Muscle Activation in Cruciate Disease

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Canine Stifle Joint

- ‘Knee’
- Formed by 3 articular surfaces
  - Femur, tibia and patella
- Joined by series of complex ligaments
  - Focus: Cranial cruciate ligament
- Biomechanics
  - Stability: cranial / caudal cruciate, collateral ligaments, menisci
  - Resists motion in all planes


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Cranial Cruciate Ligament (CCL) Rupture

- Common cause of pelvic limb lameness

- $1.32$ billion spent on medical and surgical management in dogs  
  Wilke VL, et. al., *JAVMA*, 2003

Slocum BS, Slocum TD, *Vet Clin N Am*, 1993

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Bilateral CCL Rupture

- Contralateral CrCL rupture
  Vilensky, JA, et. al., J Orthop Res, 1994
  Rumph PF, et.al., Vet Surg, 1995
  - 37-48% 16 months after initial Dx
    Doverspike M, et. al., JAAHA, 1993; Buote NJ, et. al., VOS, 2008
  - 1/3 within 8 months after initial rupture
    Doom M, et.al., Vet Immun & Histopath, 2008

- Compensatory patterns adopted
  - Uncoordinated muscle activity
  - Cranial shift in COM
  - Increased thoracolumbar flexion
  - Increased GRF in thoracic limbs

Photo courtesy of Dr. Robert Taylor

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Canine Example - CrCLR

Intact CrCL

Deficient CrCL

Videos courtesy of Dr. Scott Tashman


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Pathogenesis – CCL Disease

- **Biomechanical stresses**
  - **Ligamentous strain**
    - Abrupt / traumatic
    - Slow, repetitive degeneration
      Hayashi K, et.al., *J Am An Hosp Assoc*, 2004
  - **Conformational abnormalities/joint geometry**

- **Signalment**
  Breed, gender, age, body weight

- **Immune-mediated destruction of ligament**

Photo courtesy of Dr. Robert Taylor

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Effects of CCL Rupture

- **Joint instability – human & canine**
  - Shift in joint contact area
  - Increased joint contact forces
  - Increased reliance on muscles

- **Decreased weightbearing (ex: peak vertical force)**
  - DeCamp CE, et. al., *AJVR*, 1996

- **Increased stifle flexion**

- **Osteoarthritis**

- **Joint effusion/muscle inhibition in humans (canine?)**

- **Muscle weakness in humans (canine?)**
Motor Control

- **Muscle activation – role in joint compression and stability**  

- **Human ACLD: role of muscle activity in supporting joint stability**  
  - Asynchronous timing of mm. onset, timing and amplitude
  - Affects joint stability, stifle kinematics, joint loading
  - Inefficient muscle recruitment – increase periarticular structures to mechanical injury/failure  

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• Normally - during stretch, mechanoreceptors in peri-/intra-articular structures, increase firing of alpha-motor neuron, increasing muscle stiffness/stability
Concurrent regulation of muscle spindle sensitivity to stretch via gamma motor neuron system

ACL injury = loss joint afferent signaling, decreased gamma-motor neuron signaling, reduced mm. activation = decrease joint stability
Knowledge Gap

- Progression of CCL rupture
  - Subclinical disease

- Unknown alterations in timing and magnitude of muscle contraction following a CCL injury/rupture

- Kinetics / Kinematics

- Compensatory gait patterns

- Lack of clinical objective outcome parameters

- Poor model of natural disease progression
  - CCL transection limitations

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Purpose

Characterize alterations in:
- Electromyography (EMG) patterns
- Kinetics and kinematics
- Ipsilateral/contralateral pelvic limbs
- Monopolar radiofrequency energy (MRFE)-induced CCL degeneration and subsequent rupture
Specific Aim

Assess subclinical, acute, chronic phases of CCL disease

- Altered patterns of muscle activity
- Progression of altered GRF and kinematic patterns
- Development of compensatory gait patterns
Hypotheses

- **H0**: significant alterations in muscle onset, activation duration and amplitudes will be measured in the VL, BF and MG at all time points post-injury and rupture, compared to baseline values.

- **H1**: subtle, adaptive kinematic and kinetic gait changes are expected in CCL degeneration, prior to actual CCLR, due to pain, inflammation, and altered proprioception.
Study design

- Randomized, repeated measures design
- N = 6 female hound dogs (age: 1-3.7 years)
- Unilateral MRFE-induced CCL injury
- Assess changes in kinetics and kinematics—pelvic limbs
- Assess EMG and outcome parameters in bilateral pelvic limbs

- 6 time periods;
  - Pre-injury (Baseline)
  - Subclinical: 2 and 4 weeks post MRFE-induced CCL injury
  - 4, 8 and 16 weeks post CCL rupture
MRFE Model

- Mechanical failure with intact ligamentous fibers (Noyes F, 1977)

- Produces gradual loss of stifle stability
  - 25-40% intact fibers at 16 weeks post CCLR in 4/6 dogs
  - Clinical: graded as 2 = complete rupture
  - ‘complete’ rupture = 10-12 mm
  - ‘partial’ rupture = 5-8 mm

- Lopez (2003) = 55 +/- 3 days
• Vastus Lateralis
  ○ Stifle extensor and stabilizer in stance

• Biceps Femoris (caudal)
  ○ Stifle flexor, hip extensor, tarsal extensor and stabilizer in stance
  ○ Active just before paw strike

• Gastrocnemius (medial)
  ○ Tarsal extensor, stifle flexor and stabilizer in stance
  ○ Medial head active 73% of stance (lateral head not reported Goslow GE, et al., *J Exp Biol*, 1981

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Kinetics

- Study of forces that cause movement
Models of CCL Rupture

- **Surgical transection**
  - Acute model
  - Variable OA severity
  - Not natural disease progression

- **Monopolar Radiofrequency Energy (MRFE)**
  - Thermally induced
  - Slow, progressive model
  - Simulates naturally occurring disease
    - CCL rupture 55 days post surgery
Kinematics

- Study of motion (sagittal plane)
Electromyography (EMG)

• Measure of muscle activity
EMG
Example, processed EMG tracing

Biceps Femoris

% Stance 1: -15.00%
Duration of BF: 0.17 sec
Maximum Amplitude: 40.44 μv

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Statistical Analysis

- Repeated measures ANCOVA
- \( N = 5 \)
- Assess changes from baseline values in kinetic, kinematic and EMG parameters between treated and untreated dogs
- Over 6 time periods
  - Independent variables
    - Age
    - Injured limb
      - Treated and untreated
    - Data collection time points
      - Baseline, subclinical, acute and chronic
  - Dependent variables
    - Outcome parameters
- LS Means - individual comparisons among the interaction of time \( x \) injured limb

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Results

- **MRFE-induced injury: weeks to rupture**
  - Week 1 (2 dogs)
  - Week 6 (3 dogs)
  - Week 15 (1 dog)
    - Covariate: no difference

- **N = 5**

- **High EMG variability**
  - Low sample size
  - High standards of deviation

- **Qualitative descriptions of EMG**
Results - Post MRFE

**Kinetics**
- Tx/Untx: no sig diff

**Kinematics**
- UnTx: Decreased avg hip joint ROM by 4-5°
- ‘Stiffer’ joint

<table>
<thead>
<tr>
<th>Time</th>
<th>Treated</th>
<th>Untreated</th>
</tr>
</thead>
<tbody>
<tr>
<td>Baseline</td>
<td>26.7 ± 1.5</td>
<td>24.5 ± 1.5</td>
</tr>
<tr>
<td>Post MRFE-Induced Injury</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Week 2</td>
<td>22.6 ± 1.7*</td>
<td>25.7 ± 1.7</td>
</tr>
<tr>
<td>Week 4</td>
<td>22.2 ± 1.7*</td>
<td>25.1 ± 1.7</td>
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</tbody>
</table>

*Significant difference
Results - Post CCL Rupture

Kinetics
- Tx: Decreases in majority of parameters post CCLR

Kinematics
- Tx (compared to UnTx):
  - Incr avg hip joint ROM
  - Incr tarsal extension (up to 19°)
- UnTx:
  - Increased stance time 6%
  - Increased stifle flexion (up to 20°)
  - Increased tarsal flexion (up to 14°)
TREATED LIMB

Vastus Lateralis Activation Pattern
TREATED LIMB

Biceps Femoris Activation Pattern

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TREATED LIMB

Gastrocnemius Activation Pattern

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UNTREATED LIMB

Vastus Lateralis Activation Pattern
UNTREATED LIMB

Biceps Femoris Activation Pattern

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UNTREATED LIMB

Gastrocnemius Activation Pattern
## EMG

### Subclinical Phase*

<table>
<thead>
<tr>
<th></th>
<th>Treated Limb</th>
<th>Untreated Limb</th>
</tr>
</thead>
<tbody>
<tr>
<td>VL</td>
<td>D/↓</td>
<td>D?/-</td>
</tr>
<tr>
<td>BF</td>
<td>D/↓</td>
<td>D/↓</td>
</tr>
<tr>
<td>MG</td>
<td>D/↓</td>
<td>E/↑</td>
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</tbody>
</table>

### Post CCL Rupture*

<table>
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<td>D/↓</td>
</tr>
<tr>
<td>MG</td>
<td>E/↑</td>
<td>E/↑</td>
</tr>
</tbody>
</table>

*No significance; N = 5

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Discussion

• Novel - neuromuscular contributions to stifle stability

• Qualitative analysis of trial averaged EMG graphs suggest a relationship between neuromuscular function and CCL injury / subsequent rupture despite lack of significance

• Lack of statistical significance
  • Patient to patient EMG variability
  • Small sample size
Discussion

• Transection model of CCL rupture
  • Immediate stifle destabilization
  • Produces large between-dog kinematic variability in joint angle timing and magnitudes (up to 9°) Korvick DL, et.al., J Biomech, 1994; Tashman S, et.al., J Orthop Res, 2004

• MRFE-induced CCL injury model
  • More consistent stifle flexion changes of 3-4° in the untreated limb
  • Suggests a less variable compensatory gait pattern occurs with gradual destabilization
  • Better replicate degenerative process/gradual CCLR
  • Provide opportunities to gradually modify movement strategies?
  • Clinical relevance of differences in kinematic changes between the two models requires further research
• **Post MRFE**
  - VL immediate response to locomotor adaptations = delayed activation in both limbs/decreased activation duration.

• **TREATED LIMB**
  - Delay due to post op effusion within the stifle joint
  - Cause increased intraarticular pressure Strand E, et.al., Equine Vet J, 1998

• **UNTREATED LIMB**
  - Need additional studies to provide a better understanding of these findings

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Discussion - VL

- Compensatory strategies adapt with time?
- **Post CCLR**
  - **TREATED LIMB**
    - VL activated earlier
    - Allow better shock absorption to absorb higher loads
    - Activate sooner - increase stiffness in preparation for weight acceptance in an unstable joint
    - Stiffening (due to muscular co-contraction) plays role in joint stabilization
  - **UNTREATED LIMB**
    - VL activated earlier
    - Contralateral pelvic limb overloaded (post CCL transection)
    - Evidenced by an increase in VL EMG magnitude and duration

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Discussion

- Post CCLR
  - Single burst pattern → multiple burst pattern in all three muscles
  - In both treated and untreated limbs
  - More biphasic, inconsistent activation pattern
  - Suggests poorly controlled muscle activation patterns
  - Contribute to poorly controlled joint movements
    

- Possible cause:
  - Excitatory extensor mechanisms attempting to avoid limb collapse
  - Possible inhibitory extensor mechanisms (instability/effusion present in the stifle)
    
Herzog W, et.al., *Novartis Found Symp*, 2004
Post MRFE, UnTx, ↓ Avg Hip ROM by 5°

- Change in contralateral kinematic pattern
- Alter neuromuscular control at the stifle
- Destabilize stifle/contribute to contralateral CCLR?
  - Muscles produce/accommodate change in work performed/forces transmitted
  - Thus, activity is modulated in timing/intensity
- Change in kinematic patterns
  - Cause ↓ mm forces acting on knee joint  Osternig LR, Med Sci Sports Exerc, 1995
**Discussion**

**Post MRFE**
- Delayed VL and BF
- Concomitant early MG
- Gastroc dominance over hamstrings
- Increase load of cranial tibial thrust on CCL  
- Predisposes UnTx limb to rupture  
  Mostafa AA, et.al., *Vet Surg*, 2010

**Untreated Limb**

<table>
<thead>
<tr>
<th>Post MRFE</th>
<th>VL</th>
<th>BF</th>
<th>MG</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>D/↓</td>
<td>D/↓</td>
<td>E/↑</td>
</tr>
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Discussion

Post CCLR

- Increased stance time 6%; Incr stifle/tarsal flexion
- Corroborates ↑ VL and MG activation duration
- Muscle forces primarily determine joint loading
- Humans after ACL rupture
  - Increased knee flexion of ~4°
  - Altered hamstring recruitment Chmielewski TL, J Electromyogr Kinesiol 2005
  - Need + kinesiological EMG data in dogs

Untreated Limb

<table>
<thead>
<tr>
<th></th>
<th>Post CCLR</th>
</tr>
</thead>
<tbody>
<tr>
<td>VL</td>
<td>E/↑</td>
</tr>
<tr>
<td>BF</td>
<td>D/↓</td>
</tr>
<tr>
<td>MG</td>
<td>E/↑</td>
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Discussion

Post CCLR, UnTx, ↑ Stifle Flexion by 15°

- Altered pelvic limb kinematics—detriment of the contralateral healthy limb?
- Quad activation at 120-140° - produce ↑CCL strain
- Stifle flexion angle >90°
  - Cranially directed tibiofemoral shear force
    Pozzi A, et.al, Plos One 2013
  - 145° (baseline) to 130°-135° (post CCLR)
    - Above the ‘crossover’ point (90° stifle flexion)
    - No change in GRFz in the contralateral pelvic limb
    - Early activation /increased duration of the VL + altered limb kinematics → produce internal changes force/load distribution → contribute to contralateral CCLR?

Tobias and Johnston, Veterinary Surgery Small Animal, 2012

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Limitations

- Sample Size
- Data collection methods
  - Kinetics: velocity; consistent handler
  - Kinematics: marker location; skin motion;
  - sEMG
    - Normalization
      - MVC not possible
      - Additional studies are needed to improve comparative abilities of EMG
    - Crosstalk
      - Large, superficial muscles used
      - Electrodes near muscle belly midline
      - Acetate sheets
    - Consistent electrode placement
In Summary...

- Qualitative observations
- CCL injury/rupture
  - Effect on neuromuscular function
  - Muscle weakness? (decreased EMG amplitude)
    - Measure of intensity of muscle activity
  - Muscle atrophy?
    - Develop rehab strengthening programs
  - Reduced ability to activate the muscle?
    - Removing inhibitory sources that may prevent or delay muscle activation
    - Focus on neuromuscular retraining programs
- Further characterize muscle activity/altered gait patterns
- Improve understanding of normal, pathologic, surgical and rehabilitative biomechanics of the canine stifle joint
Clinical Relevance

- Dogs compensate for CCL rupture with muscle/kinematic versus kinetic parameters
- No clinical/subclinical methods of detecting CCL injury
- Define rehabilitation exercise protocols targeted at
  - Motor control
  - Proprioceptive training
  - Strengthening
  - Orthotics on affected pelvic limb
  - OA
Future Directions

- MRFE provides a subclinical window to further explore neuromuscular contributions to the pathogenesis of CCL disease
- Influence of compensatory gait on development of contralateral CCL rupture and OA
- Establish appropriate therapeutic interventions and determine their effectiveness
Acknowledgements

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  - Allison Arne

- Colorado State University Orthopedic Research Center staff
- Colorado State University Small Animal VTH staff
Flexion and extension joint angles (in degrees) of the stifle during stance phase at all time points. (n = 5 dogs)

<table>
<thead>
<tr>
<th>Time</th>
<th>Stifle Flexion</th>
<th>Stifle Extension</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Untreated</td>
<td>Treated</td>
</tr>
<tr>
<td>Baseline</td>
<td>139.2 ± 3.8</td>
<td>135.9 ± 3.8</td>
</tr>
<tr>
<td>Post MRFE-induced Injury</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Week 2</td>
<td>135.9 ± 4.1</td>
<td>132.1 ± 4.1</td>
</tr>
<tr>
<td>Week 4</td>
<td>133.7 ± 4.1</td>
<td>132.4 ± 4.1</td>
</tr>
<tr>
<td>Post Cranial Cruciate Ligament Rupture</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Week 4</td>
<td>119.6 ± 4.1*</td>
<td>129.7 ± 4.1</td>
</tr>
<tr>
<td>Week 8</td>
<td>122.0 ± 3.8*</td>
<td>126.1 ± 3.8*</td>
</tr>
<tr>
<td>Week 16</td>
<td>126.2 ± 3.8*</td>
<td>129.8 ± 3.8</td>
</tr>
</tbody>
</table>

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Tarsocrural flexion joint angles (in degrees) and timing of tarsocrural joint extension (% of stride) during stance phase at all time points. (n = 5 dogs)

<table>
<thead>
<tr>
<th>Time</th>
<th>Tarsocrural Flexion</th>
<th>Tarsocrural Extension (% of Stride)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Untreated</td>
<td>Treated</td>
</tr>
<tr>
<td>Baseline</td>
<td>114.1 ± 4.1</td>
<td>111.8 ± 4.1</td>
</tr>
<tr>
<td>Post MRFE-induced Injury</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Week 2</td>
<td>116.3 ± 4.5</td>
<td>114.2 ± 4.5</td>
</tr>
<tr>
<td>Week 4</td>
<td>111.1 ± 4.5</td>
<td>117.7 ± 4.5</td>
</tr>
<tr>
<td>Post Cranial Cruciate Ligament Rupture</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Week 4</td>
<td>99.8 ± 4.5*</td>
<td>124.9 ± 4.5*†</td>
</tr>
<tr>
<td>Week 8</td>
<td>99.6 ± 4.1*</td>
<td>119.1 ± 4.1†</td>
</tr>
<tr>
<td>Week 16</td>
<td>104.5 ± 4.1</td>
<td>118.8 ± 4.1†</td>
</tr>
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</table>
Results - Post CCL Rupture

Kinetics

- **Tx:** Decreases in majority of parameters post CCLR

Kinematics

- **Tx:**
  - Incr avg hip joint ROM
  - Incr tarsal extension

- **UnTx:**
  - Increased avg stifle ROM (up to 10º)
  - Increased stifle flexion (up to 20º)
  - Increased avg tarsal ROM (up to 16º)
  - Increased tarsal flexion (up to 14º)
  - More flexion at stifle and tarsus
Stifle Kinematics

Treated Limb

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Stifle Kinematics

Untreated Limb

Post MRFE

Post CCL Rupture

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