BCFit 2014: Ground Control

NEUROPLASTICITY
How to gain control of your pain by re-programming your brain

INFANT DEVELOPMENT PATTERNS
Primitive motor pattern generators

GROUND CONTROL
GARGOYLE/BEAR/SJSU/SEESAW/CENTREPOINT

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A brief history of time.
WHAT IS NEUROPLASTICITY?

- Ability to learn with each new experience
- Development of new connections
- Help neurons to form and reform neural pathways, allowing physical changes through learning and stimulation.
"Neuro" for neuron
"Plastic" is for "changeable, modifiable"

Pain inputs can cause changes in the organization in the brain. This can lead to chronic pain, BUT, the brain can often reorganize itself.

Our brain is always bursting with activity, always adapting to our inputs, conscious and unconscious.

The neuron is the fundamental unit of the nervous system. Healthy neurons need 3 major inputs;

1) Oxygen
2) Glucose
3) Stimulation
How does it happen?

• deletes old connections “synaptic pruning,”
• enables the creation of new ones.

• Must be aware of the process because awareness is the key to change.

• Certain neural mechanisms are becoming better understood that are encouraging in regards to learning, memory and pain.

What we’re talking about today;

Pain processing
Sensitization
Brain maps
Reorganization
Things we can do to help
How does this affect us?

• Neuroplasticity is a process that we can influence!

• *Occurs throughout our lifetime.*

• Changes in response to injuries or diseases that cause loss of mental functioning.
Acute pain affects neuroplasticity and sensorimotor integration by decreasing reaction time and accuracy.
There are different types of pain...

<table>
<thead>
<tr>
<th>Nociceptive (tissue) pain</th>
<th>Neuropathic (nerve) pain</th>
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</thead>
<tbody>
<tr>
<td>From tissue damage</td>
<td>Peripheral or CNS nerves</td>
</tr>
<tr>
<td>Somatic and Visceral</td>
<td>Faulty signals sent to brain</td>
</tr>
<tr>
<td>Referred pain</td>
<td>Pain follows nerve distribution</td>
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<tr>
<td>Sharp/dull/ache</td>
<td>Burning or electrical</td>
</tr>
<tr>
<td>Ex: muscle or gall bladder</td>
<td>Ex: sciatica, diabetic neuropathy</td>
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</tbody>
</table>
Pain in 60 seconds...

- Pain receptors stimulated
- Inflammatory mediators released
Pain in 60 seconds...

- Impulse Transmission
- Action potentials are generated
Pain in 60 seconds...

- **Sensory Fibers**
- Action potentials pass along pain nerve fibres (Ad and C)
- **Sensitization** of the pain fibers by inflammation can result in greater, more frequent transmission of action potentials to the nociceptive neurons than in 'normal' pain responses.
Pain in 60 seconds...

- Cord Junctions
- Incoming pain signals (Action potentials) arrive at spinal cord neurons.
Pain in 60 seconds...

- **Dorsal Horn**
- In the spinal cord, incoming information joins the spinal cord neurons in the dorsal horn.
Pain in 60 seconds...

- **Synapse**
- Action potential causes opening of calcium channels in the incoming neuron
- At the synapse level, sensitized incoming sensory fibers cause nociceptor neurons to become hyperexcitable and transmit frequent action potentials
Pain in 60 seconds...

- **Calcium Release**
- Opening of the channels enables the influx of calcium. This signals vesicles containing neurotransmitter molecules to be released in synapse.
Pain in 60 seconds...

- **Synaptic Cleft**
- Neurotransmitter molecules (e.g. glutamate, substance P) diffuse across the synaptic cleft
- Neurotransmitters bind to receptors on attached neuron
Pain in 60 seconds...

- **Post Synaptic Cell**
- Activation of receptors in the post-synaptic membrane, enables the efflux of potassium and influx of calcium and sodium into the post-synaptic cell.
Pain in 60 seconds...

- **Pain Perception**
- Information about pain is received and processed by the higher centers in the brain (thalamus, cerebral cortex) and the individual perceives pain.
- Central sensitization results and the individual perceives greater and more prolonged pain.
- Brain maps? Neurodegeneration?
Pain in 60 seconds...

- Regions where pain is perceived are shown in brown; regions where opioid receptors are found are shown in blue.

Adapted from Purdue Pharma L.P.’s Medical Education Department
http://www.georgiapainphysicians.com/
To reduce the level of perceived pain, opioids are released by interneurons in the dorsal horn in response to severe persistent pain.

These events prevent the transmission of pain to the higher centers. Pain gates.
What do the pain signals do long term?

- **Peripheral Nerves**
- Pain sensations are carried by thinly or non-myelinated nerves. The neurons become unusually sensitive and develop spontaneous pathological activity, abnormal excitability, and elevated sensitivity to chemical, thermal and mechanical stimuli. This phenomenon is called "**peripheral sensitization**".
What do the pain signals do long term?

- **Central Nerves**
- As a consequence of ongoing spontaneous activity arising in the periphery, ascending tract neurons develop an increased background activity, **enlarged receptive field** and increased responses to afferent impulses, including normally innocuous tactile stimuli. This phenomenon is called **central sensitization**. Central sensitization has been proposed as an important mechanism of persistent neuropathic pain.
Central and Peripheral Sensitization?

- Continuous pain signals cause faulty cerebral processing of neuropathic pain.
- Cortical maps are reorganized in sensory or motor areas (highly relevant for phantom limb pain),
- Increased activity in primary nociceptive areas
- Finally, brain imaging is showing significant structural changes suggesting that chronic pain syndromes may be associated with neurodegeneration (Schmerz. 2010).
What’s happening in the brain?

- For people with chronic low back pain, research is showing that the repeatedly firing nerve fibres in peripheral and central sensitization are causing changes in the brain maps of individuals causing **REORGANISATION OF THE MOTOR CORTEX**. Also, it’s showing that training induces **plasticity in the motor cortex** (Journal of Bone and Joint Surgery, 2009).
Re-organizing the brain

- When we perform an activity that requires specific neurons to fire together they release BDNF (brain derived neurotrophic factor),
- growth factor that consolidates neurons to fire together reliably in the future.
- During the critical period in development, BDNF turns on the nucleus basalis, the part of our brain that allows us to keep attention.
Re-organizing the brain

- When the nucleus basalis turns on, it helps us remember what we were experiencing.
- BDNF says "this is REALLY important" and puts brain in plastic state.
- BDNF shuts off nucleus basalis after enough is released during ending effortless learning.
- The nucleus basalis can then only be turned on when something important, surprising or novel occurs, or if we make effort to pay attention.
Dopamine is released to consolidate plastic change. We are rewarded with pleasant sensations when we learn and this begins the cementing-in of neuroplasticity.
The body is also a **map of the brain**.

It tells us where we have motor inhibitions from central sensitization.

If assessed properly, we can analyze the motor dysfunctions and normalize input to re-organize.

Mechanoreception inhibits pain.

If pain is in the brain, then why do I hurt?

“Phantom pain” experiments show that pain is “in” the brain.

Pain doesn’t mean injury, especially in central sensitization.

Most often, pain is the body’s way of limiting movement that would have been protected during an initial injury.

What I mean is that our brain is always trying to protect us from perceived pain.
Effects of exercise on mitochondrial function, neuroplasticity and anxio-depressive behavior of mice

Anxiety and Depressed Mice
Exercise on wheels in cages
6 weeks exercise
Decreased anxiety in Maze test
Anti-depressant like effects on suspension test
mRNA expression of Bdnf and other factors in metabolism were increased after exercise.
exercise appears to engage mitochondrial pathways and to potentiate neuroplasticity and might be associated to mood improvement

Exercise

Different types of exercise induce differential effects on neuronal adaptations and memory performance.

A systematic review of studies that analyzed the effect of physical exercise on the peripheral levels of BDNF in elderly individuals. Inclusion in the review if they were studies with elderly, assessed peripheral (serum and/or plasma) BDNF and evaluated an acute exercise or chronic exercise (training).

Results: Five randomized controlled trial and one randomized non-controlled trial studies were analyzed. Five out of six studies reported a significantly higher BDNF response to aerobic acute exercise and to aerobic or strength training program in healthy elderly and elderly with different pathologies.

Conclusion: It was not possible to establish a recommendation protocol for the type and intensity of physical exercise required to produce an increase in levels of BDNF. However, physical exercise, particularly, moderate-intensity exercises seem to be more effective to promote increase the peripheral levels of BDNF in the elderly.

Physical exercise modulates peripheral levels of brain-derived neurotrophic factor (BDNF): a systematic review of experimental studies in the elderly.

We hypothesize that the differential effects of exercises on memory performance are caused by different neuroplasticity changes in relevant brain regions in response to different exercise trainings.

- Treadmill Running (TR) vs Wheel Running (WR)
- Learning (amygdala) and Memory performance (hippocampus)
- Citrate synthase elevated in TR and not WR
- Neurological assay for dendritic field and proteins

Different types of exercise affect the performance of learning and memory via BDNF-TrkB signaling and neuroplasticity in specific brain regions. The brain region-specific neuronal adaptations are possibly induced by various levels of intensity/stress elicited by different types of exercise.

Does vigorous exercise have a neuroprotective effect in Parkinson disease?

Parkinsonian animal models reveal exercise-related protection from dopaminergic neurotoxins, apparently mediated by brain neurotrophic factors and neuroplasticity. Similarly, exercise consistently improves cognition in animals, also linked to enhanced neuroplasticity and increased neurotrophic factor expression. In these animal models, immobilization has the opposite effect. Brain-derived neurotrophic factor (BDNF) may mediate at least some of this exercise benefit. In humans, exercise increases serum BDNF, and this is known to cross the blood-brain barrier. PD risk in humans is significantly reduced by midlife exercise, documented in large prospective studies.

Ongoing vigorous exercise and physical fitness should be highly encouraged.

Exercise impacts brain-derived neurotrophic factor plasticity by engaging mechanisms of epigenetic regulation.

Action of voluntary exercise on the regulation of brain-derived neurotrophic factor (BDNF), a molecule important for rat hippocampal learning, could involve mechanisms of epigenetic regulation.

These results show the influence of exercise on the remodeling of chromatin containing the Bdnf gene emphasize the importance of exercise on the control of gene transcription in the context of brain function and plasticity.

Reported information about the impact of a behavior, inherently involved in the daily human routine, on the epigenome opens exciting new directions and therapeutic opportunities in the war against neurological and psychiatric disorders.


The effects of acute exercise and/or training on BDNF in healthy subjects and in persons with a chronic disease or disability. A systematic and critical literature search was conducted. 'mostly transient' increase in serum or plasma BDNF concentration following an acute aerobic exercise.

The two studies regarding a single acute strength exercise session could not show a significant influence on basal BDNF concentration.

Only three out of ten studies on aerobic or strength training (i.e. 30%) found a training-induced increase in basal BDNF concentration. Two out of six studies (i.e. 33%) reported a significantly higher BDNF response to acute exercise following an aerobic or strength training programme.

Available results suggest that acute aerobic, but not strength exercise increases basal peripheral BDNF concentrations, although the effect is transient.

We can only speculate which central regions and peripheral sources in particular circulating BDNF originates from, where it is transported to and to what purpose it is used and/or stored at its final destination.

No study could show a long-lasting BDNF response to acute exercise or training.

From that point of view, exercise and/or training would result in a higher BDNF synthesis following an acute exercise bout (i.e. compared with untrained subjects). Subsequently, more BDNF could be released into the blood circulation which may, in turn, be absorbed more efficiently by central and/or peripheral tissues where it could induce a cascade of neurotrophic and neuroprotective effects.

Voluntary exercise induces a BDNF-mediated mechanism that promotes neuroplasticity.

Rodents were exposed to voluntary wheel running for 3 or 7 days, and their lumbar spinal cord and soleus muscle were assessed for changes in brain-derived neurotrophic factor (BDNF)

Results indicate that basal levels of neuromuscular activity are required to maintain normal levels of BDNF in the neuromuscular system and the potential for neuroplasticity.

References

- Doidge, N. The Brain the Changes Itself. Penguin; December 2007
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NEUROPLASTICITY

How to gain control of your pain by re-programming your brain

INFANT DEVELOPMENT PATTERNS

Primitive motor pattern generators

GROUND CONTROL

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Infant Development Process

- Automatic, reflexive movement patterns
- “Integrated Stabilizing System” (ISS) activated for the purpose of achieving exciting levels of improved function
- Neural control programs can be combined in many ways to create complex movements
3 MONTH PATTERNS
4 – 4.5 MONTH PATTERNS

Source: Prague School of Rehabilitation
5 MONTH PATTERNS

Source: Prague School of Rehabilitation
6 MONTH PATTERNS

Source: Prague School of Rehabilitation
7 MONTH PATTERNS

Source: Prague School of Rehabilitation
7-8 MONTH PATTERNS

Source: Prague School of Rehabilitation
9-10 MONTH PATTERNS

Source: Prague School of Rehabilitation
10-11 MONTH PATTERNS

Source: Prague School of Rehabilitation
11-12 MONTH PATTERNS

Source: Prague School of Rehabilitation
11-12 MONTH PATTERNS

Babinski reflex disappears

Source: Prague School of Rehabilitation
What have we learned?

Pain process, the rewiring of the sensory maps.
Re-organizing the brain
Exercises effect on Neuroplasticity
Movement development patterns
Neuroplasticity + Infant Development
BCFit 2014: Ground Control

NEUROPLASTICITY
How to gain control of your pain by re-programming your brain

INFANT DEVELOPMENT PATTERNS
Primitive motor pattern generators

GROUND CONTROL
THEORY...GARGOYLE/BEAR/SJSU/SEESAW/CENTREPOINT

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Jeff Almon, DC, CSCS
jeff@mvmtlab.com
#615-55 WATER ST
Maintain an open mind, because what you are doing & teaching today you will have to modify in view of new facts. The task is enormous, there is a generation’s work. Go step by step - Prof. Karel Lewit
Ground Control is the process of relearning movement from the beginning, Again.
What do we need to do to RESET our PROGRAMMING?
INTEGRATE and DISINTEGRATE at the same time
1. Asymmetry
2. Breathing Stereotypes
3. Motor Control
4. Bio-psychosocial factors
5. Neuroprotective State
6. Joint Centration
7. Proprioceptive Map
8. Weber Fleschner Rule
9. Sustained Motor Patterning
10. Neurokinetic Chains
11. Sensory Motor Integration
<table>
<thead>
<tr>
<th>STRUCTURAL ASYMMETRIES</th>
<th>FUNCTIONAL ASYMMETRIES</th>
</tr>
</thead>
<tbody>
<tr>
<td>VISCERA</td>
<td>HEMISPHERICITY</td>
</tr>
<tr>
<td>KINETIC CHAIN TONE BIAS</td>
<td>MOVEMENT PATTERNS</td>
</tr>
<tr>
<td>SERIAL DISTORTION PATTERNS</td>
<td>INPUT MECHANISMS</td>
</tr>
</tbody>
</table>
HEMISPHERICITY
The traditional concept of **hemisphericity** refers to the idea that people may rely on a preferred mode of cognitive processing, which is linked to activity in the left or right cerebral hemisphere.

According to recent research, this now *appears* to be FASLE.

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**An Evaluation of the Left-Brain vs. Right-Brain Hypothesis with Resting State Functional Connectivity Magnetic Resonance Imaging**

HEMISPHERICITY

Modern concepts in functional neurology, which describe hemisphericity as differing firing rates between the left and right sides of the brain. With various kinds of stress placed upon the body and brain – some as simple gravity – there will be a lower performing cerebral cortex and a higher performing cerebral cortex.

According to Roger Sperry, the 1981 Nobel prize winner for brain research, a large proportion of the brain’s output is directed simply towards maintaining your body posture in its gravitational field, which is why posture is so important to maintain a healthy brain. ***

The LEFT brain tends to be the “accelerator” of the body,

whereas the RIGHT brain tends to be the “brake” system.

L = ACCELERATOR

R = B R A K E
HEMISPHERICITY

DECREASED ACCELERATOR
- body not stimulated enough
- depression

DECREASED BRAKES
- “runaway nervous system”
- increased allergies
- Hypersensitivity
- Emotional instabilities
The cortex is also responsible for inhibiting our “primal brain”, the brainstem. The brainstem regulates the fight/flight response through the thoracic spine. So if a stressed individual is beginning to exhibit signs of hemisphericity, their fight/flight response will be elevated and will present as ulcers, reflux or irritable bowel syndrome.

The cortex controls motor output to muscles too, so hemisphericity can create pain syndromes on one side of the body over time like carpal tunnel syndrome, Golfer’s elbow or sciatica.
Correction of hemisphericity is achieved by the two hemispheres of the brain reaching temporal coherence, i.e. firing at a similar rate and in harmony with each other.

Changes in brain function after manipulation of the cervical spine.

Accurate reproducible maps of cortical responses can be used to measure the neurological consequences of spinal joint manipulation. Cervical manipulation activates specific neurological pathways. Manipulation of the cervical spine may be associated with an increase or a decrease in brain function depending upon the side of the manipulation and the cortical hemisphericity of a patient.

Balanced activity of stabilization muscles allows for symmetrical loading of individual sections of the spine.

Poor quality of activation of stabilization musculature leads to overloading of certain segments of the spine and gradual development of degenerative changes, such as disc herniation or arthritis.
INTEGRATED STABILITY SYSTEM OF THE SPINE (ISSS) ACTIVATES WHEN WE BREATHE WITH A DIAPHRAGMATIC PATTERN

FEED-FORWARD MECHANISMS automatically and reflexively prepare for movement of the extremities

Rolling exercises designed to train the deep spinal muscles
Haynes, W. Journal of Bodywork and Movement Therapies (2003), 7(1) 2003 Published by Elsevier Science Ltd.
The central nervous system works with input mechanisms (nociception and peripheral neurogenic pain) and output mechanisms (autonomic, motor, neuroendocrine, and immune systems).

Central mechanisms are at play as well, most importantly the hyperexcitability levels of the cortexes. This is due to central sensitization,

“an amplification of neural signaling within the central nervous system that elicits pain hypersensitivity.”

Figure 1. Systems model indicating the emergent pattern of movement arising from the interaction of system elements. The model includes only the physical constraints of the system's components: it does not include 'psychological' factors for the organism (e.g. personality, motivation).
Motor Control is effected by these “dys-afferent” signals and alters processing. This in turn alters motor control.

The brain then “inhibits” certain muscles which then create a reactively tight antagonist. (ex: achilles rupture)

This creates the NEUROPROTECTIVE STATE

This disrupts JOINT CENTRATION
NEUROPROTECTIVE STATE

• Essentially what is happening is that the body organizes itself in a pattern that protects from a fall.
• The incoming information doesn’t make sense to the CNS and it is protecting itself.
• Patterns for Spine and extremities.
JOINT CENTRATION

• Joint centration is the “Co-Activation” of Agonists and antagonists in a smooth patterns that creates “suction” in a joint.

• Important for the CNS to receive safe signals to release from the Neuroprotective State.
Accurate movement depends on a good proprioceptive map, the physical areas of the brain responsible for controlling and sensing the movement at each body part. These brain areas or “maps” develop their neuronal linkages in response to physical practice and the sensory feedback that occurs as a result.

What fires together, wires together (Hebb’s Law)

Applying the **Weber Fechner Rule**, we know that gentle movement leads to a more accurate and discriminating perception of the mechanics of the movement. In other words, there is more detailed and refined information available to the brain to build the movement map. The map becomes clearer with greater resolution.

Fine tuning the movement patterns leads to easier motor output.
SUSTAINED MOTOR PATTERNING

• **Inter** and **intra** muscular coordination. The key to motor control. Motor unit recruitment throughout the kinetic chains (See Anatomy Trains – Myers).

• **Time Under tension**
Post Activation Potentiation

• *post-activation potentiation* (PAP) has surfaced as a means to maximize acute power development in athletes

• excitation of the nervous system produces an increase in contractile function due to a heavy load conditioning stimulus. The most common indicator of PAP is increased evoked isometric twitch force observed following an evoked isometric tetanic contraction.


1. STABILITY LOCK - Deep spinal stability system
2. Joint centration (Cspine, Lspine)
3. Breathing stereotypes (dec. Scissor position)
4. Eyes - Oro facial reflexes
5. Arm positions
6. Occipital lock
8. Hip and foot positions
9. Sensory Motor Integration
10. Though processes
THE BEAR

1. STABILITY LOCK - Deep spinal stability system
2. Joint centration (Cspine, Lspine)
3. Breathing stereotypes (dec. Scissor position)
4. Arm positions (corkscrew position)
5. Occipital lock
6. Thoracic spine and Serratus anterior
7. Hip and foot positions
8. Sensory Motor Integration
9. Though processes
1. STABILITY LOCK - Deep spinal stability system
2. Joint centration (Cspine, Lspine)
3. Breathing stereotypes (dec. Scissor position)
4. Eyes - Oro facial reflexes
5. Deep Squat position
6. Arm positions
7. Occipital lock
8. Hip and foot positions
9. Sensory Motor Integration
10. Though processes
Dr. Almon graduated from the University of Calgary with a degree in Exercise Physiology. The Canadian Football League (CFL) selected Jeff as one of thirty-two players to be assessed in the 2002 CFL Combine in Montreal intended for Canadian born university players in the CIS and NCAA. Jeff was selected in that year’s CFL draft by the Grey Cup champion Calgary Stampeders. He continued playing with the BC Lions during the 2003 season. His football career changed when he was accepted into the Canadian Memorial Chiropractic College’s chiropractic program which is an intensive four years of study cumulating with a degree in Chiropractic. Since that time, Dr. Almon has consulted and treated numerous athletes and teams including the Chinese National Speedskating Team, PGA tour golfers, Alpine Ski Ontario, FIVB volleyball athletes, The Vancouver Canucks development program, Canadian carded athletes, and professional sprint cyclists. He is the official conditioning coach for Miss Universe (BC) and practices out of his Gastown clinic, MVMTLAB, treating patients with a focus on dynamic neuromuscular stabilization. He offers specialty group training programs which focus on resetting the nervous system. These two, month-long intensive programs are called Ground Control and Bazooka. Jeff is excited to share his information and ideas in creating a more functional human experience.