New anatomy and motor control that results from neurological injury or disease

W. Barry McKay
Spinal Cord Injury Research Laboratory
Consequences of CNS injury or disease

**New Anatomy**

- Neuron death
- Axon demyelination
- Partial (focal or diffuse)
- Regrowth, remyelination and recalibration
- *New anatomical relationships*
  - within and between processing CNS nuclei
  - altering functional output in complex ways
- *Highly individualized New Anatomy*

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Spinal Motor Centers, "nuclei"
Somatotopic Organization

sEMG reflects CNS modulation of motor output, spinal motor excitability, and therefore muscle contraction and movement
Plurisegmental Reflex Control

Cutaneomuscular withdrawal reflex
Volitional Control

**Reticulospinal Tract**
Generalized excitation intensity based on level of alertness and possibly, effort

**Vestibulospinal Tract**
Equilibrium – excitation intensity based on antigravity demands

**Corticospinal System**
Anterior and Lateral Tracts
Supplementary Motor Area
Primary Motor Cortex

**Bulbospinal Nuclei**
Movement versus *Motor Control*

- **Movement is measured as:**
  - Range of motion
  - Speed of movement
  - Forces (most clinical scales)
  - Trajectories
  - Angular velocities

- **Motor Control can be measured as:**
  - *Selection* and firing of motor neurons
  - *Activation*, in concert with other motor units in multiple muscles
  - *Deactivation* of motor units
    - Inhibition of reflexes and spasms
    - Control over synergistic relationships
    - Cessation of activity to end task
Relaxation

- Intact nervous systems
  - can achieve EMG silence
- Damaged nervous systems
  - unbalanced input to spinal motor neurons
- **Inhibition dominates**
  - no motor unit output
- **Excitation dominates spinal pre-motor center**
  - “Spontaneous” motor unit firing results

C8 AIS-C SCI (5 minutes)
C4, AIS-D Central Cord Syndrome (20 second segments)


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6 days | 47 days | 6 months
Relaxation

Parkinson’s Disease (20 seconds)

Relaxation – Regularly-repeating background activity

SCI (5 minutes)
Relaxation – Regularly-repeating background activity

Multile Sclerosis (5 minutes)
Motor unit recruitment rate reduction

9 subjects with initial recordings between 1 and 11 days post onset (6.4 ± 3.6 days)

**Biceps Brachi**
Voluntary elbow flexion and extension

The time between the first motor unit firing and the peak of pooled firing decreases with recovery

<table>
<thead>
<tr>
<th>Muscle</th>
<th>Non-injured Onset-to-peak time</th>
<th>SCI Onset-to-peak time</th>
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<tr>
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<td>Initial recording</td>
<td>First seen</td>
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<td>(Sec ± s.d.)</td>
<td>Number of sides</td>
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<tr>
<td>Biceps brachi</td>
<td>0.28 ± 0.17</td>
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<td>Wrist extensors</td>
<td>0.53 ± 0.39</td>
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<td>Quadriceps</td>
<td>0.42 ± 0.21</td>
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<td>Tibialis anterior</td>
<td>0.59 ± 0.28</td>
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Recovery after SCI

Example of voluntary right ankle dorsiflexion “move and hold”

Complete paralysis at onset and at 11 days post-injury
27 days - Activation of prime mover (RTA) with co-activation of antagonistic and distant muscles
45 days - Increased prime mover activation with coactivation and clonus
135 days - Increased prime mover activation with decreased coactivation

Slow recruitment
Disrupted spatial distribution

Voluntary Wrist Extension and Flexion

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<thead>
<tr>
<th>Intact Subject</th>
<th>Hemiparetic Subject</th>
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<td>R Biceps Brach</td>
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Self-paced voluntary movement

*Slow recruitment of motor units in the hemiparetic subject*
Voluntary Elbow (Left) Extension and Flexion
(Supine position)

Intact Subject

Hemiparetic Subject

Self-paced voluntary movement

Slow recruitment of motor units and co-activation of muscles in the hemiparetic subject
Other causes of motor unit firing control changes - *Fatigue*

- In the normal motor neuron pool
  - firing rates are matched with muscle fiber one-half relaxation times
    - those innervating slow muscles (soleus) fire at slow rates and have low-fusion frequencies while those innervating fast muscles (tibialis anterior) that fire at high rates and have high-fusion frequencies

- During fatiguing contractions, developed force decreases while firing rates increase presumably with increasing drive (effort) 2,3

Other causes of motor unit firing control changes - *Aging*

- Decrease in force development
- Decrease in motor unit firing rates
- Decrease in normal rate fluctuations
- Lower recruitment force thresholds
- Motor unit potentials that appear increasingly polyphasic
  - suggesting denervation–reinnervation processes

Other causes of motor unit firing control changes - *Stroke*

- Decrease in motor unit baseline firing rates
- Earlier recruitment of motor units with increasing force
- Loss of the ability to modulate firing rates appropriately

Other causes of motor unit firing control changes - *Spinal Cord Injury*

- **Chronic phase, incomplete lesions**
  - Reduced joint movement torques
  - Torque development is slowed
  - ...even though peripheral nerve stimulation peak twitch forces are within normal limits 1

- **Acute and sub-acute phases**
  - Recruitment rate slowed
  - ...with recovery, recruitment rate increases, approaching times measured in non-injured subjects 2


Other causes of motor unit firing control changes - *Training*

- Strength training exercise brings
  - increase in TMS-MEP amplitude
    - increase in the number of motor units activated
  - increase in the maximum number of volitionally recruited units
  - Increase motor unit discharge rates
- However, technical limitations of this study left the question of whether the changes were due to cortical or spinal changes open.

Measuring New Anatomy

- Behavior of motor neural circuitry can be...
  - Objectively characterized
  - Quantified using spinal motor output,
    • pooled motor unit activity
    • appropriate muscles
    • during well-designed reflex and volitional motor tasks.

- **The important parameters:**
  - *Resting balance of excitation - inhibition*
  - *Rate of voluntary motor unit recruitment*
  - *Spatial distribution of motor unit activation across multiple muscles*
  - *Rate of cessation of motor unit firing*