pain
- Leads to central sensitization
- Changes occur in structures involved in the emotional aspects of pain
  - Amygdala
  - Anterior cingulate
  - Prefrontal cortex

Enter the homunculus
Patterns of cortical reorganization in complex regional pain syndrome

Christian Mahboob, MD; Hermann O. Handwerker, MD, PhD; Berhard Neunerfleisch, MD; and Frank Birklein, MD

Unaffected side

Affected side

Patterns of cortical reorganization in complex regional pain syndrome

Substantial reorganization of the somatotopic map within the primary somatosensory cortex (S1) contralateral to the affected limb in patients with CRPS.

fMRI

Substantial reorganization of the somatotopic map within the primary somatosensory cortex (S1) contralateral to the affected limb in patients with CRPS.

Neuroplasticity

• Cortical reorganization might explain:
  • The spatial distribution of sensory disturbances in a glove-like or stocking-like pattern, the occurrence of tactile induced referred sensations
  • The perception that the limb is bigger than it really is, and the presence of hemi-sensory deficits.
**Neuroplasticity**

- People with longstanding CRPS tend to perceive their affected limb to be larger than it really is.
  - CRPS patient believes the hand feels 107% bigger
    - Hurts more when it looks bigger
    - Does not move different
    - More swelling when it looks bigger

*Visual distortion of a limb modulates the pain and swelling evoked by movement*

Distortions of the mental image of their limb
Missing components or alterations in shape, posture, and temperature of the whole limb or of discrete parts of the limb.
**Neuroplasticity**

- Aware of their altered feelings towards the limb
- Although they believe that the limb is theirs, they feel as though it is not
- Neglect-like disturbances reported in CRPS are a result of:
  - Avoid provocation of pain
  - Altered representation of aspects of the limb

**Neuroplasticity**

- Feelings of hostility or disgust towards the affected limb
- Feel as though it is a separate entity
- Foreign body that they would like to have amputated

Body perception disturbance: A contribution to pain in complex regional pain syndrome (CRPS)

Jennifer S. Lewis **a**, b; Paula Kerschen **b, c, d**; Candida S. McCabe **e, f**; Kathryn M. McPherson **g, h**; David R. Blake **i, j**
I have a question about a young gal I am seeing now with CRPS. She is 18 years old. When she first came to see me she had no motion in her right arm and pain 9/10. Within 3 months she had full motion, pain at 1/10 and using her arm for all ADL’s. A month ago she hurt her wrist picking up her cat and is now almost back to square one with pain 9/10, and no motion distal to the elbow. She was catastrophizing initially, but that has stopped now. She is using her right arm for gross motor activities, so not total neglect. Today she tells me that she does not feel like she has a right arm, and when she looks in the mirror she sees two left arms. She knows she is looking at her right arm, but says it looks like her left arm. She also said that when she is touching her right arm it feels like she is touching her left arm.
Neuroplasticity

- Swelling and pain evoked by movement of the CRPS-affected limb is more severe if patients view a magnified image of the limb; if it looked bigger, it hurt more and became more swollen.
Neuroplasticity

• The perceptual disturbances in CRPS similar to disturbances associated with unilateral neglect after stroke.
• Patients can perceive touch on the affected limb if they watch the mirror image of the unaffected limb being touched.
• Acerra & Moseley 2005 Neurology 65; 751-753

Neuroplasticity

• Patients perform poorly on tasks in which they are required to judge the laterality of a pictured limb.

Neuroplasticity

• Recent work has suggested that cold-type CRPS is associated with a cold side of space—i.e., crossing the arms so that the healthy hand is on the affected side of the midline reduces the temperature of the healthy hand.

Neuroplasticity

- Cortical changes also affect the primary motor cortex in patients with CRPS.
- Decreased inhibitory mechanisms and increased excitability in the contralateral primary motor cortex in patients with CRPS.
- Widespread impairment of central motor processing in CRPS.

Summary

1. High levels of acute pain, immobilization and increased inflammation seems to predict CRPS development.
2. With the above factors, there is likely an immediate up-regulation of the peripheral and central nervous system. Thus, nerves become extra sensitive.
3. Nerves fire both ways.
4. Nerves firing down to the extremity produce increased inflammatory and immune cells, which causes swelling, heat and sensitivity. These changes likely influence the sensory and motor maps in the brain. The brain sees the hand/foot altered and becomes concerned about limited hand/foot use.
5. The peripheral nervous system fires into the CNS, causing long-lasting neuroplastic events culminating in central sensitization. This additionally confuses the brain, leading to changes in the maps related to the specific body part.
6. There is an immune response in the blood-spinal cord barrier which alters the brain’s view of the hand/foot.
7. Thoughts are nerve impulses and the impulses drive both orthodromic and antidromic (retrograde) depolarization, leading to persistent inflammation and central nervous system plasticity changes.

Summary
Summary

8. The whole brain processes all the danger and thus, busy with pain, facilitates changes in various output systems, including motor control, immune and endocrine system.

9. Maps of the body parts are altered in the brain (CNS, visual, peripheral and immune system altering the blood-brain barrier), resulting in the extremity looking different, usually larger, and problems discerning left and right.

10. With increased confusion, the brain bring in its most potent defender: PAIN.

Treatment...

- Of the 3 major pathophysiological pathways it seems neuroplasticity may be a BIG PART:
  1. Aberrant inflammatory mechanisms
  2. Vasomotor dysfunction
  3. Maladaptive neuroplasticity

**Altered Body Maps**

How to identify smudging

- AMRI, PET, TMS, MEG in a research lab
- In a clinical setting:
  - Draw a person [modified]
  - Complex sensory testing
  - Laterality or mental rotation or left-right discrimination (motor imagery & its components)
  - TPD

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**Two Point Discrimination**

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**Pressure Pain Thresholds**


Localization testing

- Where was I touched? (and where was it that I hurt?)
- Stimulus from clinician then identification from patient:
  - Grid identification
  - Tactile localization (measure error distance)
- Impaired tactile acuity relates to impaired motor control

Left/Right Discrimination/Laterality

Site-specific disruption in left right judgments have been reported with:
- Amputees
- CRPS
- Chronic back pain
- CTS
- Knee OA
- Arm and hand pain
- Dystonia
- Post stroke
- Expectation of pain
- Radiculopathy

Laterality impairment
What’s normal?

- Accuracy of >80%
- 1.6 sec +/- 0.5 for necks and backs
- 2 sec +/- 0.5 for hands and feet

1.6 sec +/- 0.5 for necks and backs
2 sec +/- 0.5 for hands and feet

Chronic situation might be opposite

Treatment

Treatment: Fundamental Flaw

As long as the brain does not understand the pain and does not even know what left and right are, there is no incentive to reduce its most potent protector:

PAIN
**Classic Rehabilitation**

- Do part of movement but no painful part
- Do part of movement with painful part
- Do more
- Increase number
- Increase strength
- Add equipment

**Treatment: Fundamental Flaw**

As long as pain and sensitivity is so high, the extremity does not want to be touched, moved or used.

**In Central Sensitization...**

- **Phase 1:**
  - Decrease sensitivity and pain
- **Phase 2:**
  - Move towards function, movement, goals, etc.
Proposed Treatment

- Theraeutic Neuroscience Education
- Graded Motor Imagery
- Sensory Discrimination
- Regular Therapy
  - Sensory integration
  - ROM
  - Function
  - Etc.

So the brain understands

Restoring the extremity/body part back in the brain

Function/ROM

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Our recommendation

Sequence Matters:
1. Therapeutic Neuroscience Education
2. Left/Right Discrimination
3. Motor Imagery
4. Sensory Discrimination and Graphesthesia
5. Mirror Therapy

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Origins of Neuroscience Education

Pain, the Tissues and the Nervous System: A conceptual model

Physiotherapy, January 1998, vol 84, no 1

Louis Gifford

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Neurophysiology of pain
No reference to anatomical or patho-anatomical models
No discussion of emotional or behavioral aspects to pain
Noception and nociceptive pathways
Neurons
Synapses
Action potential
Spinal inhibition and facilitation
Peripheral sensitization
Central sensitization
Plasticity of the nervous system

Section 12. The Brain's Body Maps

- Neuroplasticity
- Homunculus
- Sensory cortex
- Phantom limb pain
- Complex regional pain syndrome
- Use it or lose it
- Spreading pain
- Neglect
- Laterality
- Smudging
- Graded motor imagery
Phase 2: Laterality

Left/right discrimination is the accuracy and speed of identifying whether a picture or body part is a right or left.

Phase 2: Laterality

Cards – Internet Images
**Phase 2: Laterality**

- Magazines
- Make flash cards
  - Internet images
  - Take photos
  - Make a PowerPoint
- noigroup.com
  - Flashcards
  - Online
  - Smartphone publications

**Phase 2: Laterality**

- Emotions
- L/R should be continued
- Injury, pain and immobilization

**Phase 2: Laterality**

- GOAL:
  - Under 3 seconds
  - > 80% accurate (? 90)
- Hands versus feet
- Dyslexia
- 10-20% no shift; move on
Hello Again!

First, I'd like to say thank you for the guidance you've given me already; second I'd like to say that I've had success in treating my patient with CRPS, but now I am not sure how to proceed.

Here's the quick story, we started with looking at pictures of feet—playing games with the pictures (matching, sorting, etc.)... and on day 5 the patient woke up with almost total resolution of his pain. He was down to a 2/10. The pain remained low, and by the next day he had 0/10 pain. Yippee!

Phase 3: Imagery

• Why do you yawn, when you see someone else yawning?

• Easiest way to start urinating?

Evidence


Narrative synthesis of the results, based on effect size, found there was good to very good quality level II evidence that graded motor imagery is effective in reducing pain in adults with CRPS-1, irrespective of the outcome measure used.
Phase 3: Imagery

- Imagine extremity (see it visually)
  - Static
  - Dynamic
  - Doing tasks
- Giving the brain map exercise without moving the extra sensitive extremity

Phase 3: Imagery

- Been used for years in sports
- Very important potential role in acute/severe pain

Phase 3: Imagery - Example

- Ask patient for top 10 tasks needed to be performed with painful extremity
- Find 10 such activities/images on the Internet
- Draw card; imagine static hand in that position
- Imagine moving hand to similar position
- Imagine performing the task
- Repeat; repeat; repeat
Phase 3: Imagery - Example

CRPS Foot

Phase 4: Fundamental flaw

4. Sensory Discrimination

Why discrimination and not integration?

Tactile discrimination, but not tactile stimulation alone, reduces chronic limb pain

G. Lorimer Moseley, Nadia M. Zochowski, Kerjia Wächter
If you know your neighbors, you can find your house.
4. Sensory Discrimination

- Patient Example
- CRPS Face

4. Sensory Discrimination

- Patient Example – Hypersensitive feet s/p MVC

4. Graphesthesia training
- **Beans**
  - Introduce (visual) the sensation of beans
  - Deliberate
  - Close eyes – form a memory

- **Add other objects**
  - Teach what they should feel like
  - Into the beans...go hunt!
If it was only this simple

- Using mirrors to trick the brain
- Have to restore L and R first
  - If not = confusion = more pain
- Slowly expose the patient to image

Phase 5: Now...Mirror Therapy

- If it was only this simple
Phase 5. The BOX

- Sturdy (use a new box or reinforce)
- No distortion
- Avoid images/writing on the box
- Perspex mirror (otherwise 7 years of CRPS)
- Cheap

Phase 5: Mirror Therapy

5. Principles

- Prepare the patient
  - TNE
  - Emotional
- Sit evenly
- No jewelry
- ? Cover tattoos
- Outside (normal) hand works
  - If bilateral: Least affected = normal side
Phase 5: Mirror Therapy – Ideas

Mirrors for feet
There is good evidence for the use of mirror therapy alone for acute CRPS (McCabe, Haigh et al. 2003; McCabe, Haigh et al. 2004).

There are case reports of successful mirror therapy management of CRPS (Karmarker and Lieberman 2006) and post-hand surgery pain (Rosen and Lundborg 2005).

Benefits of mirror box therapy with cognitive behavioural therapy demonstrated in three patients with CRPS1. (Vladimir Tichelar, Geertzen et al. 2007)

It works best if carried out in the sequence of:

- Laterality recognition
- Motor imagery
- Mirror therapy

Our Recommendation

Review of the sequence:
1. Therapeutic Neuroscience Education
2. Left/Right Discrimination
3. Motor Imagery
4. Sensory Discrimination, Localization and Graphesthesia
5. Mirror Therapy

CRPS Treatment:
NOW – The stuff we used to do...

- Therapeutic Neuroscience Education
- Graded Motor Imagery
- Sensory Discrimination
- Regular Therapy
  - Sensory integration
  - ROM
  - Function
  - Etc.
Case Study

- 21-year old collegiate soccer player, majoring in education
- Right wrist sprain 5 months ago
- X-rays (-); wore brace x2 weeks
- c/o constant burning pain, 9/10, in hand, wrist and forearm
- Persistent edema, redness
- “Feels like a club, not a hand”
- Poor tolerance to clothing touching wrist/forearm
- Cannot write or type with right UE
- Schoolwork suffering, concerned she will lose scholarship
Is this CRPS?

1. Dominating pain, which is disproportionate to any testing event
2. Sustained period of at least one symptom in three clinical diagnostic criteria or in four research diagnostic criteria of the following categories:
   - Tactile hyperesthesia or allodynia
   - Reaction to temperature changes, skin color changes, or skin color asymmetry
   - Subdural or subcutaneous edema, swelling changes, or swelling asymmetry
   - Motor or sensory decreased range of motion, motor dysfunction (weakness, taper, or spasticity), or sensory changes (hypoesthesia, hypesthesia, or analgesia)
3. Most display or at least one sign in three of diagnostic criteria 1 or none of the following categories:
   - Decreased sympathetic or parasympathetic activity in light touch, deep sensitivity
   - Pressure, or joint movement
   - Swelling or subcutaneous edema, skin color changes or asymmetry
   - Subdural or subcutaneous edema, swelling change, or swelling asymmetry
   - Motor or sensory decreased range of motion, or motor dysfunction (weakness, taper, or spasticity), or sensory changes (hypoesthesia, hypesthesia, or analgesia)

What will you measure?

Treatment: Where will you start?
How will you progress?

Still too hot to handle, but laterality improving…what next?

What else?
Last steps…

Always keeping in mind:

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