Sensorimotor Adaptations Following ACL Reconstruction: Implications for an Evidence Based Treatment Approach?

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Outline

• Joint injury as a source of disability
• Assessment techniques
• Study findings
• Evidence of sensorimotor adaptation
Learning Objectives

• Following ACL reconstruction:
  – Identify sources of sensorimotor impairment
  – Identify and interpret assessment techniques to examine sensorimotor impairment
  – Summarize the evidence supporting sensorimotor adaptation
Musculoskeletal Pathology

Graph 1.1.1: Prevalence of Self-Reported Primary Medical Conditions for Persons Aged 18 and Over, United States

Graph 1.2.1: Self-Reported Impairment in Activities of Daily Living Due to Select Medical Conditions for Persons Aged 18 and Older, United States 2008

National Center for Healthy Statistics, National Health Interview Survey, 2008; Bone and Joint Decade.
Joint Injury Drives MSK Injury


National Center for Healthy Statistics, National Health Interview Survey, 2008; Bone and Joint Decade.
ACL Epidemiology

- ACL rupture common in sports (Beyonnon et al., 2005)
- ACL reconstruction often recommended to facilitate return to sport (Marx et al., 2003)

- 1.3% annual increase in ACL injury (‘88-’04) (Hootman et al., 2007)

(Lyman et al., 2009)
Sub-Optimal Outcomes
• **65% return to pre-injury status** (Ardern et al., 2014)
  – 55% to competitive sport

• **29.5% suffer secondary ACL injury within 24 months** (Paterno et al., 2014)

• ↓ **knee-related QOL at 9 years (range 6-15 years)** (Filbray et al., 2014)
Manifestations of Joint Injury

Muscle Weakness
Morphological Changes
Neuromuscular Alterations
Structural Changes
Impaired Movement Strategies
Altered Fitness Levels
Altered Biomechanics
Fear/Disability/Decreased Quality of Life

Identify modifiable factors!
Acceleration of Degeneration

20  30  40  50  60  70  80

GRADE 1  GRADE 2  GRADE 3  GRADE 4
ACL Injury and Osteoarthritis

ACL-R accelerates OA development

- **10-90% at 10-20 years** (Lohmander et al., 2007)
- **2014 Systematic Review** (Luc et al., 2014)
  - Total: 44%
  - 0-20 years: 36%
- **2015 Systematic Review** (Harris et al., 2015)
  - 41% (95% CI 35-48%) at 12 years

Lack of evidence to suggest ACL-R will prevent knee OA
• **Post-traumatic quadriceps dysfunction**
  - Altered biomechanics (Andriacchi, 1993)
  - ↓ physical activity (Fitzgerald et al., 2004)
  - ↑ risk of re-injury (Paterno et al., 2014)
  - ↑ self-reported disability (Ericsson et al., 2013)
  - ↑ onset of joint degeneration (Oiestad et al., 2011)
• Muscle is uninjured, yet unable to adequately contract

• Phenomenon is not unique to injury...
  – ACL tear/reconstruction
  – Meniscal tear/ meniscectomy
  – Patellofemoral pain
  – Osteoarthritis
  – Total knee arthroplasty
**Arthrogenic Muscle Inhibition**

- “Arthro” = Joint
- “Genic” (genesis) = Origin
- “Muscle inhibition” = Inability to contract muscle

- AMI = Inability to contract an uninjured muscle due to pathology at the joint
What Causes AMI?

- Altered afferent stimuli from joint receptors transmitted to spinal cord
  (de Andrade et al., 1965; Stokes and Young, 1984)
Neuromuscular Adaptation

Healthy MN Pool

Inhibited MN Pool

Healthy
Reserve
Inhibited
A Clinical Dilemma
Sensorimotor Assessment

CNS

Peripheral
Superimposed Burst Technique

- **Central Activation Ratio (CAR)**

Knee Extension MVIC Torque

\[
\text{CAR} = \frac{\text{Motor Neurons Activated}}{\text{Total Motor Neurons Available}}
\]
Measuring Quadriceps Activation

- **Reliability:**
  - Healthy
  - Patellofemoral pain
  - Closed-Chain

- **Studied in:**
  - ACL reconstruction
  - ACL deficient
  - Patellofemoral pain
  - Osteoarthritis
Hoffmann Reflex

• **Estimate of motoneuron pool activity** (Palmieri et al., 2004)
  – $\alpha$MN excitability of target muscle

• **Think stretch reflex minus the stretch**
H-Reflex Pathway

Stimulator (B) → Sensory Nerve → Spinal Cord → Motor Nerve → Muscle

EMG (A) → M wave → H reflex
Hoffmann Reflex

- **H-Reflex**
  - MN that are available for use...not what is going to be used

- **M-Response**
  - Entire αMN pool

- **H:M Ratio**
  - Proportion of motoneuron pool capable of being recruited
Measuring Spinal Reflexes

- **Reliability:**
  - Peripheral nerves

- **Studies In:**
  - Musculoskeletal injury
  - Effects of therapeutic modalities/ pain
  - Response to exercise
  - Performance of motor tasks
“Non-invasive tool for measuring neural conduction and processing time, activation thresholds, facilitation and inhibition in cerebral cortex, and neural connections” 

(Anand and Hotson, 2002)
Measuring Cortical Reflexes

- **Single or paired pulse**
  - Multiple sclerosis
  - Amyotrophic lateral sclerosis
  - Stroke
  - Movement disorders
  - Spinal cord disorders
  - Musculoskeletal injury

- **Repetitive**
  - Depression
  - Epilepsy
  - Speech impairments
  - Anxiety
  - Neurodegenerative disorders
  - Migraine/chronic pain
Creating a Motor Program
Creating a Motor Program

- Primary motor cortex
- Primary somatosensory cortex
- Premotor cortex
- Somatosensory unimodal association cortex
- Posterior association area
- Anterior association area
- Limbic association area
- Primary auditory cortex
- Auditory unimodal association cortex
- Primary visual cortex
- Visual unimodal association cortex
Evoking a Motor Potential
Understanding the Motor Threshold

Resting Membrane Potential

Threshold
Understanding the Motor Threshold
Understanding the Motor Threshold

- Lowest intensity capable of eliciting MEP
  - Reflects excitability of central core of neurons

Slide adapted from Dr. Brian Pietrosimone
What Does a Higher Threshold Mean?
Early Identification

• Theoretical temporal changes following knee joint injury
  • Strength
  • Activation
  • Fatigue
  • Spinal reflex
  • Corticospinal
  • Motor control

[Diagram showing phases of recovery: Pre, Acute, Sub-Acute, Chronic]
PICO Clinical Question

• In young, active persons with ACL reconstruction compared to healthy individuals, do a) peripheral, b) spinal, and c) supraspinal pathways of the sensorimotor system differ over time?

P – Young, active persons with ACL reconstruction
I – ACL reconstruction
C – Healthy, matched controls
O – Sensorimotor pathways
Experimental Design

• Cross Section

• Independent Variables
  – Group (ACL-R – 6 months, 1 year, > 2 years, Healthy)
  – Limb (Injured, Uninjured)

• Primary Outcome Measures
  – Knee extension torque (Nm/kg)
  – Quadriceps central activation ratio (%)
  – Hoffman Reflex (H:M ratio)
  – Active Motor Threshold (%)

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Patient Reported Outcomes

- IKDC subjective knee evaluation form (IKDC)
- Knee Osteoarthritis Outcome Score (KOOS)
  - Pain, Symptoms, ADL, Sport, QOL
- Tampa Scale for Kinesiophobia (TSK)
- Veteran’s Rand 12-Item Health Survey (VR-12)
- Tegner Activity Scale
- Godin Leisure-Time Questionnaire
- Visual Analog Scale for pain (VAS)
Participants

- **ACL Reconstruction (n = 39)**
  - 15-45 years
  - Primary, unilateral
  - Uncomplicated
  - Time from surgery

<table>
<thead>
<tr>
<th></th>
<th>6 months (n = 15)</th>
<th>1 year (n = 8)</th>
<th>2+ year (n = 16)</th>
<th>Healthy (n = 13)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>23 ± 7</td>
<td>22 ± 7</td>
<td>24 ± 5</td>
<td>23 ± 6</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>175 ± 11</td>
<td>177 ± 11</td>
<td>169 ± 11</td>
<td>173 ± 10</td>
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<tr>
<td>Mass (kg)</td>
<td>73 ± 16</td>
<td>79 ± 20</td>
<td>77 ± 20</td>
<td>73 ± 13</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>24 ± 4</td>
<td>25 ± 4</td>
<td>27 ± 5</td>
<td>24 ± 3</td>
</tr>
<tr>
<td>Time Since Surgery</td>
<td>6 ± 1</td>
<td>11 ± 2</td>
<td>78 ± 45</td>
<td>N/A</td>
</tr>
</tbody>
</table>

*P ≤ 0.05*
Assessed for Eligibility (n = 54)

Group Allocation (n = 52)

ACL-R – 6 months (n=15)  ACL-R – 12 months (n=8)  ACL-R – 2+ years (n=16)  Healthy Control (n=13)

Hoffmann Reflex

Knee Extension MVIC

Superimposed Burst

Transcranial Magnetic Stimulation

Excluded for Analysis (n=9)
  • Unable to elicit MEP
    • Healthy (n=6)
    • ACL 6 mo (n=1)
    • ACL 1 yr (n=1)
    • ACL 2 yr (n=1)

Excluded (n = 2)
  • Did not meet criteria for TMS (n = 2)
**Quadriceps Torque Reduced**

- **Group main effect, P = .029**

- **P = .024**

- **P = .024**

**Peak Knee Extension MVIC Torque**

- **Injured**
- **Uninjured**

- **6 month**
- **1 year**
- **2 year**
- **Healthy**
Quadriceps Activation Reduced

Central Activation Ratio

<table>
<thead>
<tr>
<th></th>
<th>Injured</th>
<th>Uninjured</th>
</tr>
</thead>
<tbody>
<tr>
<td>6 month</td>
<td>0.90</td>
<td>0.85</td>
</tr>
<tr>
<td>1 year</td>
<td>0.85</td>
<td>0.80</td>
</tr>
<tr>
<td>2 year</td>
<td>0.85</td>
<td>0.80</td>
</tr>
<tr>
<td>Healthy</td>
<td>0.95</td>
<td>0.90</td>
</tr>
</tbody>
</table>

P = .010
Spinal Excitability

Hoffmann Reflex: Muscle Response

- Injured
- Uninjured

6 month | 1 year | 2 year | Healthy

- Healthy
Corticospinal Excitability Reduced

Group main effect, $P = .008$

- $P = .030$
- $P = .016$
- $P = .012$
- $P = .007$

Active Motor Threshold

- Injured
- Uninjured

6 month 1 year 2 year Healthy
Clinically Meaningful?

Cohen’s d effect sizes with 95% confidence intervals

-4 -3 -2 -1 0 1 2
ACL-R Worse

ACL-R Better

Small ≤ 0.3
Medium 0.4-0.7
Large ≥ 0.8

- MVIC (6 month)
- MVIC (1 year)
- MVIC (2+ years)
- CAR (6 months)
- CAR (1 year)
- CAR (2+ years)
- H:M (6 months)
- H:M (1 year)
- H:M (2+ years)
- AMT (6 months)
- AMT (1 year)
- AMT (2+ years)
Neuromuscular Adaptations

- **Following ACL-R:**
  - ↓ quadriceps strength > 2 years*
  - ↓ quadriceps activation at 12 months*
  - ↓ corticospinal excitability at 6-12 months*
  - Spinal reflexes are unchanged (trend)

*Large magnitude Δ!

- Do neurophysiologic measures influence strength and/or patient reported outcomes?
Explaining Strength

<table>
<thead>
<tr>
<th></th>
<th>Knee extension MVIC Torque ($R^2$)</th>
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</thead>
<tbody>
<tr>
<td></td>
<td>6 months</td>
</tr>
<tr>
<td>H:M</td>
<td>.026</td>
</tr>
<tr>
<td>AMT</td>
<td>.171</td>
</tr>
<tr>
<td>Total</td>
<td>.173</td>
</tr>
</tbody>
</table>

6 months | 1 year | 2+ years

Quadriceps Strength

Spinal Reflex

Corticospinal

H:M

AMT

Total

6 months | 1 year | 2+ years
Explaining Patient Outcomes

<table>
<thead>
<tr>
<th></th>
<th>IKDC (R²)</th>
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<tbody>
<tr>
<td></td>
<td>6 months</td>
</tr>
<tr>
<td>H:M</td>
<td>0.002</td>
</tr>
<tr>
<td>AMT</td>
<td>0.019</td>
</tr>
<tr>
<td>MVIC</td>
<td>0.003</td>
</tr>
<tr>
<td>CAR</td>
<td>0.000</td>
</tr>
</tbody>
</table>

6 months  1 year  2+ years

Quadriceps Strength
Quadriceps Activation
IKDC
Spinal Reflex
Corticospinal
Meaningful to Patient?

Cohen’s d effect sizes with 95% confidence intervals

Small $\leq 0.3$
Medium 0.4-0.7
Large $\geq 0.8$

- IKDC (6 month)
- IKDC (1 year)
- IKDC (2+ years)
- KOOS (6 months)
- KOOS (1 year)
- KOOS (2+ years)
- TSK (6 months)
- TSK (1 year)
- TSK (2+ years)
- VR-12 (6 months)
- VR-12 (1 year)
- VR-12 (2+ years)
Self-Perceived Function

• Following ACL-R:
  – ↓ knee-specific function (IKDC, KOOS) > 2 years
  – ↑ fear of movement (TSK) at 6 months
  – ↓ global health (VR-12) at 6 months

Large magnitude Δ!
Immediate Change in Spinal Reflexes

• Early effusion models used to demonstrate an acute decrease in quadriceps motor neuron pool (DeAndrade et al, 1965; Spencer et al, 1984; Baxendale et al, 1985; Jensen and Graf, 1993; McNair et al, 1996; Wood et al, 1996)

• Immediate changes in:
  – ↓ Quadriceps
  – ↑ Soleus
    (Hopkins et al., 2000, 2001)
Spinal Reflexes in Pathology

- **Alterations in spinal reflexes**
  - Functional ankle instability (Palmieri-Smith et al., 2009; McVey et al., 2005)
  - Acute ankle sprain (Klykken et al., 2011)

- **ACL Injury?** (Heroux and Tremblay, 2006)
  - N=5 ACL deficient
  - Injured limb trended toward lower H-reflex amplitude ($p = .07$)
Evidence of Cortical Changes?

- **ACL deficient**
  - 10 ACL-D
  - 8 Healthy
- **Injured limb had lower resting threshold in ACL group**
- **Chronic adaptation to maintain function?**

22 (4-108) months from surgery

Corticospinal Adaptation

• 20 healthy knees artificially effused

• Results
  – No immediate changes in corticospinal excitability!

• Evidence for chronic adaptation?

Cortical Change and Ankle Instability

• **Case control**
  – Chronic ankle instability (n=10)
  – Control (n=10)

• **Results**
  – Higher resting threshold in fibularis longus

Neural Changes and ACL-R

- **Case control** 48 ± 35 months from surgery
  - ACL-R (n=29)
  - Matched healthy (n=29)

Neuromuscular Asymmetry

34 ± 23 months since surgery

What Matters?

• **Neural alterations are related to quadriceps strength**
  – CAR (37%) + H:M (10%) + AMT (2%) predicted 49% of variance in MVIC (Lepley et al., 2014)

• **Strength and cortical excitability are related to self-reported disability**
  – MVIC (61%) + AMT (5%) predicted 66% of variance in IKDC score (Pietrosimone et al., 2013)
Evidence of Neuromuscular Change?

Yes!

<table>
<thead>
<tr>
<th>Time From Injury (Phase of Recovery)</th>
<th>Outcomes</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;6 months</td>
<td>Good</td>
</tr>
<tr>
<td>6 months</td>
<td></td>
</tr>
<tr>
<td>&gt;6 months</td>
<td>Poor</td>
</tr>
<tr>
<td>&gt;12 months</td>
<td></td>
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<tr>
<td>&gt;24 months</td>
<td></td>
</tr>
</tbody>
</table>

Quadriceps activation: Level 1-2, B
Quadriceps spinal reflex: Level 3-5, C
Quadriceps corticospinal reflex: Level 2-3, B
Significance

• **Research**
  – Understanding nature of post-traumatic neurophysiologic modulation
    • Validate patient-specific intervention

• **Clinical Implications**
  – Early identification = Early treatment!
What’s Next

6 months  12 months  >24 months
Take Home Points

• Modulation of the sensorimotor system occurs following knee joint trauma

• Worthwhile to look beyond gross strength measures as an outcome to identify source of impairment

• Identifying sensorimotor adaptations, specifically mal-adaptations early may provide a targeted treatment approach
Clinical Bottom Line

• Early identification is key!

• Cost-effective treatment approaches to target sensorimotor impairments exist that may supplement traditional rehabilitation techniques after ACL reconstruction
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Thank You

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Department of Kinesiology

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Resources for EBP

• Centre for Evidence Based Medicine
  – http://www.cebm.net/

• PubMed Literature Searching Tutorial
## Patient Reported Outcomes

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<tbody>
<tr>
<td>IKDC</td>
<td>81 ± 13</td>
<td>89 ± 7</td>
<td>87 ± 11</td>
<td>99 ± 1</td>
</tr>
<tr>
<td>KOOS</td>
<td>88 ± 11</td>
<td>91 ± 5</td>
<td>93 ± 6</td>
<td>99 ± 1</td>
</tr>
<tr>
<td>KOOS: Pain</td>
<td>91 ± 9</td>
<td>93 ± 6</td>
<td>94 ± 5</td>
<td>99 ± 1</td>
</tr>
<tr>
<td>KOOS: Symptoms</td>
<td>86 ± 14</td>
<td>83 ± 13</td>
<td>90 ± 9</td>
<td>98 ± 4</td>
</tr>
<tr>
<td>KOOS: ADL</td>
<td>95 ± 8</td>
<td>99 ± 2</td>
<td>97 ± 4</td>
<td>100 ± 1</td>
</tr>
<tr>
<td>KOOS: Sport</td>
<td>77 ± 19</td>
<td>87 ± 12</td>
<td>90 ± 12</td>
<td>98 ± 5</td>
</tr>
<tr>
<td>KOOS: QOL</td>
<td>65 ± 22</td>
<td>77 ± 15</td>
<td>85 ± 13</td>
<td>99 ± 2</td>
</tr>
<tr>
<td>VAS (cm)</td>
<td>0.6 ± 0.8</td>
<td>0.6 ± 0.9</td>
<td>0.4 ± 0.6</td>
<td>0.1 ± 0.2</td>
</tr>
<tr>
<td>Tegner</td>
<td>6 ± 2</td>
<td>8 ± 2</td>
<td>7 ± 2</td>
<td>8 ± 1</td>
</tr>
<tr>
<td>Godin</td>
<td>62 ± 23</td>
<td>63 ± 21</td>
<td>55 ± 26</td>
<td>67 ± 30</td>
</tr>
<tr>
<td>Tampa</td>
<td>34 ± 5</td>
<td>31 ± 7</td>
<td>30 ± 6</td>
<td>28 ± 7</td>
</tr>
<tr>
<td>VR-12</td>
<td>80 ± 10</td>
<td>84 ± 6</td>
<td>85 ± 5</td>
<td>88 ± 6</td>
</tr>
</tbody>
</table>
Treating Neural Impairment

- **Paradigm shift?**

![Graph showing studies over years](chart.png)

- Cryotherapy
- TENS
- Lumbopelvic manipulation
- TMS
- NMES
- Vibration
- Active release
- Acupuncture
- Counterirritant
- Taping
- Bracing

![Images of treatments](treatment_images.jpg)