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Effect of Experimentally-Induced Anterior Knee Pain on Postural Control

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The Effect of Experimentally-Induced Anterior Knee Pain
On Postural Control

Emily Elizabeth Falk

A thesis submitted to the faculty of
Brigham Young University
in partial fulfillment of the requirements for the degree of

Master of Science

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ABSTRACT

The Effect of Experimentally-Induced Anterior Knee Pain on Postural Control

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Context: Knee pain is experienced by many people. Because of this, authors have started researching the effects of pain on lower extremity mechanics and also on static and dynamic postural control. However, the effects of pain are difficult to study due to associated confounding variables. **Objective:** We asked: (1) Will experimentally-induced anterior knee pain alter perceived pain using the visual analogue scale? ; (2) will perceived pain affect postural control as measured by center-of-pressure during static and dynamic movement? **Design:** Crossover. **Setting:** Biomechanics laboratory. **Participants:** Fifteen healthy subjects. **Intervention:** Each subject participated in single leg quiet stance, landing, and walking trials under three conditions (pain, sham, control), at three different times for each condition (pre-injection, injection, and post-injection). **Main Outcome Measures:** The dependent variables were measured at pre-injection, injection, and post-injection. Pain was measured using the visual analogue scale across all three times during each condition. Center-of-pressure sway was measured during single leg quiet stance to calculate the average center-of-pressure velocity in the anterior-posterior and medial-lateral directions. The center-of-pressure time to stabilization was measured in anterior-posterior, medial-lateral, and vertical directions, and center-of-pressure trajectory excursion was measured in the medial-lateral direction during walking. **Results:** Perceived pain was significant ($P < 0.05$) but did not affect postural control as measured by center-of-pressure medial-lateral and anterior-posterior sway during single leg quiet stance, in time to stabilization during landing, and in medial-lateral excursion during walking. **Conclusions:** Injection of hypertonic saline resulted in statistically significant perceived pain but did not affect postural control as measured by center-of-pressure medial-lateral and anterior-posterior sway during single leg quiet stance, in time to stabilization during landing, and medial-lateral excursion during walking.

Key Words: postural control, anterior knee pain, hypertonic saline, center-of-pressure

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Introduction

Joint pain is experienced by athletes, adults, the elderly, and others. Impingement, joint effusion, instability,^{1,2} inflexibility,² and overuse injuries cause joint pain. Joint pain is often associated with osteoarthritis,³ where the knee is the most affected weight bearing joint.⁴⁻⁷ Osteoarthritis affects over 27 million Americans⁸ and costs \$5700 per patient per year.⁹ Joint pain is also associated with patella disorders that affect 25% of young adults and more than 25% of athletes.¹⁰

Related to osteoarthritis, patella disorders, and other conditions, joint pain impairs muscle strength,^{7,11,12} overall performance,^{7,13,14} and static and dynamic postural control (PC).^{11,14} PC is “controlling the body’s position in space for the dual purposes of stability and orientation”.^{15,16} Static PC involves stabilizing the body’s base of support during minimal movement like quiet stance,¹⁷ while dynamic PC involves stabilizing the body’s base of support during movement like walking.¹⁸ Static and dynamic PC can be measured by center-of-pressure (COP) movements.^{11,19}

PC requires integration of sensory inputs like proprioception, which senses the body’s position.¹¹ Sensory inputs affect motor components like muscle strength, muscle activation, and contraction patterns.²⁰ Pain can alter sensory inputs, affect motor components, and change overall PC.^{1,21} For example, anterior knee pain (AKP) interferes with nociceptor and mechanoreceptor signals at central processing, delaying the returning efferent messages and altering proprioception.¹ Since proprioception affects the nervous system (providing sensory information on joint position and movement), deficits in proprioception alter various motor outputs like recruitment, patterning, and coordination.^{1,22,23} As these modifications are made due to AKP, the body’s base of support during static and dynamic movement changes to maintain stability, thus altering PC.^{1,22-24} The changes to static and dynamic PC may be observed using

COP: excursion,¹⁹ anterior-posterior and medial-lateral velocities,^{19,25} and time to stabilization.²⁶⁻²⁹

The effects of pain on PC are difficult to study because of confounding variables like inflammatory factors, joint degeneration, and related muscle weakness. These potentially confounding variables make it difficult to prove that altered proprioception is a direct result of pain.³⁰ Also, because joint pain influences many people over the age of sixty-five,³¹ it can be difficult to understand if weakened muscles and poor proprioception are the result of pain or of aging. An experimentally-induced pain model will potentially eliminate these confounding variables by inducing pain in healthy, young individuals. This model will help to better understand the effects pain has on PC.

The purpose of this study is to quantify PC alterations due to experimentally-induced AKP during quiet stance and dynamic movement. An increase in COP anterior-posterior and medial-lateral velocities, time to stabilization, and excursion of the COP will show that pain decreases PC.

Methods

Experimental Design

A counterbalanced cross-over study using a 3x3 analysis to evaluate the influence of condition (pain, sham, control) and time (pre-injection, injection, post-injection) on the following dependent variables: (1) COP anterior-posterior (AP) and (2) medial-lateral (ML) velocity, (3) anterior-posterior time to stabilization (APTTS), (4) medial-lateral TTS (MLTTS), (5) vertical TTS (VTTS), (6) COP trajectory excursion in the ML direction, and (7) perceived pain. The dependent variables except for pain were measured at pre-injection, injection, and post-injection to see if there were differences as a result of pain. Pain was measured before and after each

established condition (pre-injection, injection), and before each test (single leg quiet stance, landing, walking) and after walking. In addition, pain was assessed every 5 minutes for 20 minutes after finishing walking.

Subjects

Fifteen subjects ages 18-26 were recruited from Brigham Young University and completed this study (8 females and 7 males; age = 23 ± 2 yrs; height = 1.71 ± 0.10 m; mass = 73.4 ± 17.3 kg). Each subject's dominant leg was determined by their kicking leg and was used for testing. Most often the right leg was used (93.3%). Screening was done using a questionnaire. To participate, subjects had to be healthy, physically active (exercising a minimum of 3 times a week for 30 minutes), have no current lower extremity pathology, have no current muscle or joint pain, and have no history of surgery with the involved limb. This study was approved by Brigham Young University's Institutional Review Board before recruiting. A consent form was signed by each subject prior to participating.

Instrumentation

Anterior Knee Pain Model

A one-time injection using a 25 gauge needle was inserted at a depth of 10 mm into the lateral aspect of the infrapatellar fat pad on the subject's dominant limb. A 1 ml syringe (Becton Dickinson Medical Systems Inc, Franklin Lakes, NJ) was filled with 5% hypertonic saline (5% sodium chloride, Baxter Healthcare Corporation, Deerfield, IL) or isotonic saline solution (0.9% sodium chloride, Hopsira, Inc., Lake Forest, IL). Isotonic saline is a physiologic neutral solution used to ensure the irritation of the fat pad is due to the hypertonic saline solution and not the mechanical effects of the injection.

Instruments

A force plate (AMTI Force and Motion, Watertown, MA) measured the COP during each subject's single leg quiet stance and landing off a 31.1 cm height (200 Hz). The AP and ML velocities, and APTTS, MLTTS, VTTS were then calculated using the COP data that were collected during stance and landing. F-scan insole (Tekscan Inc., Boston, MA) pressure sensors (3.9 sensels per cm²) measured plantar pressures during walking (100 Hz). The insoles were custom fit to the subject's shoes and used to measure plantar pressures and compute the COP trajectory excursion in the ML direction during walking. A treadmill (Quinton Instrument Co., Bothell, WA) was used for warm-up walking and testing. Subjective pain perception was quantified using a 10 cm visual analogue scale (VAS). The VAS has been shown to be a reliable method of measuring pain.³²

Procedures

Test set-up procedures

Subjects reported to the lab wearing shorts. Subjects were weighed to calibrate the F-scan insoles and their leg length measured (ASIS to medial malleolus) to standardize walking speed.³³ Each subject participated in 3 conditions in a counterbalanced order: (i) injection of hypertonic saline solution (5%), (ii) injection of isotonic saline solution (0.9%), and (iii) no injection. There were 3 test days with 2 to 4 days in between test days. Within each condition, force plate data for single leg quiet stance and landing, as well as the plantar pressures during walking were recorded at pre-injection, injection, and post-injection.

All subjects used the Nike T-Lite shoe for testing. The insoles of the Nike T-Lite shoe were removed and replaced with a custom fit F-scan pressure insole. Subjects warmed up

walking on the treadmill for 5 minutes at the standardized walking speed. After warm-up, the F-scan pressure insoles were calibrated using the manufacturer's directions.

Treatment Conditions

Pain and Sham Conditions

After the warm-up, subjects laid supine on a treatment table. The lateral side of the knee, inferior to the patella was sterilized using a Povidone-Iodine Swabstick (10% solution, Professional Disposables, Inc., Orangeburg, NY). After swabbing the area and allowing it to dry, 0.75 ml of solution was injected laterally, to a 10-mm depth, into the subject's infrapatellar fat pad of the dominant leg using a 25 gauge needle at a 20° angle in a superolateral direction.^{1,30} To spread the solution throughout the infrapatellar fat pad, the needle was moved around at several angles inside the fat pad while the solution was injected. After the injection, the subject remained laying supine for 30 seconds, then sat up for 30 seconds, and stood for 30 seconds, all to avoid nausea.

Control Condition

After the warm-up, subjects laid supine on the treatment table. No saline solution was injected into the infrapatellar fat pad. The subject lay supine for 30 seconds, then sat up for 30 seconds, and stood for 30 seconds.

Data Collection

Single Leg Quiet Stance Test

After the condition was established, force plate data for a single leg quiet stance of the dominant leg was recorded for 3 trials, each lasting 30 seconds.²⁵ Fifteen seconds of rest was given in-between each trial. During stance, subject had their hands on their hips, their eyes open looking straight ahead, and their non-dominant leg raised off the ground. If the subject touched

the ground during the trial with their non-dominant leg, the trial was tallied and rerecorded. Subjects had a single practice for the single leg quiet stance prior to their 5-minute warm-up. Subjects performed the practice without difficulty.

Landing Test

Ground reaction force applied to the dominant leg while landing off a 31.1 cm platform was recorded for 3 trials.²⁷ The subject had their hands on their hips with their eyes open, looking straight ahead. The subject was not allowed to lower their dominant leg to the force plate or jump off the platform. The subject practiced landing on the force plate prior to their 5-minute warm-up until they felt comfortable with the task.²⁸ The subjects were directed to stabilize as quickly as possible after landing and had to remain standing for 6 seconds.²⁷ Fifteen seconds of rest was given in-between each trial. If the non-dominant leg touched the force plate during the trial, the trial was tallied and rerecorded.

Walking Test

Using the pressure insoles, COP was measured for 15 seconds while walking on the treadmill with no incline. The subject then sat for 20 minutes while pain subsided. After 20 minutes of rest, the same single leg quiet stance, landing, and walking trials were performed. These were the post-injection trials and were compared to the pre-injection and injection trials.

Data Reduction

AP and ML average velocities were derived from the force plate data during single leg quiet stance (COP distance over time). The COP velocity was derived for each instant in time over the entire 30-second trial (200 Hz). All COP velocities were then averaged. APTTS, MLTTS, and VTTS were derived from the force plate data during the landing trials.^{28,29} A sequential estimation was calculated for each direction (AP, ML, V) to find a stabilization time.

A subject was considered stable when the sequential estimation remained within 0.25 standard deviations of the overall series mean and the vertical GRF remained within 5% of the subject's body weight.^{26,27,34}

For walking trials, heel strike and toe off were visually identified and then the COP trajectory excursion was evaluated in the ML direction. Heel strike was identified as the first sensel that detected force at the heel and toe off was the last sensel that detected force at the toes. After identifying three stance phases from the 15 seconds of recorded walking data, each stance phase was time normalized to 100 samples, then averaged.

Statistical Analysis

A 3x10 mixed model analysis of variance (ANOVA) and Tukey *post hoc* ($P < 0.05$) were used to determine significant differences in pain. A 3x3 mixed model analysis of variance (ANOVA) was used to evaluate the influence of the independent variables on the following dependent variables ($P < 0.05$): COP AP and ML velocities, APTTS, MLTTS, and VTTS. A functional ANOVA was used to determine differences between conditions and times with respect to COP excursion during walking ($P < 0.05$). This analysis allowed us to compare variables as polynomial functions rather than discrete values over the entire stance phase of each movement. Recorded measurements in all 3 conditions and times were compared to see if pain had an effect on PC.

The significant level was chosen as ≤ 0.05 . We used the software SAS 9.2 (SAS Institute Inc., Cary, NC) for all data analyses except COP excursion during walking. We used R 2.14.0 for COP excursion during walking.

Results

Injection of 5% hypertonic saline solution increased perceived pain (interaction: $F_{18,46} = 14.13$, $P < 0.01$). Perceived pain started directly after the injection of hypertonic saline solution and remained significant until 5 minutes after testing was finished ($P < 0.05$; Figure 1). Testing lasted an estimated 6-7 minutes. The summary data of the COP, AP, and ML, velocities during quiet stance and APTTS, MLTTS, and VTTS after landing for each condition over time are shown in Figures 2-6. We did not find any significant changes in COP AP (interaction: $F_{4,112} = 0.94$, $P = 0.45$) and ML (interaction: $F_{4,112} = 0.55$, $P = 0.70$) average velocities during single leg quiet stance, or APTTS (interaction: $F_{4,112} = 0.16$, $P = 0.96$), MLTTS (interaction: $F_{4,112} = 0.39$, $P = 0.82$), and VTTS (interaction: $F_{4,112} = 0.27$, $P = 0.90$) after landing among the three conditions. We also did not detect any significant changes to the ML COP trajectory during the stance phase of walking (Figures 7-8).

Discussion

The primary objective of our study was to examine the effects of AKP on static and dynamic PC using an experimentally induced pain model. To quantify PC, we measured the COP by ML and AP sway during single leg quiet stance, in TTS during landing, and ML excursion during walking. Our results showed that statistically significant experimentally-induced AKP does not significantly affect PC, as measured by COP changes. This is evidenced by the insignificant changes to COP in AP or ML velocities during single leg quiet stance, in TTS during landing, and in the COP trajectory excursion of the stance phase during walking.

Pain measurements

We injected 0.75 ml of hypertonic saline solution to induce knee pain. We reported a pain average of 2.9 cm on a 10 cm VAS scale and subjects were pain free within 20 minutes after the

injection. In addition, our pilot data showed an average of 2.65 cm using 0.25 ml hypertonic saline (5%). A single injection of 0.25 ml^{1,13,23,30}, 0.75 ml³⁵, and 1.0 ml³⁶ 5% hypertonic saline have been used to induce experimental knee pain. Bennell et al.³⁰ and Hodges et al.¹³ used single injections of 0.25 ml hypertonic saline solution (5%) to induce pain and reported that pain peaked within two to three minutes after the injection. Our data were slightly inconsistent with Bennell et al.³⁰ who reported a pain average of 5.8 using an 11-point NRS scale and also found subjects to be pain free fifteen minutes after the injection.¹³ Consistent with our data, Henriksen et al.³⁵ used a single injection of 0.75 ml hypertonic saline (5%) and had an average 2.58 cm on the VAS scale. We are unsure why our pain average is inconsistent with Bennell since our injections were similar but with different amounts of hypertonic saline. Our study using 0.75 ml hypertonic saline had a greater effect on perceived pain than our pilot data using 0.25 ml hypertonic saline.

Single leg quiet stance

An increase in COP velocity suggests a decrease in PC.¹⁹ We found no differences in COP as measured by the average AP or ML velocities during single leg quiet stance. Our COP AP average velocities (m/s; mean \pm SD) for the pre-injection (or baseline) time across the 3 conditions (control, sham, pain) were 0.026 ± 0.006 , 0.026 ± 0.005 , and 0.026 ± 0.006 . Our ML average velocities (m/s; mean \pm SD) were 0.030 ± 0.006 , 0.030 ± 0.004 , and 0.029 ± 0.003 . Salavati et al.²⁵ reported an average COP AP velocity (cm/s; mean \pm SD) of 1.29 ± 0.48 , and an average COP ML (cm/s; mean \pm SD) of 1.48 ± 0.31 . In addition, Hirata et al.³⁷ reported COP AP average velocities (cm/s; mean \pm SD) for the baseline time across 4 conditions (injection of hypertonic saline in (1) vastus medialis, (2) vastus lateralis, and (3) biceps femoris muscles, and (4) control [isotonic saline injection into vastus medialis muscle]) to be 5.5 ± 0.4 , 6.1 ± 0.9 , $5.2 \pm$

0.3, and 5.2 ± 0.3 while observing experimental muscle pain and postural stability. Their COP ML average velocities (cm/s; mean \pm SD) were 1.7 ± 0.2 , 2.5 ± 0.3 , 2.3 ± 0.3 , and 2.1 ± 0.4 . Our AP average velocities were twice as much as Salavati but half as much as Hirata. Our ML average velocities were twice as much as Salavati and were similar but more than Hirata. Differences may be due to Salavati and Hirata's use of a bilateral quiet stance. In addition, Salavati was observing a painfree musculoskeletal population (low-back pain, ACL injury, functional ankle instability) and did not test healthy individuals, however, he used similar methods of averaging 3 trials of 30 second stance.²⁵ Hirata recorded one base 60 second bilateral quiet stance trial (1000 Hz compared to our 200 Hz) before each randomized condition. In each testing session he observed 2 conditions, where the second condition was tested 60 minutes into the session.³⁷ So, a baseline 60 second stance trial was not recorded until 60 minutes into the session for another condition. The subject may have been fatigued by the forward and backward perturbations of the force platform that were applied in the previous 60 minutes. With these differences, our COP average velocities still fall within the range of other average velocities previously reported.

Our findings are consistent with the idea that pain has no effect on static PC.²³ In further support of this idea, Bennell et al.²³ observed a bilateral quiet stance during experimental knee pain, and found no differences in balance or static PC while measuring COP displacement. Attributed by Bennell et al.,²³ no changes were made to static PC because the noninvolved leg was compensating for the induced leg during the bilateral quiet stance. However, our results for a single leg quiet stance show no differences in static PC which does not support Bennell's idea. It could be noted COP's average AP and ML velocities may not be sensitive enough to uncover differences in static PC when observing the effects of pain.

Landing

An increase in TTS suggests a decrease in stability or PC. We did not observe any significant differences in APTTS, MLTTS, or VTTS, after inducing pain, during a landing task, indicating that pain does not have an effect on dynamic PC, as measured by TTS. Our APTTS (sec; mean \pm SD) for the control condition across time (pre-injection, injection, post-injection) were 2.77 ± 0.72 , 2.90 ± 0.61 , and 2.93 ± 0.73 . Gribble²⁸ reported an APTTS (sec; mean \pm SD) of 1.34 ± 0.16 for a control group while comparing a chronic ankle instability group. Our MLTTS (sec; mean \pm SD) for the control condition across time (pre-injection, injection, post-injection) were 2.93 ± 0.67 , 2.99 ± 0.65 , and 2.90 ± 0.62 , whereas Gribble reported a comparable MLTTS of 2.51 ± 0.93 for his control group.²⁸ Our APTTS was twice as much as Gribble. These differences may be due to variations in the jump-landing method. Gribble used a vertical jump that consisted of a double-leg take off with a single leg landing onto a force plate 70 cm away. The vertical jump was 50% of the subject's vertical maximum.²⁸

To our knowledge, TTS has not been used as a measurement of dynamic PC in an experimental pain model. Several authors have reported that TTS is a sensitive and effective measurement of dynamic PC while studying fatigue and chronic ankle instability.^{28,38,39} Because of detected differences in these populations, TTS might have been a valuable method of measuring the effects of pain on dynamic PC. Changes to dynamic PC as measured by TTS might not have been observed due to compensatory mechanics utilized to relieve or avoid knee pain. For example, joint moments at the ankle, knee, and hip may have been reduced in certain planes while increased in others to help avoid painful positions of the knee.³⁵ This loading alteration could have been partially transmitted to and absorbed by joints other than the knee in the lower extremity. If this were the case, it implies that the PC system is making modifications

to the lower extremity to compensate for knee pain, but TTS is not a sensitive measurement to show these alterations. Furthermore, alterations at various individual joints could be occurring simultaneously as a result of pain but are not being observed in the TTS measurement because it is dependent upon the ground reaction force (GRF). The GRF only reflects the acceleration of the whole body center-of-mass, and alterations at multiple various lower-extremity joints could potentially cancel each other out. This idea may also apply to walking. Therefore, kinetic and kinematic data for joints above and below the knee are needed to support these ideas.

Walking

An increase in COP excursion during walking suggests a decrease in PC or balance.¹⁹ Our results showed that there were no changes to the COP trajectory excursion in the ML direction during the stance phase of gait. This is consistent with the idea that pain does not have an effect on dynamic PC as measured by COP ML excursion. However, Henriksen et al.³⁵ did find reduced knee joint adduction, flexion, and extension moments during walking using the present pain model.³⁵ Their results are comparable to less severe OA patients. In addition, the unloading that occurred in their study during walking is similar to OA patients.³⁵ The reduced knee joint moments found by Henriksen et al.³⁵ may be a result of compensation in the lower chain to relieve pain by decreasing the amount of loading to the involved leg.

Furthermore, experimental pain produces weak knee muscles (specifically the quadriceps) as found in OA patients.^{13,36} Henriksen et al.³⁶ discovered acute knee pain decreases muscle strength during knee flexion and extension. This change to muscle strength around the knee causes new timing and activation patterns, as well as altered loads in the lower chain.¹³ Alterations in joint loading could cause further joint degeneration and increased pain.⁴⁰ Although no differences in COP were found during static and dynamic activity, others have

reported differences as mentioned that could affect dynamic PC. Henriksen et al.³⁵ found that pain reduced knee joint adduction, flexion, and extension moments, and decreased muscle strength, however, the present study did not detect these alterations in the COP trajectory excursion as measured in the ML direction during walking. We did not measure the COP trajectory excursion in the AP direction. These changes found by Henriksen et al.^{35,36} in the sagittal plane might have been detected in the AP COP trajectory excursion of the stance phase if measured. Although we did not see the PC changes that are supported by Henriksen et al.,^{35,36} our intentions were to look at ML excursion. The COP may not be an effective method of measuring the effects of pain on dynamic PC in the ML direction.

The effects of pain on PC are difficult to study due to associated confounding variables. Thus, an experimental pain model has been created to eliminate these variables. The pain model is complex because it has to closely mimic osteoarthritis and anterior knee pain.³⁰ The infrapatellar fat pad and joint capsule have nociceptors spread throughout making the fat pad sensitive to pain.⁴¹ Nerve fibers in the fat pad contain substance-P, a protein that triggers nociceptors, which is consistent with musculoskeletal nociception.^{30,42,43} Similar pain is produced using the experimental pain model. Hypertonic saline solution is injected into the infrapatellar fat pad, releasing substance-P from nerve fibers, targeting and causing chemical irritation to the nociceptors.⁴⁴ Animal experiments have shown group III and IV nociceptive afferents are stimulated by hypertonic saline, which means the pathways used by the pain model are consistent with musculoskeletal pain.^{45,46} Though experimental pain is similar to AKP and OA patients, there are still differences in the quality of pain. Pain in this model does not last long. Pain lasted an estimated 12 minutes in our study and caused no changes to PC. If pain levels remained longer, changes may have been observed. Bennell et al.²³ thought that it is

possible that the nociceptors in the infrapatellar fat pad need longer stimulation to make changes to PC. We speculate that changes to PC could be made after stimulating the nociceptors for about an hour.

Further Research

We need to better understand how the pain model affects PC. PC is a complex process and components of PC were found to be affected by pain as shown by Henriksen et al.^{35,36} This study should be repeated using lower extremity EMG to observe muscle activation of certain lower-extremity muscles, especially the anti-gravity muscles (i.e. quadriceps and soleus), in correspondence with lower extremity joint kinematics and kinetics. Further measurements of COP other than velocity and TTS need to be observed to see if they are more sensitive to changes in static and dynamic PC. Experimental pain should also be studied alongside other controlled confounding variables like effusion to see if there are changes in PC as measured by COP. Furthermore, COP should be studied alongside strength to see how they contribute to PC limitations.

Limitations

Our study did not consider the possible accompanying compensatory alterations at the knee joint or other joints in the lower chain after inducing experimental knee pain. There were no COP differences but this may be due to alterations in the recruitment strategies and patterns of the lower extremity mechanics that were not recorded (i.e. soleus facilitation and quadriceps inhibition). In addition, TTS has limitations. Time to stabilization measures postural stability in 3 directions (AP, ML, V) which may not be a functional outcome measure of stability since it is not a global measurement.²⁶ However, TTS may be beneficial since it is sensitive to postural stability in each direction. Furthermore, the pain model is limited to nociceptive stimulation of

the infrapatellar fat pad and not nociceptive stimulation of the knee joint capsule or other structures surrounding the knee that are included in clinical AKP. Lastly, we observed a low intensity pain and do not know the effects of a high intensity pain.

Conclusion

In conclusion, injection of hypertonic saline significantly increased perceived pain but did not significantly alter static or dynamic PC, as measured by COP in ML and AP sway during single leg quiet stance. Injection of hypertonic saline did not significantly influence TTS during landing or ML trajectory excursion during walking. The present measures that were derived from COP data may not be an effective measurement in showing the effects of experimental knee pain on PC because it is not sensitive to the alterations already found by authors in the lower extremity.

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Legend of Figures

Figure 1. Pain perception. Perceived pain (hypertonic) was statistically significant ($P < 0.05$) at injection until 5 minutes post-injection (7). Time on the graph represents (1) pre-injection, (2) injection, (3) pre-stance, (4) pre-land, (5) pre-walk, (6) post-walk, (7) 5-min. post, (8) 10 min. post, (9) 15 min. post, and (10) 20 min. post.

Figure 2. Center-of-pressure anterior-posterior average velocity. The center-of-pressure anterior-posterior average velocity (m/s) was not statistically significant ($P < 0.05$) over three times (pre-injection, injection, and post-injection), between three experimental conditions (control, sham, and pain).

Figure 3. Center-of-pressure medial-lateral average velocity. The center-of-pressure medial-lateral average velocity (m/s) was not statistically significant ($P < 0.05$) over three times (pre-injection, injection, and post-injection), between three experimental conditions (control, sham, and pain).

Figure 4. Anterior-posterior time to stabilization. The anterior-posterior time to stabilization was not statistically significant ($P < 0.05$) over three times (pre-injection, injection, and post-injection), between three experimental conditions (control, sham, and pain).

Figure 5. Medial-lateral time to stabilization. The medial-lateral time to stabilization was not statistically significant ($P < 0.05$) over three times (pre-injection, injection, and post-injection), between three experimental conditions (control, sham, and pain).

Figure 6. Vertical time to stabilization. The vertical time to stabilization was not statistically significant ($P < 0.05$) over three times (pre-injection, injection, and post-injection), between three experimental conditions (control, sham, and pain).

Figure 7. Functional analysis of center-of-pressure trajectory (control vs. pain). The functional analysis of center-of-pressure trajectory of the stance phase during walking was not statistically significant ($P < 0.05$) when observing control vs. pain. The red line represents the effect of pain on control and the vertical axis represents differences in x coordinate data. Shaded areas equal 95% confidence. The center-of-pressure trajectory is not statistically significant in control vs. pain because the confidence interval never goes above or below the zero line.

Figure 8. Functional analysis of center-of-pressure trajectory (sham vs. pain). The functional analysis of center-of-pressure trajectory of the stance phase during walking was not statistically significant ($P < 0.05$) when observing sham vs. pain. The red line represents the effect of pain on sham and the vertical axis represents differences in x coordinate data. Shaded areas equal 95% confidence. The center-of-pressure trajectory is not statistically significant in sham vs. pain because the confidence interval never goes above or below the zero line.

Figure 1.

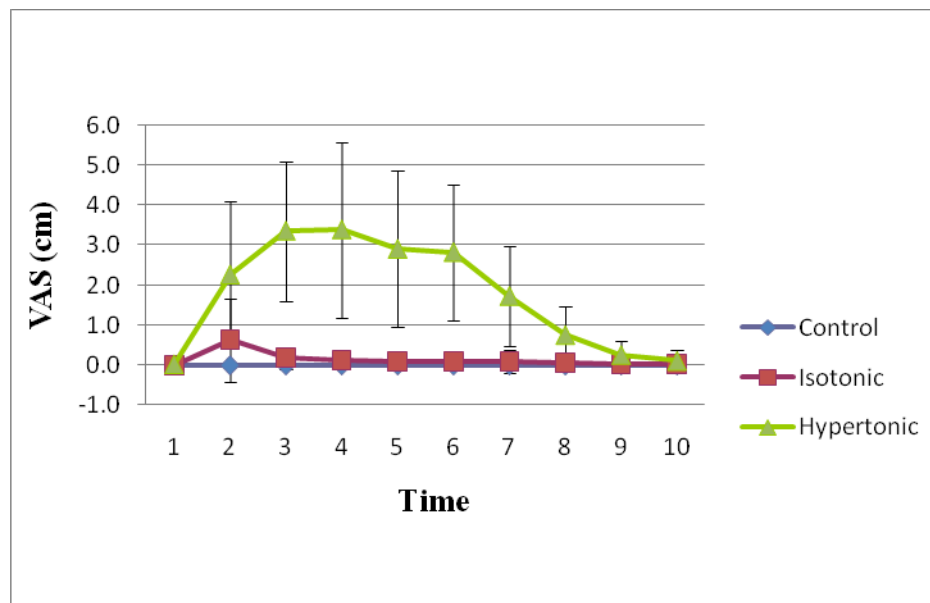


Figure 2.

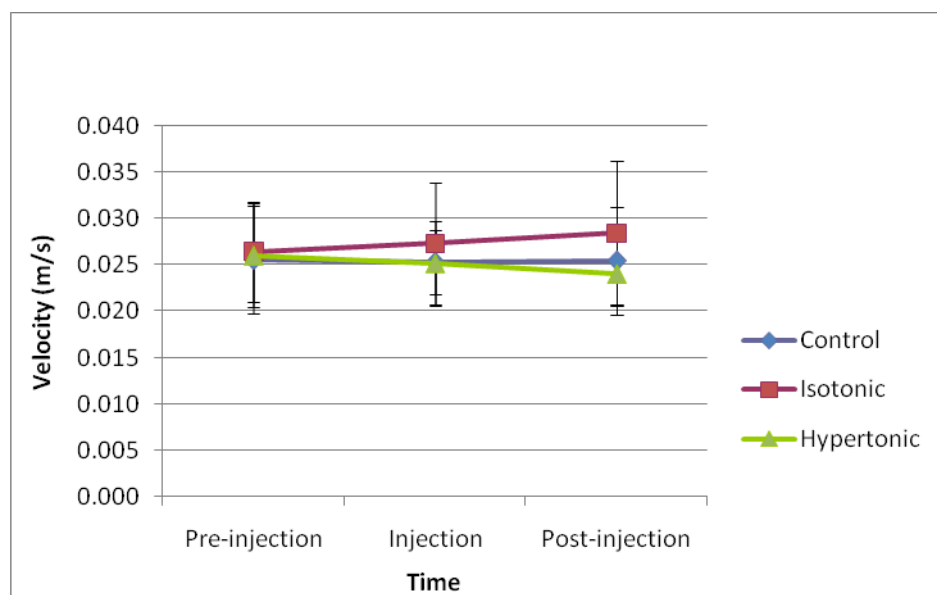


Figure 3.

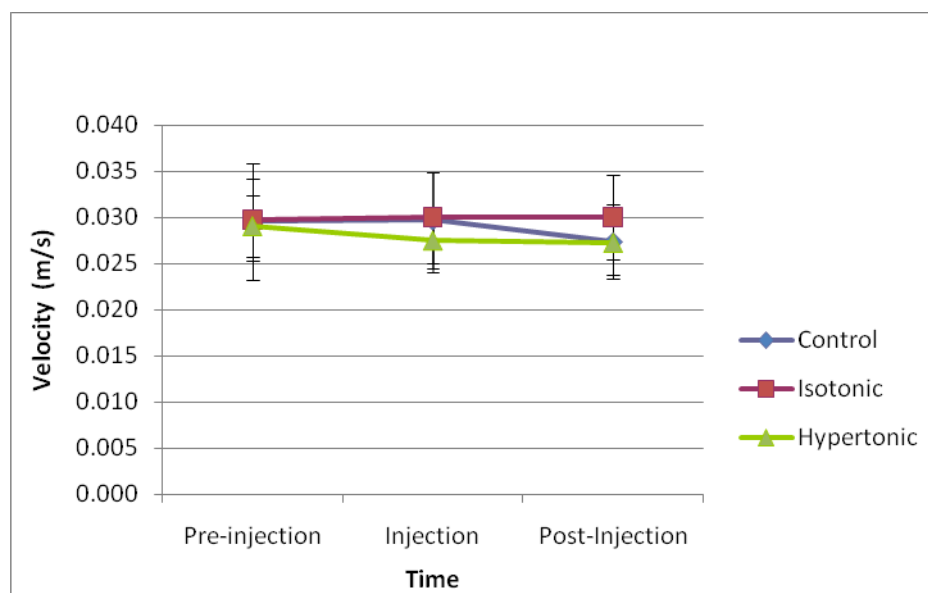


Figure 4.

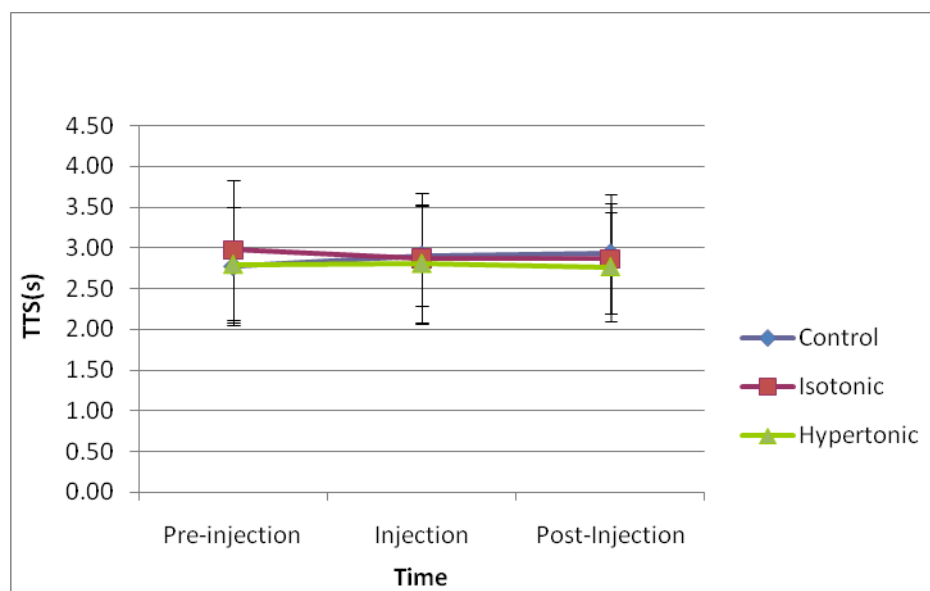


Figure 5.

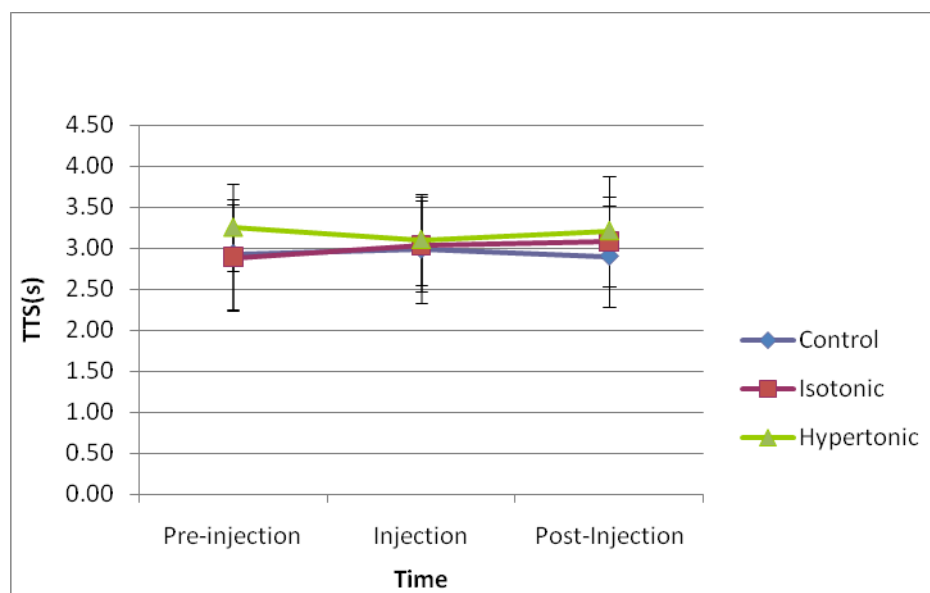


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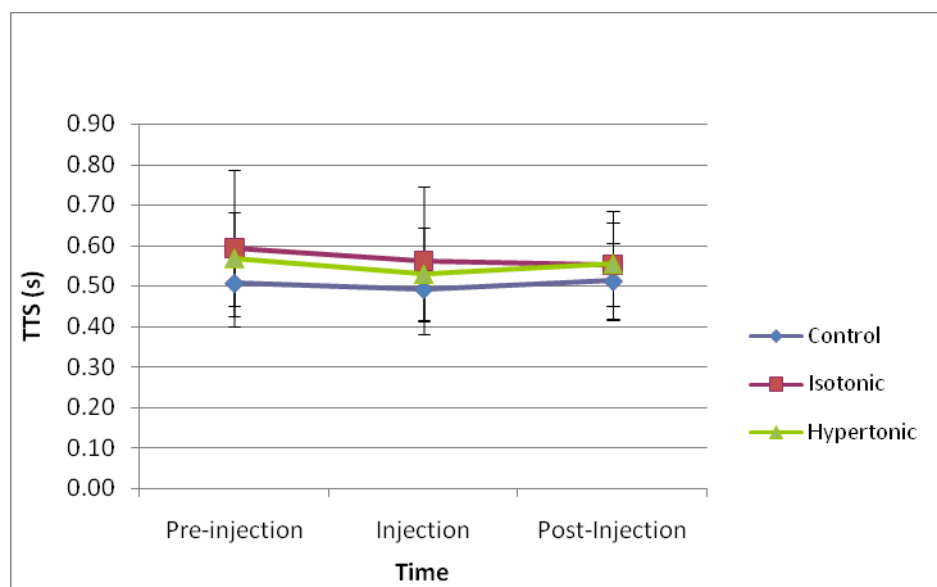


Figure 7.

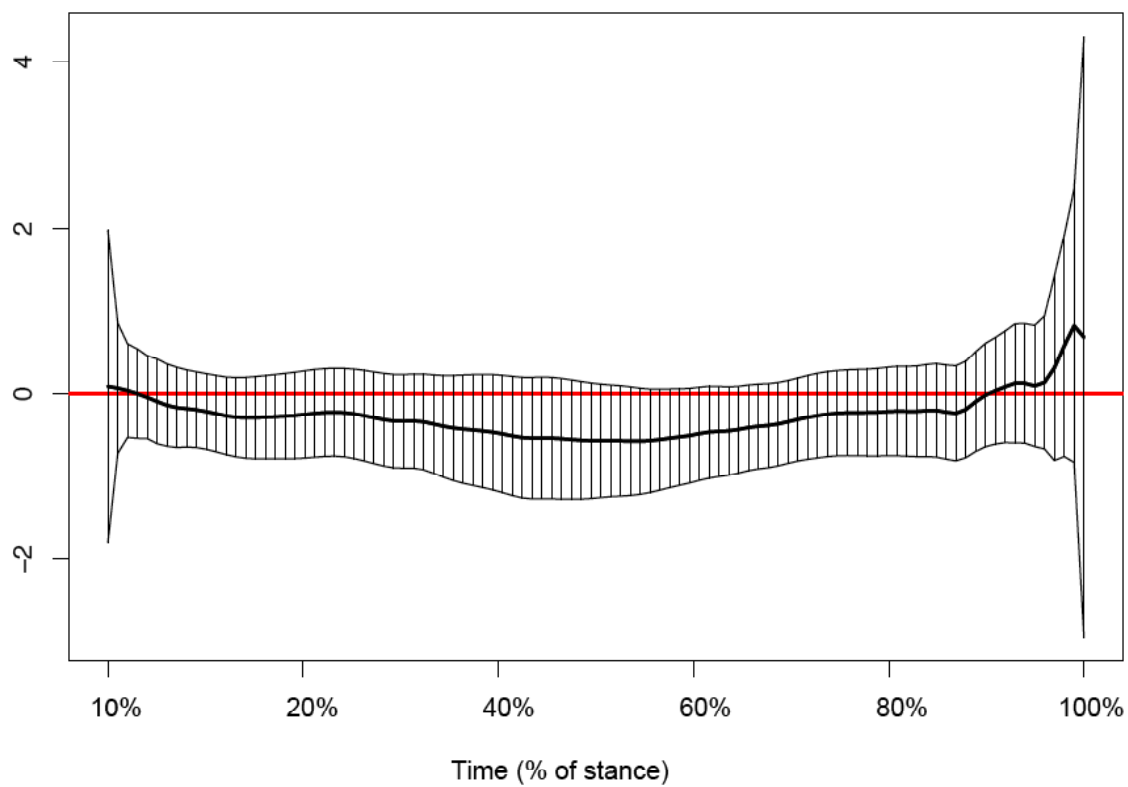
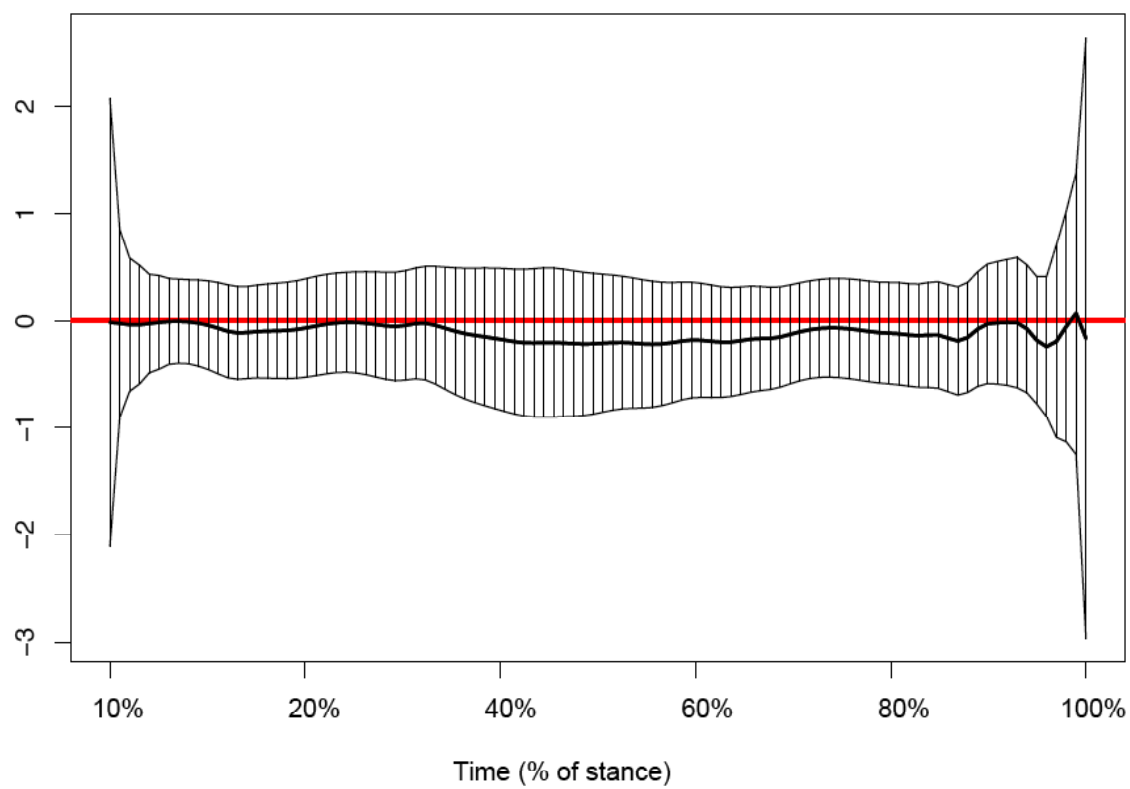


Figure 8.



Prospectus

Chapter 1

Introduction

Joint pain is experienced by various populations: athletes, adults, and the elderly. Causes of joint pain are impingement, joint effusion, instability^{1,2}, inflexibility², and overuse injuries. Often, joint pain is associated with osteoarthritis³, where the knee is the most affected weight bearing joint.⁴⁻⁷ Osteoarthritis affects over 27 million Americans⁸ and costs \$5700 per patient per year⁹. In addition, joint pain is associated with patella disorders which are found in about 25% of young adults, with a higher percentage revealed among athletes.¹⁰

Related to osteoarthritis, patella disorders, and other conditions, joint pain impairs body movement, muscle strength^{7,11,12}, overall performance^{7,13,14}, and static and dynamic postural control (PC).^{11,14} PC is “controlling the body’s position in space for the dual purposes of stability and orientation”^{15,16}. Static PC involves stabilizing the body’s base of support during minimal movement like quiet stance¹⁷, while dynamic PC involves stabilizing the body’s base of support during movement like walking¹⁸. One method of evaluating static and dynamic PC is by measuring center of pressure (COP) movements.^{11,19}

PC requires integration of sensory inputs like proprioception, balance, and vision.¹¹ These sensory inputs affect motor components like muscle strength, muscle activation, and contraction patterns.²⁰ Pain can alter sensory inputs, affect motor components, and change overall PC.^{1,21} For example, anterior knee pain (AKP) causes interference of nociceptor and mechanoreceptor afferent signals at central processing, which produces a delay in returning efferent messages, thus altering proprioception.¹ Since proprioception affects the nervous system (providing sensory information on joint position and movement), deficits in proprioception can alter various motor

outputs like recruitment, patterning, and coordination.^{1,22,23} As these modifications are made due to AKP, the body's base of support during static and dynamic movement will change to maintain stability, thus altering PC.^{1,22-24} The changes to static and dynamic PC may be observed using COP: excursion¹⁹, anterior-posterior and medial-lateral velocities^{19,25}, time to stabilization.²⁶⁻²⁹

The effects of pain on PC can be difficult to study. Bennell states that studying the effects of pain on PC in subjects already suffering from knee pain is difficult since there are confounding variables³⁰ like inflammatory factors, joint degeneration, and other injuries; i.e., the confounding variables make it difficult to prove that altered proprioception is a direct result of pain. Also, as joint pain influences a large population over the age of 65³¹, it can be difficult to understand if weakened muscles and poor proprioception are a result of pain or aging. An experimentally- induced pain model will potentially eliminate the aforementioned confounding variables by inducing pain in healthy, young individuals. This model will help to better understand the effects pain has on PC.

The purpose of this study is to quantify PC alterations due to experimentally-induced AKP during quiet stance and dynamic movement. An increase in COP anterior-posterior and medial-lateral velocities, an increase in time to stabilization, and excursion of the COP will show pain has an effect on PC.

Null Hypothesis

The following null hypothesis will be tested:

There will be no change in the COP anterior-posterior and medial-lateral velocities, COP's time to stabilization, or COP excursion during quiet stance and dynamic movement after inducing AKP.

Delimitations

This study will be delimited to:

1. The knee joint;
2. Anterior knee pain (AKP), infra-patellar fat pad;
3. Hypertonic and Isotonic saline solution;
4. F-scan, Tekscan, pressure insoles;
5. Force plate (AMTI);
6. Single leg stance, landing, walking;
7. College age adults;

Limitations

1. Results may only be applied to a similar population.
2. Results may only be applied to acute pain.

Terminology

Postural Control – the body's ability to control its position and be stabilized.^{15,16}

Center of Pressure (COP) – is the average location of all plantar pressures.

Center of Pressure (COP) excursion – the amount the subject journeyed from the baseline COP during activity; quantifies the subject's amount of PC.

Time to Stabilization (TTS) – an objective measure of dynamic PC²⁶. TTS is the time it takes the landing GRFs to get within a range of the baseline GRFs of the static stance.²⁶

Anterior-posterior TTS, medial-lateral TTS, and vertical GRF will be analyzed to see if there are differences after inducing pain.

Pain – The pain of each subject will be assessed using a 10 cm visual analog scale (VAS) that uses descriptors of ‘no pain’ and ‘pain as bad as it could possibly be’. Any mark above 0 represents pain.

Chapter 2

Review of Literature

Joint pain is experienced by various populations: athletes, adults, and the elderly. Some causes of joint pain are impingement, joint effusion, instability^{1,2}, inflexibility², and overuse injuries. Often, joint pain is associated with osteoarthritis³, where the knee is the most affected weight bearing joint.⁴⁻⁷ Osteoarthritis affects over 27 million Americans⁸ and costs \$5700 per patient per year.⁹ In addition, joint pain is associated with patella disorders which are found in about 25% of young adults, with a higher percentage revealed among athletes.¹⁰

Joint pain can cause severe changes to the lower extremity, like an altered gait pattern which will then shift loading to different areas of the joint. This altered loading, seen in osteoarthritic patients, may lead to additional pain and degeneration to other areas of the joint. Moreover, joint pain impairs body movement, muscle strength^{7,11,12}, overall performance^{7,13,14}, and static and dynamic postural control (PC).^{11,14} PC is particularly important since it contributes to the overall stability and orientation of the body.^{15,16} Static PC involves stabilizing the base of support during minimal movement like stance¹⁷, while dynamic PC involves stabilizing the base of support during movement of the body like gait.

PC requires integration of sensory inputs like proprioception, balance, and vision.¹¹ These sensory inputs affect motor components like muscle strength, muscle activation, and contraction patterns.²⁰ Pain has been shown to alter sensory inputs, which then affects the motor components. This causes alterations in the body's center of mass (COM) and center of pressure (COP) to stay stabilized, which changes overall PC.^{1,21} The purpose of this study is to quantify PC alterations in subjects experiencing experimentally-induced AKP during stance and dynamic

movement. An increase in COP anterior-posterior and medial-lateral velocities, an increase in time to stabilization, and excursion of the COP will show pain has an effect on PC.

Postural Control

PC is “controlling the body’s position in space for the dual purposes of stability and orientation”.^{15,16} Synonyms for PC are stability and equilibrium.⁴⁷ PC can be described as static or dynamic. Static PC involves stabilizing the base of support during minimal movement like standing¹⁷, while dynamic PC involves stabilizing the base of support during movement of the body’s limbs, or movement of the whole body like walking, running, or performing activities of daily living (ADL).

PC is affected by vestibular, visual, and somatosensory systems which work in accordance with the nervous system to balance a person’s COM.^{11,22,24,48} Somatosensory systems are systems that incorporate sensory stimuli from the skin and deep tissue like muscle and organs. Consequently, PC is directly affected by proprioception. In addition to vision and proprioception, other variables that affect PC are balance, muscle strength, and inflexibility or muscle tightness.

Proprioception

Hurley defines proprioception as, “the conscious and unconscious awareness of body position, movement and forces acting on the body”.⁴⁹ Proprioception is the ability of a joint to sense its position (using mechanoreceptors) during limb movement.¹ Other than mechanoreceptors, organs included in detecting body position are muscle spindles and Golgi tendon organs.⁴⁹ Muscle spindles sense when a muscle is being stretched while Golgi tendon organs sense the tension of the muscle contraction.¹⁸

Proprioception includes the initial sensory stimuli and the resultant muscle contraction leading to body movement. This occurs starting with the mechanoreceptor, Golgi tendon, or muscle spindle, which is the afferent signal to central processing or nervous system that analyzes the signal.¹⁸ Then, central processing sends an efferent signal to the muscles, producing a muscle contraction and body movement. This is called neuromuscular control (NC). NC is the resultant efferent message to the muscles given from central processing after integrating and analyzing the afferent messages from the proprioceptors. Proprioception helps guide central processing with body movement and the magnitude of movement needed to keep the base of support stable.⁴⁸

The nervous system is responsible for producing muscle force and the amount of muscle force or muscle contraction produced and the timing of the muscle activation. In effect, the nervous system, with help from the sensory stimuli, is controlling a person's COM or the base of support.²² If the muscles force changes, the COM or base of support will change thus shifting balance and PC of the body.

The body uses proprioception as it moves in gait, running, ADLs, etc. It is continually giving feedback so the body can balance and stabilize its COM. In addition, proprioception may help protect the joint. Looking at the knee joint, proprioception may control the muscles surrounding the joint, which allow for a smooth, low peak load during heel strike of gait.⁵⁰ As stated earlier, PC is being able to control the body's position to stay balanced, so, proprioception is giving constant feedback in order for PC to control the body's position.

Vision

PC is influenced by vision.^{14,22,24,48} Just like proprioception gives feedback to the nervous system to move the body, vision also gives feedback to the nervous system to assist in

body movement. Vision also assists in the body's spatial awareness. Since PC is affected by visual input, studies have performed testing with eyes closed and/or open to compare differences in balance.^{11,14,21,23} The results of the studies tend to find subjects more off balance with their eyes closed. This proves visual input's influence on balance and PC.

Balance

PC and balance are related in that they both rely on sensory input. In addition, they both rely on muscle strength for proper body movement. Balance relies on sensory inputs to stabilize the COM and stay upright to avoid falling.⁵¹ Balance is being able to control the body's COM against external forces during static and dynamic movement.⁵² As defined by Horak⁵³, balance "is the ability to maintain the center of gravity within the limits of stability as determined by the base of support". With axillary movement or whole body movement such as gait, the body is continually adjusting its COM to stay balanced.⁵³ As the COM changes, COP also changes.

The COP is measured by the ground reaction forces of the body.¹⁹ The COP is the center of distribution of the pressure divided by the total ground reaction forces.⁵⁴ This would be different between 2-foot stance and 1 foot stance.⁵¹ The center of gravity (COG) is different from the COP. The COG is the vertical location of the COM.^{19 51} The COG changes as the body moves. The COP changes depending on where the COG is.¹⁹ Falling occurs if the base of support is not under the COG.^{19 55} This deals with postural sway. Postural sway refers to the amount of change that occurs in the COG.⁵¹ Many authors talk about postural sway as a way to measure static PC or poor balance.²⁴

The COP excursion is the amount the COP traveled in a given time.¹⁹ When studies look at the amount of excursion during testing, an increased amount of excursion means there is an increased instability.⁵⁶ As pointed out in the review by Palmieri¹⁹, an increase in excursion

could mean increases in alterations needed to be made in the body in order to stay balanced or keep PC, not that there is instability. Palmieri says COP excursion has not been proven to represent changes in PC.¹⁹

Muscle Strength

Muscle strength is needed for controlled balance and smooth body movement.¹¹ The nervous system is responsible for producing muscle force and the amount of muscle force or muscle contraction produced. This is a direct effect of proprioception since the amount of muscle force is determined from the sensory input. Improper timing and activation of the muscles causes an abnormal use of the muscles which decreases strength and changes body mechanics.

Looking at the knee joint, improper timing and activation of the muscles instigates a misuse of the muscles and decreases the quadriceps strength which then alters gait.⁵⁷ An imbalance in the strength of the muscles within the quadriceps group can cause abnormal patellar tracking. For example, a decrease in strength of the vastus medialis obliquus (VMO), or an increase in the strength of the vastus lateralis, causes the patella to deviate from its usual tracking.⁵⁷

In order to have good PC, strong muscles are needed. Weakened muscles can predispose a person to injury. For example, weak muscles in the lower extremity are unable to absorb as much load during activity, causing degeneration of the joint.⁵⁰ In addition, in older patients, falling is associated with poor balance caused by weak muscles.²⁴ These weak muscles are incapable of producing the movement the body needs, which alters and creates new learned patterns and a change in the COM. With any new learned motor pattern, PC has to be adjusted to keep balance and maintain stability.

Muscle Flexibility

Muscle flexibility allows a body to move in its full range of motion (ROM), helping with functional movement; the body is able to move as it needs. If muscles are inflexible, the ROM of joints decrease and the length of the muscles shorten. As the muscle loses length, the muscle cannot generate as much torque, and indirectly weakens the muscle. Inflexibility decreases the magnitude of movement.

As muscle flexibility changes, PC changes. In gait, inflexibility may shorten the cadence which modifies the PC since the body's COM has to change to remain stable. This creates a new gait pattern and PC changes to compensate for the new movement. As stated before, inflexibility indirectly weakens the muscle. Then, not only is PC is affected by inflexibility, but also by weak muscles.

Pain and Effects on Postural Control

The definition of pain is, "an unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage".¹⁴ Pain alters the body's motor variables like movement, muscle strength, muscle activation, and also sensory variables which include proprioception, and balance. Pain may also affect ROM and muscle flexibility. Anterior knee pain (AKP) influences the motor and sensory variables about the knee. OA and AKP have similar changes in motor and sensory variables. Because of this, knee OA studies are a good assessment of pain on joint injury and can assist in theorizing the long term effects of AKP.

Two theories of pain have been presented in research. Both theories agree when pain is present, body movement is altered to reduce pain. One theory presented by Lund et al. says pain may be a protective mechanism.⁵⁸ When pain is present, the body alters its movement and

repositions the loads on the body to decrease pain and prevent further damage.⁴⁰ He stated this theory according to chronic muscle pain. In the second theory, Travell et al. states, “dysfunction caus[es] pain which then reinforces dysfunction”.⁵⁹ For example, this would be the result of improper mechanics. The dysfunction modifies body movement to decrease pain, but because of altered loads, causes joint degeneration to different areas, which then furthers pain and injury. The patient is caught in a “vicious cycle”.⁵⁹ After performing research, Lund states Travell’s theory is incorrect.⁵⁸

Anterior Knee Pain

Several adolescents and young athletes suffer from anterior knee pain (AKP). It is unclear where AKP originates but it is thought that the pain comes from the structures within the knee. AKP can be caused by patella femoral pain syndrome, OA, joint effusion, compression of the fat pad³⁰, and poor patellar tracking from muscular imbalance.⁵⁷ AKP affects the proprioception of the lower extremity and the muscles surrounding the knee, specifically the quadriceps strength and activation.

AKP may derive from the structures in the knee like the infra-patellar fat pad.³⁰ Nociceptors have been found to be spread throughout the knee capsule.⁴¹ Substance-P is a protein that triggers nociceptors, and subjects with AKP have been found to have an increased amount of substance-P.^{30,43} The infra-patellar fat pad is very sensitive to pain. The pad may be very sensitive⁶⁰ because it is highly vascularized and is supplied by the posterior tibial nerve.³⁰ Because it is sensitive, and an increased amount of substance-P is present and stimulating nociceptors, an increased amount of afferent signals are being sent. This interferes with the afferent signals of the mechanoreceptors, which alters the timing and activation of the muscles

around the knee, which affects proprioception. With delayed timing, PC must be adjusted to try and maintain balance and stability of the body.

Proprioception and Muscle Activation

Deficits in proprioception have been found in knee disorders like OA^{61,62}, patellofemoral pain syndrome⁶³, and anterior cruciate instability.^{64,65} Pain may cause deficits in proprioception because of interfering afferent signals at central processing. The nervous system is responsible for producing muscle force and the amount of muscle force or muscle contraction produced, which is a direct effect of proprioception. If proprioception is altered by pain and the nervous system makes changes to the muscle force or contraction and muscle activation, the COM or base of support will adjust to stay stabilized, thus affecting PC.¹⁴ Adjusting the COM may predispose a person to injury because of the altered loads and new mechanics.

If pain is present, mechanoreceptors (sensory receptors for joint position used for proprioception) are sending signals the same time nociceptors (pain receptors) are sending signals to central processing.¹ Multiple signals take longer to analyze and therefore increase the timing of the returning efferent message. When mechanoreceptor and nociceptor afferent signals interfere with each other, the muscle spindles function differently and affect joint position.⁶⁶

Poor proprioception may cause chronic problems. If sensory inputs from mechanoreceptors are being blocked or delayed by other signals like pain receptors, the neuromuscular or efferent messages to the muscles will also be delayed. Delayed messaging may decrease muscle strength or cause abnormal use of muscles during activities of daily living (ADL) like gait. Delayed messaging causes certain motoneurons to be inhibited while others are excited, which decreases the muscle strength of the inhibited muscles.⁵⁸

Studies show pain does inhibit and excite certain motoneurons associated with proprioception. In one study, bradykinin, a chemical released by the body that causes pain, was injected into the gastrocnemius. The results show the chemical inhibited the flexor motoneurons and excited the extensor motoneurons.⁶⁷ According to this study, if applied to the knee joint, the presence of pain would cause the knee flexors (hamstrings) to be excited or more active, and the knee extensors (quadriceps) to be inhibited. In addition to inhibition of the quadriceps muscles, ROM of the knee and the velocity of gait are also decreased. In support of Lund's theory, when the muscle nociceptors are excited and cause the knee extensors (quadriceps) to be inhibited and the flexors (hamstrings) to be excited⁵⁸, it's thought that this change prevents further injury. Lund also concludes in his study that this difference in excitation reduces the ROM and the velocity to prevent pain and injury [19].

Another study used electromyography (EMG) to observe and compare the muscle activation during contraction of the rectus femoris (RF), VL, and VMO of AKP subjects and healthy subjects during a knee flexion-extension isokinetic exercise. No significant difference was found in the EMG between the AKP subjects and healthy subjects during contraction of the RF or VL. However, the EMG for AKP subjects showed the VMO during concentric contraction (muscle shortening) peaked at 45% of extension during isokinetic exercise. This was a delayed muscle activation pattern compared to the EMG of healthy subjects. From these results, the authors hypothesized that AKP affects the neural control and strength of the quadriceps.⁵⁷ If pain is causing a delayed reaction of the quadriceps, stability and orientation will change, therefore affecting the overall PC of the subject.

A decrease in quadriceps contraction time due to knee pain is also found in another study by Radin et al.⁵⁰ Proprioception of subjects with knee pain is shown to be affected during testing

of gait.⁵⁰ Subjects with knee pain had a 37% increase in peak loading at heel strike than the control group. In addition, results showed subjects with knee pain had decreased quadriceps contraction time. The decrease in quadriceps contraction time may be the cause of the subjects' decreased control in the fall of their leg, which in turn results in decreased time to heel strike. This may have produced the increased peak load at heel strike found in the study.⁵⁰ This contradicts Lund's theory that pain is a protective mechanism and prevents further injury. With an increased peak load at heel strike, the joints of the lower extremity have to absorb more force, which could cause injury through joint degeneration.

Decreases in proprioception can lead to future OA.⁵⁰ Just like osteoarthritis (OA) affects quadriceps strength and activation²⁴, AKP may also affect quadriceps strength and activation. There is a need for proprioceptive acuity and muscle contraction for a smooth gait.⁶⁸ Weakened quadriceps strength can be an effect of poor proprioception, which then affects balance.⁶⁸ As pain modifies proprioception of the muscles surrounding the knee, and changes in gait pattern occur, the COM must be changed to maintain balance and PC.

Balance

Balance is needed for ADLs like gait and climbing stairs. When pain becomes involved, it can affect the balance during these activities. In the elderly, poor balance and decreased postural stability increases the number of falls.⁶⁹ Chronic pain like OA causes balance impairments and inhibits muscles surrounding the knee.²⁴ Although pain causes balance impairments, the balance impairments may be directly related to the decreased muscle strength and poor proprioception.²⁴

The quadriceps muscle is a major muscle that helps to stabilize the knee joint and balance the body.⁷ If pain changes the timing of muscle activation and decreases muscle strength around

the knee (specifically the quadriceps), the balance of the COM over the base of support will change.^{7,24} In a study performed by Hinman et al.²⁴, it was found that osteoarthritic knee patients have decreased balance. The results showed OA patients took fewer steps during the step-test (a test of standing balance) as compared to its controls. Mohammadi⁷ performed a similar study using only females and found the same results. By performing fewer steps during the step-test, it is assumed OA patients are off balance while standing. In another study, after inducing knee pain in healthy older individuals, balance was unaffected during standing balance.¹ This result may have been reached because of the use of acute pain. A confounding variable of these studies is age. It is difficult to understand if pain or aging is causing the balance impairment. Balance impairments will affect PC. As balance changes, the body will have to continually adjust its COM or base of support to remain stable.

Muscle Strength

People that suffer from patella femoral pain syndrome, AKP, joint effusion, or OA, lack quadriceps strength.^{57, 70,71} The decrease in muscle strength may be the cause of poor proprioception, which is a result of pain. However, AKP can be caused from joint effusion and if present, can also cause muscle inhibition which then causes a decrease in quadriceps strength.⁷⁰ If joint effusion is present, it is unknown if the pain or the effusion is the cause of decreased quadriceps strength. Either one inhibits the knee flexors while the extensors are excited.

The total amount of inhibition of the quadriceps muscle can be measured by finding the difference in maximal voluntary contraction (MVC).⁷⁰ In a study when MVC was tested, OA subjects compared to controls had a decreased MVC while the hamstrings were unaffected.⁷⁰ This is understandable if strength of the quadriceps is weakened.¹¹

Strength rehabilitation programs can be incorporated to relieve the pain that is associated with AKP. A study showed that strengthening of the quadriceps (specifically the VMO) in athletes suffering from AKP can help alleviate pain, and in just 2 weeks of strengthening, the athletes were able to return to play with no pain.⁷²

Muscle strength, specifically the quadriceps muscle, seems to decrease when proprioception is affected by AKP, which alters muscle activation, and then alters muscle strength. With a decrease in muscle strength, the body will have to alter its movement to stay stabilized and have PC. For example, without adequate quadriceps strength, the leg may not fully extend, which may shorten stride length. With a shortened stride length, the body has to reposition its COM to stay balanced during gait.

Muscle Flexibility

Not only does pain inhibit the quadriceps and decrease strength, but it also decreases range of motion (ROM) at the knee. With a weakened quadriceps, the knee tends to stay in slight flexion rather than fully extending⁷⁰, perhaps because of the reduced strength or a patient's motive to prevent pain. As stated by Young et al.⁷⁰, inhibition will decrease MVC and decrease muscle size, which will keep the knee in flexion and result in contracture.

Decreased muscle flexibility may be caused by reduced ROM at the joint due to pain. Subjects with knee pain suffer from weakened quadriceps and reduced range of motion (ROM) of knee extension.⁷¹ With the inability to fully extend the knee, the quadriceps and gastrocnemius may shorten since the full length of the muscle is not being used. A decreased flexibility may occur to increase the stability at the knee joint.⁷³

A decreased ROM at the knee has been found in active students with patella femoral pain syndrome. The students experiencing pain had reduced flexibility in their quadriceps and

gastrocnemius compared to those students with no pain.² A stretching program may improve inflexibility, which may improve some of the causes of knee pain, but has not yet been proven.²

Dynamic Movement

It is hypothesized that the load on the knee joint of OA patients during gait shifts to the medial compartment to reduce pain and protect the joint. Studies conclude gait velocity, stride length, cadence, and ROM of the knee joint are all decreased to diminish pain.⁷¹

A belief is as the load of the knee joint is modified, the load must be compensated for at another joint.^{71 50} Messier et al.⁷¹ suggests compensation occurs at the hip. Manetta et al.⁷⁴ proves this theory incorrect and found no compensation at the hip. Manetta et al.⁷⁴ suggests instead of altering movement at the hip, the decreased gait velocity and decreased knee joint ROM are the compensation that decreases the load at the knee. At heel strike, decreased knee flexion was seen and because of this, a decreased load at the knee was measured. If gait velocity and knee flexion in OA patients remained the same with knee pain, greater shock absorption would be needed because of the increased force at heel strike.⁵¹

Looking at OA, pain causes a decreased gait velocity, stride length, and cadence. As these decrease, PC of the patient has to change in order to stay stabilized. Young adolescents experiencing AKP have been found to have problems with abnormal gait as well.²¹ This could be due to poor proprioception, and may lead to a future diagnosis of OA.²¹ Looking at the future of AKP patients, it could be similar to that of OA patients if rehabilitation is not undergone. Altered knee joint loading by weak quadriceps and poor muscle activation can cause AKP patients to suffer from joint degeneration and also increase their chance of injury. It will only worsen as time passes and alterations to gait, running, and activity of daily living (ADL), are made.

Affects of Pain Reduction on Postural Control

Many studies have been performed on different joints like the knee and ankle to see how pain reduction affects PC and its independent variables like proprioception and muscle strength. Results have varied among studies and have found significant or insignificant results in increased knee flexion, increased muscle strength, and increased compression loads on the joints.⁴⁰

Some athletes use anesthetic at the ankle joint for pain relief. The study by Down et al.⁷⁵ conducted a study to evaluate anesthetic injection on proprioception. No significant differences in small ankle movements between saline and the anesthetic injection were found, meaning, when pain was relieved, there was no change in proprioception. This could mean two things: i) pain may not cause poor proprioception or ii) poor proprioception caused from chronic pain is not corrected instantly with pain relief. A patient may have to retrain to gain proprioception back. However, if pain is a protective mechanism, removing pain may cause further damage to the joint. From this study, we can conclude knee pain may also be unaffected by pain reduction.

In a study by Hassan et al.²¹, knee pain was reduced in OA patients for a short period of time using bupivacaine or a placebo. The authors found an increase in quadriceps MVC with the placebo and drug, which means quadriceps strength or force increased. The reasoning behind this result is that muscle force is easier to produce with no pain, and/or reducing pain reduces spasms caused by pain. Also in this study, proprioception worsened and it was concluded pain was not a primary variable that affects proprioception. Postural sway was not improved in either placebo or drug. OA is a chronic disorder that changes gait patterns over time. Reduced pain for a short period of time cannot improve postural sway. Longer pain relief, as suggested, may be needed to show improved results and in addition, strengthening may be needed along with retraining.

It was suggested pain is a protective mechanism, and that if removed it could cause further damage. Henriksen et al.⁴⁰ used 10 subjects with knee OA to study analgesia and its effects on compression, load, and knee angles. Subjects had 10 ml of lidocaine injected into the knee joint to reduce pain. After pain reduction, testing consisted of walking at 1.1 m/s. Test results found subjects extended their leg 4 degrees farther at heel strike and early stance. In addition, compressive flexor muscle forces, total compressive forces, and medial knee compartment compression increased. Increased loads may cause further degeneration in the OA knee.⁴⁰ In addition, since medial loads increased, the study concluded pain is a protective mechanism since it caused the OA patient to alter its movement away from medial compartment loading.

Similar results were found when using a different method of reducing pain in OA patients. Piroxicam, an NSAID, was given to OA patients at least 2 weeks before testing. Their gait was evaluated before and after pain reduction.⁷³ The result was an increase loading to the knee joint and an increase in knee flexion angle. Increased loading to the joint causes further damage and deterioration to the knee.⁴⁰

Methods

Measuring Postural Control

Studies typically use similar methods to evaluate static and dynamic PC. Studies measure static and dynamic PC using COP measurements^{11,21,76}, commonly using a force plate. Other instruments used in these studies are an EMG to measure activation of specific muscles, and reflective markers or diodes to observe the velocity of movement and ROM. A newer method of measuring COP is plantar pressure insoles, which map the distribution of pressure on the foot during continuous time periods of activity. The insoles follow the COP through the entire stance

phase. Trials of insole data can be compared to see if COP has deviated. The force plate and pressure insoles can be used to compare the effects pain has on COP and thus PC.

Force Plate

The force plate has been a popular method in evaluating static and dynamic PC. Because of its validity, it is the gold standard.⁷⁷ A force plate is useful in finding the COP, COP excursion, and ground reaction forces during quiet stance and walking.¹⁴ A limitation of a force plate is it can only record one step with one plate or a few steps at a time with multiple force plates.⁷⁷

A force plate is an effective method of studying the influences AKP has on PC by observing COP velocity and time to stabilization during quiet stance and jump landing. This method is used often when measuring quiet stance and is adequate since it does not require the subject to move. This method, however, is inadequate when recording measurements for multiple steps during natural gait unless a series of force plates are available.

F-scan

An instrument that has not been commonly used to evaluate PC is pressure insoles. Pressure insoles have been used to look at plantar pressure distribution in studies evaluating: the gait of diabetic patients with foot ulcers⁷⁸, athletes running on different terrain, the use of different orthotics⁷⁹, etc. However, they have not been used to evaluate the effects AKP has on PC by examining COP measurements. Rose et al.⁸⁰ said the F-scan system, “could be useful for making comparisons and evaluating changes in a well-controlled clinical study.”

These insoles evaluate the pressures on the bottom of the foot and how the load is distributed upon the plantar foot through the stance phase. The F-scan, Tekscan system insole has 960 transducers spread evenly every 5.08mm with 21 rows and 60 columns.⁸¹

This method of measurement could be used instead of a force plate since the insoles can evaluate the COP and excursion during natural gait.⁸¹ In addition, it is a method that can examine the effects pain has on PC by observing differences in COP excursion during various activities like gait, running, landing, and ascending and descending stairs.

For the F-scan system, a level of acceptable variation for foot pressure measurements has not been established.⁷⁸ Many studies have investigated the reliability of this system. It has been noted to use the system carefully but found to be a reliable.⁷⁷

Inducing Anterior Knee Pain

To induce AKP, injecting hypertonic saline solution has been a common and accepted method.^{1,82-84} It has been shown that an injected infra-patellar fat pad creates AKP.³⁰ The hypertonic saline solution gives continuous pain and slowly wears off.³⁰

AKP and OA are chronic disorders. A disadvantage of injecting hypertonic saline solution into the infrapatellar fat pad is that it causes acute pain. Chronic AKP incorporates a longer period of time to slowly affect muscle strength and proprioception. Affected muscle strength and activation and proprioception over time may then show deficits in PC.

COP Velocity

Static PC can be measured and analyzed by collecting force plate data during quiet stance and then examining the COP²⁵. Many methods have been used to analyze the COP data during force plate quiet stance. A reliable method of analyzing quiet stance force plate COP data is to calculate the COP velocity.^{25,85,86} COP velocity is the distance the COP traveled over the total time. COP velocity can be calculated in the anterior-posterior (AP) and medial-lateral (ML) directions. The absolute value of the ML COP distance will have to be taken before averaging the velocities. An increase in the COP velocity represents a decrease in stability or PC.¹⁹

Observing COP velocity is a reliable method in analyzing static stance force plate data after inducing pain.

Time to Stabilization

Time to Stabilization (TTS) is an objective measure of dynamic PC.^{26,39,87} It is a preferred test since it uses a sport specific activity of jump landing.³⁹ TTS has been used to measure the effects of fatigue on neuromuscular control and stability or PC.²⁷ In addition, TTS has been used to measure ankle instability.²⁹

In TTS, a baseline static stance ground reaction force (GRF) is recorded to have a measurement of when the subject is in a stabilized state. This is needed in order to evaluate when the jump landing GRF data reaches a stabilized state. TTS is the time it takes the jump landing GRF to come within a range of the baseline GRF of static stance.²⁶ When using TTS, the anterior-posterior TTS (APTTS), medial-lateral TTS (MLTTS), and vertical GRF (VGRF) are analyzed. The APTTS, MLTTS, have to stay within 0.25 standard deviations of the GRF of the baseline static stance to be stable.²⁷ The VGRF also has to stay within 5% of the subject's body weight to be considered stable. An increase in time to stabilization represents the body's altered response to reach stability of PC.²⁷

Although TTS has been used most often to show the effects of fatigue on stability, TTS would be a good method to show the effects pain has on stability or PC. In addition, jump landing is a sport specific activity which can give insight on how pain affects sport specific activity. When studying static PC, studies have failed to show a difference in stability or PC when measuring static stance because of the easiness of the task. The dynamic jump landing task is not easy and may show more of the effects pain has on stability or PC.

Visual Analogue Scale

Pain is difficult to measure since it is subjective. Pain must be assessed in order to compare any difference made after an intervention in a study. A popular method used in studies to assess pain quantitatively is the Visual Analogue Scale (VAS) where subjects rate their pain. The subject rates their pain on a line from 0 or no pain experienced to worst pain ever experienced.³² This method has been shown to be reliable for rating chronic^{88,89} and acute pain.

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Conclusion

As shown, pain impairs body movement, muscle strength^{7,11,12}, overall performance^{7,13,14}, and static and dynamic postural control (PC).^{11,14} Pain also has been shown to alter sensory inputs, which then affects the motor components and changes overall PC.^{1,21} Because pain causes deficits, the body is forced to make changes to stay balanced and maintain PC. For example, deficits in proprioception may alter various motor outputs like recruitment, patterning, and coordination. As these modifications are made due to pain, the COM or base of support during stance and dynamic movement will adapt to maintain stability, thus affecting PC.

It is critical to study and isolate the effects AKP has on PC to better understand the future effects AKP will have on the body if the pain is not alleviated. The purpose of this study is to quantify PC alterations in subjects experiencing experimentally-induced AKP during stance and dynamic movement. An increase in COP AP and ML velocities, an increase in TTS, and excursion of the COP will show pain has an effect on PC. Using COP velocity to evaluate stance has been shown to be reliable, TTS is an effective method in studying dynamic movement and PC, and pressure insoles for walking is the best method since excursion can be evaluated for a continuous amount of time.

When inducing pain, if COP velocity increases, TTS increases, and a significant amount of excursion occurs during walking, we can conclude pain has an effect on PC during quiet stance, landing, and walking. Knowing the effects pain has on PC in long term OA patients, we can hypothesize chronic AKP may be subject to similar outcomes in PC and may be at risk of further injury if pain is not alleviated.

Chapter 3

Methods

Research Design

This study will be a counterbalanced cross-over study. It is a 3x3 analysis that will detect differences between groups (pain, sham, control) over time (pre-injection, injection, post-injection). The independent variables are group and time. The dependent variables are COP anterior-posterior (AP) and medial-lateral (ML) velocities, AP time to stabilization (TTS), MLTTS, vertical TTS (VTTS), COP excursion, and quantification of pain. The dependent variables will be measured at pre-injection, injection, and post-injection to see if there are differences in COP AP and ML velocities, APTTS, MLTTS, VTSS, and COP excursion as a result of pain.

Subjects

Fifteen subjects will complete this study. All subjects will be recruited from Brigham Young University (BYU), ages 18-26. To participate in this study, subjects must be healthy, physically active (exercising a minimum of 3 times a week for 30 minutes), have no current lower extremity pathology, have no current muscle or joint pain, and have no history of surgery with the involved limb. A questionnaire will be completed by each subject to collect all of these data (Appendix A). This study will be approved by Brigham Young University's Institutional Review Board before recruiting. Each subject will read and sign a consent form before participating in this study. Subject confidentiality will be maintained and names will not be used in publication.

Instruments

1. F-scan insoles (Tekscan Inc., Boston, MA): pressure sensors with 3.9 sensels per cm² that measure plantar pressures and compute COP. It has a sampling rate of 256 Hz. Pressure sensors are custom fit to the shoes. F-scan Research version 6.31(Tekscan, Inc., Boston, MA) will be used to record the plantar pressures measured during walking for all subjects. The COP excursion will be evaluated on this software (100Hz). The F-scan insoles have been proven reliable. ^{78,81}
2. A force plate (AMTI Force and Motion, Watertown, MA; 200Hz) will measure COP AP and ML velocities from a single leg stance, and COP time to stabilization from the subject's landing off a 31.1cm stool.
3. Isotonic saline solution (0.9% sodium chloride, Hopsira, Inc., Lake Forest, IL) is a physiologic neutral solution used to ensure the irritation of the fat pad is due to the hypertonic saline solution and not the mechanical effects of the injection. ¹
4. Hypertonic saline solution (5% sodium chloride, Baxter Healthcare Corporation, Deerfield, IL) is the solution that will be used to cause chemical irritation to the infrapatellar fat pad. ¹
5. Treadmill (Quinton Instrument Co., Bothell, WA) will be used to set the velocity during testing of walking.
6. Visual Analog Scale (VAS) is a common method used to assess pain of subjects and will be used throughout this study. This method of assessing pain has been proved reliable and valid. ³² (Appendix B)

Procedures

Subjects will report to the lab wearing shorts. Subjects will then be weighed to calibrate the F-scan insoles and their leg length measured to standardize walking velocity. Each subject will participate in 3 conditions in a counterbalanced order (Appendix C): (i) injection of hypertonic saline solution (5%), (ii) injection of isotonic saline solution (0.9%), and (iii) no injection. There will be 3 test days with a washout period of a minimum of 2 days and a maximum of 4 days in between each test day.

Within each condition, force plate data for single leg quiet stance and landing, as well as the plantar pressures during walking will all be recorded at pre-injection, injection, and post-injection (the amount of time needed for pain to subside). The velocity of the subject's walk will be standardized according to leg length (ASIS to medial malleolus) (Appendix D).³³

All subjects will use the Nike T-Lite shoe for testing. The insoles of the Nike T-Lite shoe will be removed and replaced by the F-scan pressure insole. Subjects will warm up walking on the treadmill for 5 minutes at the calculated standardized walking velocity from their leg length (Appendix D). After warm-up, the F-scan pressure insoles will then be calibrated according to the manufacturer's directions as the subject is wearing the shoes (Appendix E). Next, the force plate will be zeroed (Appendix F).

Hypertonic and Isotonic Saline Solution Conditions

After the warm-up, subjects will lay supine on a treatment table. The lateral side of the knee, inferior to the patella will be sterilized using a Povidone-Iodine Swabstick (10% solution, Professional Disposables, Inc., Orangeburg, NY). After swabbing the area and allowing it to dry, a one-time 0.75 ml solution will be injected laterally into the subject's infrapatellar fat pad of the dominant leg using a 25 gauge needle at a 20° angle in a superolateral direction.³⁰ The needle is

inserted at a depth of 10 mm.^{1,30} To spread the solution throughout the infrapatellar fat pad, the needle will be moved around at several angles inside the fat pad while the solution is injected. The isotonic saline solution (0.9%) is a neutral solution that does not cause pain. The isotonic saline solution is used to make sure the injected needle does not cause the pain. The hypertonic saline solution (5%) will cause chemical irritation and produce pain. After injection, the subject will remain laying supine for 30 seconds, then sitting up for 30 seconds, and standing for 30 seconds, all to avoid nausea.

No Injection Condition

After the warm-up, subjects will lay supine on the treatment table. No saline solution will be injected into the infrapatellar fat pad. The subject will lay supine for 30 seconds, then sit up for 30 seconds, and stand for 30 seconds, which is the time taken to avoid nausea after injecting a solution.

Data Collection

Single Leg Quiet Stance Test

After the condition has been established, force plate data for a single leg quiet stance of the dominant leg will be recorded for 3 trials, each lasting 30 seconds.²⁵ Fifteen seconds of rest will be given in-between each trial. During stance, subject will have their hands on their hips, their eyes open looking straight ahead, and their non-dominant leg raised off the ground. If the subject touches the ground during the trial with their non-dominant leg, the trial will be tallied and rerecorded. Subjects will practice the single leg quiet stance once prior to their 5-minute warm-up.

Landing Test

After stance, force plate data of the dominant leg after landing off a 31.1 cm high platform will be recorded for 3 trials.²⁷ The subject will have their hands on their hips with their eyes open, looking straight ahead. The subject cannot lower their dominant leg to the force plate or jump off the platform. The subject will practice landing on the force plate prior to their 5-minute warm-up and pre-injection testing until they feel comfortable with the task.²⁸ The subject will be directed to stabilize as quickly as possible after landing and remain standing for 6 seconds.²⁷ Fifteen seconds of rest will be given in-between each trial. If the non-dominant leg touches the force plate during the trial, the trial will be tallied and rerecorded.

Walking Test

After landing, COP excursion calculated from plantar pressures will be recorded while walking on the treadmill with no incline. The walking velocity is standardized according to leg length using Hof's equation³³ (Appendix D):

$$\hat{v} = \frac{v}{\sqrt{gl_0}}$$

Plantar pressures will be recorded for 15 seconds.

After walking plantar pressures are recorded, the subject will sit for 20 minutes to make sure pain has subsided. After 20 minutes of rest, force plate data for single leg quiet stance and landing, as well as the plantar pressures during walking will again be recorded for comparison to baseline measurements.

Data Reduction

The force plate data recorded during single leg quiet stance will be analyzed using the COP velocity in the AP and ML directions. The COP velocity is the COP distance traveled over

the total time. The COP velocity will be derived from the COP coordinates for each instant in time. The COP averages will be calculated by averaging all calculated velocities. An increase in COP velocity suggests a decrease in PC.¹⁹ The force plate data recorded during landing will be analyzed using TTS in the anterior-posterior (APTTS) and medial-lateral directions (MLTTS).^{28,29} An increase in the time to stabilization suggests a decrease in stability or PC. The subject is considered stable when the APTTS and the MLTTS are close to zero or within 0.25 standard deviations of the overall mean and the vertical GRF is within 5% of the subject's body weight.^{26,27}

For walking trials, heel strike and toe off will be identified and COP trajectory excursion of the stance phase evaluated. The COP coordinates will be exported then compared over the stance phase which will be time normalized to 100 samples. The heel strike will be visually identified from the exported data as the first COP point seen and toe off as the last COP point. After identifying the middle 3 stance phases from the 15 seconds of recorded walking data, the phases will be time normalized to 100 samples and then averaged. An increase in COP excursion suggests a decrease in PC or balance.¹⁹ Recorded measurements of all 3 groups and times will be compared to see if pain has an effect on PC.

Pain Measurements

The pain of each subject will be assessed using a 10 cm visual analogue scale (VAS) that uses descriptors of 'no pain' and 'pain as bad as it could possibly be' (Appendix B). Pain will be assessed before and after each established condition; after lying supine, sitting up, and standing for 90 seconds; and again before each test (stance, landing, walking) and after walking. In addition, pain will be assessed every 5 minutes for 20 minutes after the last test.

Statistical Analysis

Raw data will be used from the force plate and pressure insoles recorded trials. A 3x10 repeated measures analysis of variance (ANOVA) and Tukey Post Hoc ($p < 0.05$) will be used to determine significant differences in pain. A repeated measures ANOVA will be used to determine significant differences in COP AP and ML velocities during single leg quiet stance and APTTS, MLTTS, and VTTS during landing. A functional ANOVA will be used to determine differences between groups over time with respect to COP excursion during walking ($\alpha = 0.05$). This analysis will allow us to compare variables as polynomial functions rather than discrete values over the entire stance phase of each movement.

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Appendix A

Questionnaire

1. Do you exercise a minimum of 90 minutes a week or 3 times a week for 30 minutes?

Yes No

2. Are you currently suffering from any lower extremity injury (muscle strains, ligament sprains etc.)?

Yes No

If Yes, what injury are you suffering from?

3. Are you currently experiencing any lower extremity pain? Have you experienced lower extremity pain in the last week?

Yes No

If Yes, where are you experiencing pain, and for how long?

4. Have you had surgery on your dominant lower extremity limb?

Yes No

Appendix B
Visual Analogue Scale

Pain will be assessed before and after each established condition; after lying supine, sitting up, and standing for 90 seconds; and again before and after each test (stance, walking, jogging).

No pain

**Pain as bad as it
could possibly
be**

Appendix C

Counterbalanced Order of Conditions for Subjects

1 = Control

2 = Hypertonic Saline Solution

3 = Isotonic Saline Solution

Subject #

1. 1-2-3

2. 2-3-1

3. 3-1-2

4. 1-3-2

5. 2-1-3

6. 3-2-1

7. 1-2-3

8. 2-3-1

9. 3-1-2

10. 1-3-2

11. 2-1-3

12. 3-2-1

13. 1-2-3

14. 2-3-1

15. 3-1-2

Appendix D

Calculated Walking Velocity (mph) According to Leg Length (m)

LEG LENGTH (m)	WALK SPEED (mph)
0.63	2.8
0.64	2.8
0.65	2.8
0.66	2.8
0.67	2.9
0.68	2.9
0.69	2.9
0.7	2.9
0.71	2.9
0.72	3.0
0.73	3.0
0.74	3.0
0.75	3.0
0.76	3.0
0.77	3.1
0.78	3.1
0.79	3.1
0.8	3.1
0.81	3.1
0.82	3.2
0.83	3.2
0.84	3.2
0.85	3.2

0.86	3.2
0.87	3.3
0.88	3.3
0.89	3.3
0.9	3.3
0.91	3.3
0.92	3.4
0.93	3.4
0.94	3.4
0.95	3.4
0.96	3.4
0.97	3.4
0.98	3.5
0.99	3.5
1	3.5
1.01	3.5
1.02	3.5
1.03	3.5
1.04	3.6
1.05	3.6
1.06	3.6
1.07	3.6
1.08	3.6
1.09	3.6
1.1	3.7

Appendix E

F-scan Insole Calibration

To normalize plantar pressure data, the sensors have to be calibrated using the subjects' body weight.

Taking an F-Scan (In-Shoe) Recording

Step 1 Prepare Patient

STEPS

Details

Seat Patient	
Remove Footwear	
Place ankle bands on ankles	Wrap ankle bands snugly around legs just above ankles
Trim Sensors	Locate patients shoe size on sensor Cut sensor on trim guidelines Trim off any partially cut connecting dots on both sides of sensors
Place sensors in footwear so that tab exits shoe on lateral side of leg	Insert sensor into shoe to check fit. The sensor should lie flat within the shoe so that there is no curling up on the sides.
Replace footwear	Instruct the patient to put on there shoes taking care that the sensors remains flat and in position.
Connect sensors to cuff units	Listen for "click" Look for 2 Green Lights on Cuffs
Stick cuff units to Ankle Bands	Stick cuff units to ankle bands leaving slack for ankle flexion
Stand patient	
Place belt around waist	Postion belt so that velcro flap is on small of back

Secure cables to belt	Make loop in cables and slide velcro flap through loop leaving enough length between belt and cuff units for leg extension
-----------------------	--




Step 2: Launch Software**Enter Patient Info**

Start Software	Double click on F-Scan Icon on Desktop						
Enter Patient Data							
	<table border="0"> <tr> <td style="vertical-align: top;">If....</td> <td style="vertical-align: top;">Then....</td> </tr> <tr> <td>New patient</td> <td> Click New Patient Enter patient info Click New Movie Sensor Selection: Options-> Select Sensor F-Scan - Check off Handles A and B Click OK </td> </tr> <tr> <td>Old patient</td> <td> Click Open Patient Click on Patients name to highlight Click Open Patient Click New Movie Select sensor Check off handles A and B Click OK </td> </tr> </table>	If....	Then....	New patient	Click New Patient Enter patient info Click New Movie Sensor Selection: Options-> Select Sensor F-Scan - Check off Handles A and B Click OK	Old patient	Click Open Patient Click on Patients name to highlight Click Open Patient Click New Movie Select sensor Check off handles A and B Click OK
If....	Then....						
New patient	Click New Patient Enter patient info Click New Movie Sensor Selection: Options-> Select Sensor F-Scan - Check off Handles A and B Click OK						
Old patient	Click Open Patient Click on Patients name to highlight Click Open Patient Click New Movie Select sensor Check off handles A and B Click OK						


Observe Realtime Window	<p>You should see two feet Left & Right Have the Patient rock back and forth. Make sure you can see the Landmarks of the feet. Look for any crinkles they will appear as bright red spots. If everything looks good Calibrate. If the images have too many crinkles consider redoing or retrimming. If everything looks good Calibrate.</p>
-------------------------	--

Step 3: Calibrations


If the Subject is....	Then....
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Select Calibration Method	Walking	Select Walk Calibration  Enter in Subjects weight Hit Enter Procede to Take a Recording
	Standing / Balance or Running / Jumping	Select Step Calibration  Enter in Subject Weight Hit Start and Follow Prompts Procede to Take a Recording
	Other	Select Advanced Calibration 

Step 4: Take a Recording


Create a Clear Walking Path	Mark starting and stopping point	
Check acquisition parameters	Option -> Acquisition Parameters	
	Enter / Check Acq. Parameters:	> Duration: Length of recording > Frequency: Sample rate; frames /sec. > Period: Sec/frame > Frames to record or Click default (8 sec. 50 hz)
	Triggering	Does not need to be selected for F-Scan Click OK
Instruct subject to begin walking/running	Click record 	Hit stop when the Patient is done Walking or it will automatically stop once time (in duration) is reached.

Step 5: Save Recording

Save Movie	OR File -> Save movie
Click FD Icon 	

Confirm Patient Info	OR Enter New Patient if patient is New
Enter Comments	
Enter Diagnosis / Procedure	Type in or use drop down window to select procedure
Click Yes	To save Patient to Database

Step 6: Analysis

What do we want to Analyze.....?	If	Then
	Highest Area of Pressure	Click Show Panes Icon  Create new graph - OK
Refer to 4P Method Application Sheet	Timing COF / COF Trajectory left v. right Symentry Integral / Impulse	SECTION 3: ANALYZE TIMING SECTION 4: ANALYZE TRAJECTORY SECTION 5: ANALYZE SYMMETRY SECTION 6: ANALYZE INTEGRAL / IMPULSE

Appendix F

Vicon Force Plate Calibration

- Turn on Vicon switch
- Open up Vicon Nexus 1.5.1 program
- Under System, select falk_thesis*
 - Go live
- Under Local Vicon System on left hand side
 - Select Force Plates
 - Right click on #2 BYU FP2 East AMTI 3421
 - Select zero level