Teaching People About Pain

The Neuroscience of Pain

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Pain is 100% from the brain...

Pain is a multiple system output, activated by an individual’s specific pain neural signature. The neural signature is activated whenever the brain perceives a threat.

Pain is produced by the brain...

- Altering information the brain receives can potentially alter the pain experience

Traditionally...

Adapted from Gifford, L.S., Pain, the tissues and the nervous system. Physiotherapy, 1996. 82, p. 27-33.
What about a top-down approach?

Example # 1

• Is Lumbar Radiculopathy an indication for Surgery?

• **YES**

Diagnosing Radiculopathy

- Straight Leg Raise – very high sensitivity in ruling out LR


SO...Theoretically – who needs surgery?
But…


Example 2
The Efficacy of Sham Surgery in Orthopedics: A Systematic Review of the Literature*
Louw A, Diener I, Puenteura L and Fernandez de-Las Penas C.
Submitted for Publication 2012 - 2014

* Rejected by all major spine and orthopedic journals
The Efficacy of Sham Surgery in Orthopedics: A Systematic Review of the Literature*

Traditional Education Models...
Very prevalent models...

- Prevailing biomedical models focus on tissues and tissue injury.
- Orthopedic-based professions such as physical therapy commonly use anatomy and patho-anatomy based models to explain pain to their patients.

The problem with these models: 1

- Not only have these models shown limited efficacy in decreasing pain and disability, but they may increase fear in patients, which in turn, may increase their pain.


The problem with these models: 2

- People in pain want to know more about pain…NOT anatomy, biomechanics or patho-anatomy

- It cannot explain complex pain states


The problem with these models: 3

- Our models of pain are outdated and wrong


NOW: Update your knowledge of pain
Would this hurt...?

Pain is produced by the brain based on perception of...THREAT
Pain Mechanisms

OUTPUTS
• Pain
• Action programs
• Stress regulation

PROCESSING via BODY-SELF
NEUROMATRIX:
• Sensory
• Cognitive
• Affective

Tissues
Environment

Input: Tissues

Gifford, L.S., Pain, the tissues and the nervous system. Physiotherapy, 1998. 84: p. 27-33.

Gifford, L.S., Pain, the tissues and the nervous system. Physiotherapy, 1998. 84: p. 27-33.
Input: Neuropathic

Gifford, L.S., Pain, the tissues and the nervous system. Physiotherapy, 1998. 84; p. 27-33.

1. Ion channel expression

Louw, Puenteedura: Therapeutic Neuroscience Education 2013 OPTP
Unhealthy Peripheral Nervous System:
Physically and Emotionally

Bombardment of C-fiber activity into the CNS dorsal horn resulting in permanent changes over time

Retrograde firing of nerves resulting in increased inflammation, swelling and immune responses

Unhealthy Peripheral Nervous System: Physically and Emotionally

Processing: CNS

Tissues
Environment
Sample
**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>Alldynia</td>
</tr>
<tr>
<td>Upregulation 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>Alldynia and Hyperalgesia</td>
</tr>
</tbody>
</table>
Decreased endogenous mechanisms = Increased sensitization/pain experience

How does the brain process information?
The Brain’s processing during a painful experience

- Common areas are frequently “ignited”
- Via connections, backfiring neurons, and neurotransmitters, pain is perceived – the pain neural signature

A TYPICAL PAIN NEURAL SIGNATURE

1. PREMOTOR/MOTOR CORTEX
   organize and prepare movements

2. CINGULATE CORTEX
   concentration, focusing

3. PREFRONTAL CORTEX
   problem solving, memory

4. AMYGDALA
   fear, fear conditioning, addiction

5. SENSORY CORTEX
   sensory discrimination

6. HYPOTHALAMUS/THALAMUS
   stress responses, autonomic regulation, motivation

7. CEREBELLUM
   movement and cognition

8. HIPPOCAMPUS
   memory, spatial recognition, fear conditioning

9. SPINAL CORD
   gating from the periphery

But...there's more complexity

Denotes synaptic modulation

A neuroscience approach to managing athletes with low back pain
Emilio J. Puentedura a,b,*, Adriaan Louw b
“My pain is not your pain....”

Beliefs
Knowledge, logic
Social context
Anticipated consequences
Other sensory cues
Physical therapy

End Result...How Dangerous is this?

Louw, Puente: Therapeutic Neuroscience Education 2013 OPTP
Homeostatic shift for survival

Louw, Puente de: Therapeutic Neuroscience Education 2013 OPTP
Adrenaline
- Adrenal medulla

Cortisol
- (Hydrocortisone)
- Adrenal cortex
**ACTH**

- **Tissues**
  - Sore
  - Tired
  - Sensitive
  - Fatigued

- **Brain**
  - Memory
  - Sleep
  - Concentration
  - Blood pressure
  - Reproduction
  - Other

**Cortisol**

- **Immune**
  - Cytokine signaling
    - IL – 1
    - IL – 6
    - TNF- \( \alpha \)
  - Increased nerve sensitivity
  - Persistent inflammation
  - Brain plasticity
We’ve come full circle…

OUTPUTS
• Pain
• Action programs
• Stress regulation

PROCESSING via BODY-SELF
NEUROMATRIX:
• Sensory
• Cognitive
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Gifford, L.S., Pain, the tissues and the nervous system. Physiotherapy, 1998. 84: p. 27-33.

Teaching people about pain: the origin


Since then...

Interest in pain science and manual therapy
- Gifford
- Butler

Secondary studies of TNE of other conditions: chronic whiplash, chronic fatigue syndrome, fibromyalgia, case studies on content, more CLBP, post-op lumbar surgery
- Meuris, Nijs, Ryan, Louw and Puntedura

Various RCT's by Mosely to determine effectiveness of TNE on chronic LBP
- Pain, Cognitions, Fear, Movement, Brain Activation, Catastrophization, Knowledge of Pain

Two systematic reviews on TNE
- Clark
- Louw

Ongoing research into uses of TNE for chronic pain
- TNE as preemptive treatment to prevent chronicity (Louw)
- Lumbar Surgery
- Whiplash
Emerging research shows that explaining to patients their pain experience from a biological and physiological perspective of how the nervous system/brain's processes pain allow patients to move better, exercise better, think differently about pain, push further into pain, etc.

**Conclusions:** For chronic MSK pain disorders, there is compelling evidence that an educational strategy addressing neurophysiology and neurobiology of pain can have a positive effect on pain, disability, catastrophization, and physical performance.


**TNE: Adjunct treatment**

- Manual therapy including spinal mobilization and manipulation
- Soft tissue treatment/massage
- Neural tissue mobilisation
- Spinal stabilisation exercises
- Home exercises
- None (neuroscience education only)
- Circuit training
- Aerobic exercise


**TNE: Content**

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

Clinical Example

- Patient with 3 years of LBP
- High fear
- Limited motion – afraid to move
- Failed treatments
- Various explanations
- Stopped working
- Spreading and worsening pain

Nerves work like an alarm system

Normal electrical activity

Electrical activity "waking up"

Persistent pain

End-Result

Turning the alarm system down therapeutically

- Therapeutic Neuroscience Education
- Aerobic Exercise
- Manual Therapy
- Breathing, relaxation, mediation
- Modalities
- Etc.
After TNE reconceptualizing pain

- After TNE: Tissues heal; tissues sensitive; sore and deconditioned

Threat smaller
- Won’t hurt tissues

Subsequent therapy

- 2x/week – total of 8 visits
- Session 30-45 minutes
- Exercise
- Pacing and graded exposure
- Constant pain education – more in-depth including:
  - The Brain and Pain
  - Output mechanisms and Pain
- Home Exercise Program
- Goal Setting

Summary: Chronic


• Changes in regards to pain beliefs
• Changes in regards to attitudes
• Improved cognition
• Improved physical performance
• Increased pain thresholds
• Improved outcomes from exercise
• Decreased brain activation
• Improved function
• Patients able to take on complex pain issues

What about TNE for Acute MSK Pain?
Immediate Effect of Preoperative TNE for Lumbar Radiculopathy: Case Series


- Physical Measurements after TNE-only (48-hours preop):
  - Passive SLR increased 9°
  - Active trunk flexion increased 5cm
  - Similar outcome as chronic LBP studies

Preoperative TNE for Lumbar Radiculopathy: A Multicenter RCT

Submitted – Louw et al 2014

- 67 Patients scheduled for L-Surgery
- Randomized
- 35 Surgeon Education
- 32 Surgeon Education + Neuroscience Ed
- Low Back Pain & Leg Pain
  - Oswestry
  - Fear Avoidance
  - Pain Catastrophization
  - Pain Knowledge
  - Surgery Experiences
  - Cost Analyses

1, 3, 6 and 12 months postop
Preoperative TNE for Lumbar Radiculopathy: A Multicenter RCT

Submitted – Louw et al 2014

- One year follow-up (superior results):
  - Back Pain
  - Leg Pain
  - Catastrophization
  - Fear Avoidance
  - Pain Knowledge
  - Satisfaction with surgery
  - 42% healthcare savings

Why is TNE effective?

- Redefine pain and thus change cognitions regarding pain
- Pain and Tissue injury are two different things
- Reduces threat
TNE Evidence

- We TREAT pain; not merely MANAGING it…


Thank you & acknowledgements…

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- Colleen, Hailey and Samuel Louw
- Ina Diener
- Louis Gifford
- Louie Puenteleda
- Lorimer Moseley
- David Butler
- Merrill Landers
- Steve Schmidt
- ISPI staff and faculty