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**Session 206: Combining Mirror Visual Feedback & Graded Motor Imagery with Neuroscience Education**

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**BRAIN**

**NEUROPLASTICITY**

- Persistent anatomical or physiological changes in a neuron that occurs during development, regeneration, experimental manipulation or repeated activity across a synapse
- Throughout life, the brain is able to restructure itself to change by adapting
INJURIES

- Leads to biomechanical and central nervous system changes
- Decrease afferent input to somatosensory area due to injury
- Have mechanical instability and nervous system deafferentation
- Treatment must include neuro-education, neuro-cognition, visual input

Evidence from neuroscience, motor control, and psychology

UNDERSTANDING THE BRAIN CHANGES FOLLOWING INJURY
IMMOBILIZATION, PERSISTENT PAIN, AND INFLAMMATION.

DORSAL HORN - SPINAL CORD

PAIN IS AN OUTPUT OF THE BRAIN – INTIMATE RELATIONSHIP

Close allies – Brain is Protector
Both depend on brain map of body

Moseley G.L. et al Arthritis Rheum; 2008; May 15:59 (5) 623-31
PAIN AND THE BRAIN
INTIMATE RELATIONSHIP

PAIN
PAIN IS NOT A SIMPLE PROCESS AND IT IS NOT A DIRECT RESPONSE TO INJURY - PATHOLOGY.

NEUROMATRIX MODEL OF PAIN:
PAIN IS AN OUTPUT

Models the theory that the brain has a neural network that integrates information from multiple sources to produce the "experience that is labeled pain".

Melzack R. From the gate to the neuromatrix. Pain, 1999.
NEUROMATRIX MODEL OF PAIN

Models the theory that the brain has a neural network that integrates information from multiple sources to produce the “experience that is labeled pain”.

This is the brain's coding space

The brain response according to the threat.

Melzack R. From the gate to the neuromatrix. Pain, 1999.

THE BRAIN

- Protector of the body
- It decides what to do and if there is danger
- It meets with all the brain parts and can inhibit or facilitate pain signals

PERSISTENT PAIN AND SYMPTOMS

- THIS IS A DISEASE OF THE BRAIN

THE END
DARK SIDE OF PLASTICITY—MALADAPTIVE NEUROPLASTICITY

- Changes in CNS function compared to non injured
- Longer you have pain the better your system gets at producing it.
- Pain is triggered more easily
- Painful events such as hyperalgesia become more painful
- Actual increase sensitivity with decreased precision
- Abnormal intracortical inhibitory mechanisms

HOMONCULUS

Brain areas normally devoted to specific body parts or functions start to overlap. In the motor cortex this may make it more difficult to isolate and move that body part, in the sensory cortex too sensitive to move, perhaps as protective strategies.

SMUDGING
PERSISTENT PAIN AND/OR SYMPTOMS

- Brain develops a fuzzy view -
- The more cortical disorganization the greater the symptoms
- Correcting the brain's view of the image helps to decrease symptoms.

PAIN INTERACTION BY MOSELEY

- Interaction of multiple things so pain is real
- Between environmental events, cognitions, behaviors including the proposition that symptoms and dysfunctional behaviors are often cognitively mediated.
- Can be improved by modifying problematic thinking and inaccurate beliefs.
- Pain itself is modulated by beliefs and appears fundamental to the idea that pain is a biopsychosocial phenomenon.

BIO-PSYCHO-SOCIAL EDUCATION

- All areas of brain involved - basic protection
- Brain determines if pain or symptoms are a threat
- Representation of pain in the brain when there is no tissue damage
- Beliefs, knowledge, social context, culture and work environment
The mechanisms of cortical plasticity, according to current and widely accepted opinions, involve the unmasking of previously ineffective connections or the sprouting of intact afferents from adjacent cortical or subcortical territories. Although significant strides have been made in our understanding of cortical plasticity following nerve transfer and during motor relearning, a great deal remains that we do not understand. Cortical plasticity and its manipulation may one day become important contributors to improve functional outcome following nerve transfer.

With increasing clinical experience, peripheral nerve surgeons have come to appreciate the important role that cortical plasticity and motor relearning play in functional recovery following a nerve transfer. Neurostimulation (transcranial magnetic stimulation), and neuroimaging (functional MRI, structural MRI, magnetoencephalography) measure different aspects of cortical physiology and when used together are powerful tools in the study of cortical plasticity.

What is fMRI?

Functional MRI is based on the increase in blood flow to the local vasculature that accompanies neural activity in the brain (Functional Brain Imagery). Magnetic resonance imaging can be used to map changes in brain hemodynamics that correspond to mental operations of neural activity as detected by a blood oxygen level dependent signal.
ROLE OF MIRROR NEURONS

- 1996: Mirror neurons fire when observing or watching an activity by Gallese, Fadiga et al.
- Respond to non-visual stimuli such as the noise of an action and imagination of an action.
- Emotional events activates the brain circuitry – someone you love is in pain by Swinger and Seymour et al. 2006.

Mirror Neuron Role in Rehab

- Improve motor performance by using visual & motor imagery.
- Motor imitation and motor execution excite the corticospinal pathway.

MIRROR NEURON CELLS

- Active during execution and observation of an action.
- Monkeys: Object directed actions such as grasping, tearing, manipulating, holding.
- Monkey observed someone else.
- Role in understanding interventions of other people.
NEURAL MECHANISMS OF PAIN

PERIPHERAL AND CENTRAL NERVOUS SYSTEM

PATHOPHYSIOLOGY OF PAIN MECHANISMS

Nociception
Peripheral Neuropathic
Neurogenic
Central Sensitization
PAIN SYSTEMS

- Sensitive enough to detect harmful stimuli
- Too sensitive causing pain that provides no benefit
- Adaptive response occurs after injury
- Pain pathway increase in sensitivity.
- After body has healed no value so manifestation of pathological changes in nervous system

EVIDENCE BASED SIGNS AND SYMPTOMS

NOCTOCEPTIVE PAIN
Pain localized to the area of injury or dysfunction
Clear, proportionate mechanical/anatomical nature to aggravating and easing factors
Usually intermittent and sharp with movement or mechanical provocation
Dull ache or throb at rest

NOCTICEPTION: THE SIMPLEST PATH

- Everyday experience that occurs in discomfort in response to simple insult or injury
- Protective state that warns us to move away from the cause and take care of the trauma
- Afferent nerve transfer sensory input from PNS to CNS
- When information transferred to parts of the brain responsible for perception the actual sensory experience occurs
NEUROGENIC PAIN
Pain referred in a dermatome or cutaneous distribution
History of nerve injury, pathology or mechanical comprise
Pain/symptoms provocation with mechanical/movement test – Neurodynamic testing

EVIDENCE BASED SIGNS AND SYMPTOMS

PAIN DRIVER HAS MOVED INTO THE CENTRAL NERVOUS SYSTEM

CENTRAL SENSITIZATION

NERVOUS SYSTEM AND MECHANISM OF PAIN SENSATION

EVIDENCE BASED SIGNS AND SYMPTOMS

CENTRAL SENSITISATION
Disproportionate non mechanical, unpredictable pattern of pain provocation in response to multiple/nonspecific aggravating/easing factors
Pain disproportionate to the nature and extent of injury or pathology
RECOGNITION OF CENTRAL SENSITIVITY

- Localized pressure pain threshold
- Often unrelated to primary pain source
- Hypersensitivity to pain, noise, smell, chemical stimuli, cold / heat, electrical stimuli, stress and emotions
- Widespread symptoms but not segmentally related
- Fatigue and sleep disorders

CENTRAL SENSITIZATION FOLLOWING NERVE INJURY

- Following peripheral inflammation and nerve injury there is a change in some dorsal root neurons causing non-nociceptors to induce central sensitization.
- This results in light touch inducing a progressive tactile pain hypersensitivity which can last for hours.
- Activated microglia in dorsal horn fire thus causing additional neuropathic pain
- All the above changes the somatosensory and motor cortex

ABNORMAL PAIN STATE

Allodynia
- Pain response to non-noxious stimuli

Hyperalgesia
- Exaggerated or spontaneous response to noxious stimuli
HYPERALGESIA AND ALLODYNIA

WHAT HAPPENS IN CENTRAL SENSITIZATION

- Exhibit lower pain threshold due to altered Central Processing
- Produces pain hypersensitivity by changing the sensory response elicited by normal input
- Net effect of Central Sensitization - recruiting sub-threshold synaptic inputs to NOCICEPTIVE NEURON GENERATORS
- Pain memories which fire erratically so maladaptive pain

BRAIN PLASTICITY AND CENTRAL SENSITIZATION

- Central Sensitization results in brain changes in response to repeated nerve stimuli.
- Levels of neurotransmitters and brain electrical signals change as neurons develop a memory for those signals.
- Brain is activated or sensitized by previous stimuli to become more excitable
- Brain has a memory even after painful stimuli is removed.
- Entrapment neurpathies follow surgery still causes pain due to pain memory
SUMMARY

- With Central Sensitization tissue injury leads to a constellation of changes in spinal excitability. Which includes elevated spontaneous firing, increased response amplitude and duration decreased threshold, enhanced discharge to repeated stimuli and expanded receptive fields.
- Clinically the injury has healed but the PAIN persists.

Woolf, CJ, 2011 Pain 152;S2-S15

CONSIDERING CENTRAL SENSITIZATION

-KISS METHOD When a patient isn’t getting better based on tissue healing and expertise look immediately to central nervous system instead of questioning your competence in assessment and treatment of mechanical tissues.

WHAT DO THESE HAVE IN COMMON
Mismatch between Motor Output and Visual Feedback
Tx: Make the brain think there is not tissue damage

CNS AND PAIN

PAIN: CNS, SYMPATHETIC AND PARASYMPATHETIC CHANGES
Neurons that are wired together fire together to produce an output. Many possible neurotags depending on what the brain has stored from its past experience.

Neurotag is network of interconnected neuron/brain cells that are distributed throughout brain. Neurotag is activated and produces output. Example: smells of brownies cooking.

Multiple brain connections.
BRAIN INVOLVEMENT

- Cerebellum - movement and cognition
- Hippocampus - memory, special cognition, fear conditioning
- Spinal Cord - gating from the periphery

BRAIN INVOLVEMENT

- Premotor/motor cortex - organize to prepare movement.
- Cingulate cortex - concentrating and focus
- Prefrontal cortex - problem solving and memory

BRAIN INVOLVEMENT

- Amygdala - fear, addiction and fear conditioning
- Sensory cortex - sensory discrimination
- Thalamus and Hypothalamus - stress response, autonomic and motivation
PERSISTENT PAIN AND INFLAMMATION

Pain without obvious accompanying tissue damage
Might be caused by discordance between motor intent and movement.
This hypothesis is the same way that motion sickness
Might result from discordant sensory input between BODY AND BRAIN.

SENSORY MOTOR INCONGRUENCE

Vestibular system and proprioceptors.
Pain may result from changes in cortical representation
Of somatic input which falsely signals incongruence between motor intention and movement.
PAIN RESULTS FROM A BRAIN OUTPUT
BRAIN DECIDES IS THIS A THREAT
MULTIPLE FACTORS GENERATE PAIN AND CAN BE MANIPULATED TO CHANGE AND MODULATE.
PAIN IS NOT JUST A NOCICEPTIVE SENSATION

Why does local injury from trauma lead to intractable pain in some patients?
Who do some patients have an inflammatory process that persists?
Why does some pain respond to anti-inflammatory drugs whereas other types require opiates?
Pain is NEUROIMMUNE PLASTICITY
Glia cell involvement

ALTER THE THREAT SINCE IT IS MALADAPTIVE COGNITIVE PROCESS
PAIN IS DEPENDENT ON AN EVALUATION, CATASTROPHIZING IS THOUGHT TO WORSEN PAIN BY ENHANCING THE NEURAL MECHANISMS.
PRELIMINARY EVIDENCE FOR A CAUSAL EFFECT OF CATASTROPHIZING ON PAIN AND FUNCTION (Terry Thompson)
**TREATMENT MECHANISMS**

- MECHANISMS BY WHICH PAIN RECONCEPTUALIZATION RESULTS IN LESS PAIN AND BETTER FUNCTION ARE UNKNOWN. (LEE, MOSELEY; PAIN 2016)
- VALUE OF A NOXIOUS INPUT BY CHANGING THE MEANING - NEUROSCIENCE EDUCATION
- REDUCE CATASTROPHIZING

**PATIENTS THAT DON'T GET BETTER**

**WHY**
- Three significant psychosocial reasons:
  - Fear avoidance
  - Kinesiophobia
  - Catastrophizing

**BIO-PSYCHO-SOCIAL**

Provide education that knowledge and movement are the greatest pain and stressor liberators. David Butler

Educate, Educate, Educate

BUTLER, D AND MOSELEY, L: EXPLAIN PAIN
BIOPSYCHOSOCIAL APPROACH

- Persistent pain is a massive burden throughout the world.
- Management of symptoms must be understood.
- Provides patients with knowledge, understanding, and skills to reduce pain and disability.
- Brain changes are maladaptive neuroplasticity.

Pain is clearly related to exogenous (external) and internal (endogenous stimuli). These relationships are modulated by other sensory inputs such as cognitive, emotional, and social factors.

NEUROSCIENCE EDUCATION

- Goals
  - Help patients understand brain processes and perceive pain and how persistent pain alters the structure and function of the nervous system.
  - Research has shown short term reduction in pain and disability. This represents the biological info that justifies a biopsychosocial approach.

MIRROR NEURON CELLS

- Greatest number located in premotor cortex and inferior parietal lobes.
- Monkey – premotor cortex
  - Discharge when performer a given motor act and when observe the same motor act
- Human – ample evidence
  - Cortical network that discharges in some way – observing and executing movement.

Cattaneo L, Rizzolotti G. 2006
Mirror Neuron Role in Rehab

- Improve motor performance by using visual & motor imagery
- Motor imitation and motor execution excite the corticospinal pathway

NEUROPLASTICITY
Retrain the Brain

- Repetitions
- Challenging
- Pacing

Rewiring occurs when new connections (synapses) are formed

KEEP THE AMYGDALA UNDER CONTROL

- THREAT
- FEAR
- STRESS
- ANGER
THERAPEUTIC GOAL

- Lower the threatening inputs - fear avoidance model
- Bottom up - structural pathology model
- Top down - education to understand that pain doesn’t mean harm and treat cortical reorganization

TREATMENT

- Mirror therapy and Graded Motor Imagery for non fearful intervention by using vision of uninvolved part
- Treat from the “TOPDOWN” as well as “BOTTOM UP”

THE CLINICAL APPROACH FOCUSES ON:

- Decreasing all inputs that imply that body tissue is in danger
- Then on activating components of the pain neuromatrix without activating its output
- Rehabilitation progresses to increase exposure to threatening input across sensory and non-sensory domains.”

1996: Ramachandran on phantom pain
1999: Altschuler and Ramachandran on stroke
1999: Ramachandran et al. on hemi neglect
2003: McCabe et al. on CRPS Type I
2004: Moseley et al. on CRPS as part of graded motor imagery

2005: Rosen et al. on hand surgery
2008: Selles et al. on CRPS Type II
2009: Merzenich, Brain Plasticity
2014: Mirela, C. Stroke
2015: Jin-Young Park et al. UE stroke
MIRROR THERAPY

- Cha H, Oh D, Effects of mirror therapy integrated with task-oriented exercise on the balance of patients with post stroke; Int J of Rehab Res; 2016
- Amasyali S, Yalım A, Comparison of the effects of mirror therapy and EMG triggered NMS on hand function in strokes; Int J of Rehab Res Dec 2016

One method that has been used to activate cortical network representations

Theory:
- Reconcile motor output and sensory feedback (Ramachandran 1995)
- Activates pre-motor cortices which is associated with activation of the visual processing areas. (Seitz 1998)
Ramachandran hypothesized that the disruption of the normal interaction of motor intention to move the limb and the absence of appropriate sensory feedback resulted in phantom limb pain.

They speculated that visual feedback would interrupt this pathological cycle.

Perception that limb is normal
- cramped position goes away
- prompts awareness
- decreases pain and unpleasant sensation
- decreases kinesiophobia
- decreases telescoping with PLP

Touching specific areas on the face of a person with an amputated arm will often evoke precisely localized sensations in the fingers.

PHANTOM LIMB

- 3 weeks post amputation upper extremity – sensations from ipsilateral face are referred to the amputated limb
- Ice will elicit cold
- Vibration will elicit vibration

- This effect caused by sensory input from face invading and activating deafferented hand zones in cortex and thalamus

- See that the phantom limb is moving in response to brain command from the non-involved side.

Using Mirror Therapy to Improve Sensorimotor Recovery After Stroke: Current Evidence & Clinical Considerations

- Analysis of 14 Studies: 12 RCTs & 2 cross-over designs; total of 567 participants
- Participant Characteristics:
  a) Mean age range: 51-79 years
  b) 55% left hemiparesis
  c) 57% female
  d) Mean time post-stroke range: 5 days – 5 years
  e) Stage of Recovery: Acute/sub-acute (4 studies)
     Chronic (8 studies)

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GRADED MOTOR IMAGERY

- GMI is a rehabilitation brain based treatment used to treat pain and movement dysfunction.
- The dysfunction is related to an altered nervous system.
- By exercising the brain in measured and monitored steps as well as progressing to functional activities helps reorganize cortical networks.
Top Down Training

- Laterality stimulates premotor areas
- Visual imagery used for relearning cognitive and planning aspect of movement
- Mirror and motor imagery used to re-educate or retrain the brain for basic motor skills by concentrating on the non-painful movement
- Smooth and controlled movements must act as example for brain to reset circuitry that mediates voluntary movement

GMIP

- Tricks the brain into correcting its distorted image of the body
- Pain results from a mis-match in the way the brain perceives the body and the actual condition of the body
- Brain is tricked into thinking that the limb is actually better than the brain thinks it is

Right/Left Identification

- Hands & feet
- Shoulders and back
Case Study- CVA

Recognise

[Graph showing data]

Recognise

[Graph showing data]
Conscious access to brain

Think - preparation and carrying out movement

Imaging or watching an activity

Start static posture then imagine it moving

Fires areas of the brain that are related to functions of planning and control of movements

Mental Imagery
Capacity to imagine objects or events that are not there

Motor Imagery
Covert Cognitive
Process of imagining a movement of your own body without actually moving your body

Movement Observation
Perception of action of others
IMPLICIT MOTOR IMAGERY

- View pictures of limbs that aren't yours
- Imagine someone else doing a task
- Looking at someone else's limb in the mirror
- Less threatening so start with this

EXPLICIT MOTOR IMAGERY

- Individual is aware of thinking about what they want or are doing. So the term is defined in The Graded Motor Imagery Handbook as EXPLICIT MOTOR IMAGERY

Moseley L, Butler D, Beames G, Giles T
NID Publications 2012

GMI-Convince patients in a non-threatening way that they can move
TIME FRAME GUIDELINES

- Butler and Moseley suggested time frames
- Hands and feet: 2.2-2.4 per picture
- Neck and back: 1.8-2.2 seconds
- Guidelines are not specific to ages
- Correct accuracy is 80-90%
- Look for improvement from baseline test

SCIENCE OF PAIN

- TEACHING PATIENTS THAT PAIN DOESN'T MEAN HARM, YOU SHOULD MOVE DESPITE PAIN, AND THAT PAIN IS UNAVOIDABLE, BUT SUFFERING IS AN OPTIONAL

- SHIFT ONE'S CONCEPTUALIZATION OF PAIN FROM FOCUSING ON TISSUE DAMAGE TO PERCEIVED NEED TO PROTECT TISSUE.

TREATMENT IDEAS

- EDUCATE AND RECONCEPTUALIZE
- BIOPSYCHOSOCIAL APPROACH: KNOWLEDGE, UNDERSTANDING AND SKILLS
- CALM DOWN THE NERVOUS SYSTEM: MANAGE IT
- EVALUATE PATIENTS' BEHAVIORAL ACTIVITIES
- DEVELOP RELATIONSHIP OF TRUST
- REMIND PATIENTS THEY CAN MOVE AND PAIN ISN'T HARM
- REMOVE THE THREAT FEAR BY PROGRESSING FROM NON-THREATENING TO THREATENING
CONSIDERING PAIN NOT AS A MARKER OF INJURY, BUT AS A HUMAN EXPERIENCE, SHOULD NOT BE AN ALTERNATIVE OR NICHE THERAPY, BUT THE VERY THING THAT UNITES US.

OUR ROLE GOOD NEUROPLSTICITY