Simulating Quadriceps Muscle Atrophy and Activation Deficits during Gait

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Simulating Quadriceps Muscle Atrophy and Activation Deficits during Gait
Webinar Objectives

- Background on prevalence of quadriceps muscle weakness
  - 2 types of weakness: atrophy and activation deficit
- Motivating questions
- How we addressed questions using OpenSim
- Methodological details of simulating weakness
- Major findings and take-away
**Background**

- **Osteoarthritis (OA):**
  - musculoskeletal disease
  - progressive deterioration of the articular cartilage of the joint

- **Very Common**
  - 49.9 million in U.S. (22.2% of the population) between 2007 and 2009\(^1\)
  - 67 million (25%) by 2030\(^2\)
  - 37% over age 60 have radiographic evidence of OA\(^3\)

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\(^1\) MMWR, 59: 1261-65, 2010.
\(^3\) Dillon et al., J Rheumatol 33: 2271-79, 2006.
Background

- Approximately 21.1 million adults in the U.S. report activity limitations due to symptoms of arthritis\(^1\)

- Increased dependence and difficulty during activities\(^2\):
  - Climbing stairs, Walking

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• Quadriceps weakness, in particular, has been linked to functional impairment\(^3\)\(^-\)\(^5\)
  • Increased fall risk
  • Slower walking speed

\(^1\) MMWR, 59: 1261-65, 2010.
Quadriceps Weakness

• Quadriceps weakness is one of the earliest and most common symptoms of OA\(^1\)

• Two sources of muscle weakness:
  
  • **Atrophy**
    
    • Decrease in number or size of muscle fibers

  • **Reduced voluntary activation**
    
    • Inability to recruit (activate) all of the muscle’s motor units\(^2\)
      (groupings of muscle fibers)

Quadriceps Weakness

- **Strength deficits**
  - As high as 38% in late stage OA\(^1\)
  - As high as 64% after total knee replacement for treatment of knee OA\(^2\)

- **Activation deficits**
  - As high as 34% in OA\(^3\)

- Underlying mechanism relating quadriceps function to gait impairments is unknown

Dynamic Computer Simulations

- Powerful tool for investigating cause-effect relationships\(^1\)

- Allow us to determine individual roles of muscles in coordinated movement

- Predictive studies: how muscle function changes in response to rehab, surgery, or gait re-training

Two major motor functions used to transport the body in human gait are:\(^1\):

- **Forward progression** (forward acceleration of the body)
- **Vertical support** (vertical accel. of body against gravity)

\(^1\) Winter, University of Waterloo Press, 1991.
• Previous research has investigated muscle function in healthy and some pathological populations\textsuperscript{1-6}

• Main contributors to progression and support during gait are the quadriceps, gluteus maximus, and plantarflexors

• Muscle force generally increases with gait speed

Motivating Questions

- **Muscle compensations in populations with weak quadriceps**
  - Do other muscles compensate for weakness in the quadriceps? How?
  - Do compensations differ between the two types of weakness (atrophy and activation failure)?
Motivation

1. Estimate

   Muscle compensations in response to weak quadriceps
   - Maintain normal gait

   Apply similar method

   Muscle compensations in pathological gait (OA, ACL)

   Inform and Guide Rehabilitation
   - Improve patient outcomes
• To estimate changes in muscle forces and contributions to support and progression to maintain normal gait in response to two sources of quadriceps muscle weakness: atrophy and activation failure
Methods

- Motion capture data collected in the OSU Movement Analysis and Performance Lab
- 7 healthy subjects (4M/3F, 21.9 ± 2.3 years)
  - IRB-approved written consent
- Bilateral gait data collected from each subject
  - Walking on level ground at a self-selected speed (1.32 ± 0.13 m/s)
  - Full-body Point-Cluster Technique
  - Surface EMG from bilateral lower extremity muscles

Generated walking simulations of one gait cycle using the open-source software package OpenSim\textsuperscript{1} and gait2392 model

Computed Muscle Control (CMC)

- Produces a muscle-driven simulation of subject’s movement\(^1\)

Dark Red = fully activated (“on”)
Dark Blue = de-activated (“off”)

Induced Acceleration Analysis (IAA)

• Computes the contributions of individual muscles to forward progression and vertical support

• Foot-contact constraints combined with equations of motion are used to solve for accelerations caused by each muscle force from CMC
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1. “Atrophy Only” - Decreased quadriceps’ peak isometric force to 40% of normal
Simulated Weakness

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  3. “Atrophy + Activation Failure” - Combination of simulated atrophy and activation failure

• Re-ran CMC and IAA for each weakened case
  • While tracking normal gait
  • Re-calculated muscle forces and contributions
Simulating Atrophy

- Decreasing peak isometric force to 40% of normal:
  - Vastus lateralis in generic model = 1871 N
  - Weakened model = 1871 * 0.4 = 748.4 N
Simulating Atrophy
Simulating Activation Deficit

Constraining peaks to 65% of their full-strength value (ie, 35% deficit):

1\textsuperscript{st} peak: $0.399 \times 0.65 = 0.259 \quad t=0.57-0.923 \text{ s}$

2\textsuperscript{nd} peak: $0.175 \times 0.65 = 0.114 \quad t=1.54-1.709 \text{ s}$

Muscle “off”: $t = 0.923-1.54 \text{ s}$
Simulating Activation Deficit
Evaluating Results

- Compare CMC with experimental EMG
- Check coordinate errors
  - <2 degrees (or 2 cm for translations)
- Check residual forces and moments
  - <20N or 50 Nm
## Results: Gluteus maximus and soleus compensate for quadriceps

<table>
<thead>
<tr>
<th>Muscle</th>
<th>Force</th>
<th>Forward Progression</th>
<th>Vertical Support</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Change from Normal (N)</td>
<td>% change</td>
<td>Change from Normal (m/ s²)</td>
</tr>
<tr>
<td>RF</td>
<td>-152.4</td>
<td>-37.7</td>
<td>0.20</td>
</tr>
<tr>
<td>Vasti</td>
<td>-73.8</td>
<td>-9.0</td>
<td>0.16</td>
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<tr>
<td>Glute Max</td>
<td>95.9</td>
<td>26.0</td>
<td>-0.06</td>
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<tr>
<td>Soleus</td>
<td>166.6</td>
<td>9.8</td>
<td>0.06</td>
</tr>
<tr>
<td>MG</td>
<td>-58.1</td>
<td>-5.1</td>
<td>-0.07</td>
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<td>BFlh</td>
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<td>-4.4</td>
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<tr>
<td>Glute Med</td>
<td>-19.7</td>
<td>-2.1</td>
<td>0</td>
</tr>
<tr>
<td>TA</td>
<td>4.5</td>
<td>4.2</td>
<td>0.03</td>
</tr>
</tbody>
</table>

- Green = greatest compensation: gluteus maximus and soleus muscles
Gluteus maximus compensates in early stance

- Gluteus Maximus generates more force in early stance
  - Average peak increase of 162.9 N (42.4% change from normal) for weakest case (p=0.0003)
Soleus compensates in late stance

- Soleus generates more force in late stance
  - Greater compensation needed to overcome activation deficit
  - Average peak increase of 217.2 N (13.1% increase over normal) in response to “Activation Failure Only” (p=0.0016)
Gluteus maximus contributes more to braking and support

- To compensate for weak quads:
  - Glute Max contributes more to slow forward progression (45.8% increase over normal) \((p=0.0003)\)
  - Contributes more to maintain vertical support (32.2% increase over normal) \((p=0.0001)\)
Soleus contributes more to propulsion and support

- To compensate for weak quads:
  - Soleus contributes slightly more to maintain forward progression (7.0% increase over normal) \((p=0.0039)\)
  - Contributes more to maintain vertical support (12.1% increase over normal) \((p=0.0418)\)
Discussion

• First study to develop muscle-driven simulations investigating the two sources of quadriceps weakness:
  • Atrophy
  • Activation failure

• To maintain normal gait pattern, gluteus maximus and soleus show greatest potential to compensate for weak quadriceps
  • Different responses to atrophy and activation failure
Discussion

• Limitations
  • Forced simulations to track healthy gait
  • Activation deficit assumptions
  • Generic musculoskeletal model

• Future work in impaired populations (OA)
  • Patient-specific muscle properties
  • Evaluation of compensation strategies through clinical interventions
Take-Home Message

• Gluteus maximus and soleus muscles may be potential targets for strength training during rehabilitation

• Understanding compensation strategies that are necessary to maintain normal gait provides a foundation to investigate role of muscle weakness in pathological gait

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