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# Arthrogenic muscle response induced by an experimental knee joint effusion is mediated by pre- and post-synaptic spinal mechanisms

Riann M. Palmieri [a,*,1], James A. Tom [b], Jeffery E. Edwards [c], Arthur Weltman [d], Ethan N. Saliba [e], Danny J. Mistry [e], Christopher D. Ingersoll [e]

[a] *Neuromuscular Research Laboratory, Division of Kinesiology, University of Michigan, 401 Washtenaw Avenue, Ann Arbor, MI 48109-2214, USA*
[b] *Drexel University Health Sciences, Orthopedic Surgery Control, New College Building, 245 North 15th Street, Philadelphia, PA, USA*
[c] *Human Performance Laboratory, Department of Physical Education, Indiana State University, Terre Haute, IN 47809, USA*
[d] *Exercise Physiology Laboratory, Department of Human Services, University of Virginia, P.O. Box 400407, 210 Emmet Street South, Charlottesville, VA 22904-4407, USA*
[e] *Sports Medicine/Athletic Training Research Laboratory, Department of Human Services, University of Virginia, P.O. Box 400407, 210 Emmet Street South, Charlottesville, VA 22904-4407, USA*

**Abstract**

Knee joint effusion results in quadriceps inhibition and is accompanied by increased excitability in the soleus musculature. The purpose of this study was to determine if soleus arthrogenic muscle response is regulated by pre- or post-synaptic spinal mechanisms. Ten healthy adults (two females and eight males) were measured on two occasions. At the first session, subjects had their knee injected with 60 ml of saline and in the other session they did not. Pre- and post-synaptic spinal mechanisms were measured at baseline, immediately following a needle stick, immediately following a Xylocaine injection, and 25 and 45 min post-saline injection. A mixed effects model for repeated measures was used to analyze each dependent variable. The a priori alpha level was set a $P \leq 0.05$. The percentage of the unconditioned reflex amplitude for recurrent inhibition ($P< 0.0001$) and reflex activation history ($P < 0.0001$) significantly increased from baseline at 25 and 45 min post-effusion. Soleus arthrogenic muscle response seen following knee joint effusion is mediated by both pre- and post-synaptic mechanisms. In conclusion, the arthrogenic muscle response seen in the soleus musculature following joint effusion is regulated by both pre- and post-synaptic control mechanisms. Our data are the first step in understanding the neural networks involved in the patterned muscle response that occurs following joint effusion.


*Keywords:* Renshaw; Knee; Injury; Muscle weakness; H-reflex

## 1. Introduction

Recent evidence suggests that increased motor output to the soleus musculature, referred to as an arthrogenic muscle response (AMR) [35], accompanies quadriceps inhibition in the presence of knee joint pathology [19,21]. An inverse relationship was found between the soleus and quadriceps H-reflex following induction of an experimental joint effusion, resulting in a soleus H-reflex amplitude increase as the quadriceps H-reflex amplitude decreased. This is likely a compensatory response to the quadriceps inhibition promoting the maintenance of upright posture and locomotion. A decreased knee extensor moment accompanied by an increase in the hip extensor impulse and hip extensor work was revealed during walking gait in the presence of knee joint effusion, suggesting compensatory neuromuscular strategies occur to facilitate motion and protect the injured joint [46]. It is of clinical interest to determine the processes involved in the soleus facilitation in order to better understand the neuromuscular consequences that occur following joint injury.

The neuromuscular system is an intricate network operating to regulate motor output and control body

* Corresponding author. Tel.: +1-734-615-3154; fax: +1-734-936-1925.
*E-mail address:* riannp@umich.edu (R.M. Palmieri).
[1] Present address: 3060 D CCRB 401 Washtenaw Avenue, Ann Arbor, MI 48109-2214, USA.

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movement. Interneurons are key components in the spinal circuitry accounting for the majority of all neurons in the spinal cord and function to transmit excitatory and inhibitory signals to other interneurons as well as to alpha and gamma motoneurons [2]. Although an increased alpha motoneuron output is ultimately responsible for the soleus facilitation, the interneurons involved in regulating the excitability of the soleus motoneuron pool remain unknown. AMR has been hypothesized to involve interneurons controlling both pre- and post-synaptic mechanisms [17,27]. Two spinal mechanisms responsible for regulating motor output can be estimated using modified H-reflex protocols: (1) recurrent inhibition mediated by Renshaw cells (a post-synaptic mechanism) [4] and (2) reflex activation history (a pre-synaptic mechanism) [47].

Antidromic potentials in motor axons diminish alpha motoneuron excitability in the same as well as synergistic musculature [39]. This mechanism, known as recurrent inhibition, is due to activation of recurrent collaterals that excite Renshaw cells, which in turn inhibit motoneurons [11,16]. Renshaw cells not only inhibit alpha motoneurons, but also establish connections with gamma motoneurons [1,12–14,33], Ia inhibitory interneurons [15,23–26,29], other Renshaw cells [42–44], and ventral spinocerebellar neurons [15,30]. In addition to the above connections, recurrent collaterals monosynaptically cross-connect with other alpha motoneurons [6] and Renshaw cells also receive input from descending pathways [38,40].

Previous activation of the Ia–motoneuron synapse results in decreased neurotransmitter release and inhibition of the reflex pathway [7]. The depression associated with reflex activation is thought to be involved with other pre-synaptic mechanisms functioning to regulate the gain of motoneuron output [47]. Modulation of reflex depression associated with the frequency of reflex activation may allow for summation of afferent impulses and could potentially contribute to the soleus facilitation seen following knee joint effusion.

The functional importance of both recurrent inhibition and reflex activation is unknown, but both are thought to gate motoneuron output [8,9]. The ability to modulate activity in lower extremity motoneurons following knee joint injury may allow for enhanced soleus motor output, possibly compensating for the quadriceps inhibition seen following knee joint effusion. Pre- and post-synaptic pathways are possibly involved in these neuromuscular responses following joint injury. Therefore, the purpose of this investigation was to examine two spinal regulatory mechanisms responsible for gating motoneuron pool output before and after the induction of an experimental knee joint effusion. Specifically, reflex activation history and recurrent inhibition were examined in the soleus musculature to determine their role, if any, in the neuromuscular excitability changes seen following knee joint effusion.

## 2. Methods

A $2 \times 4$ factorial design was used in this study. The two independent variables were condition (effusion/control) and measurement interval (post-needle stick, post-Xylocaine, 25 and 45 min post-effusion). The dependent variables were percentage of the unconditioned reflex amplitude expressed as a percentage change from baseline for both recurrent inhibition and paired reflex depression (PRD) (a measure of reflex activation history).

### 2.1. Subjects

Ten healthy adult subjects (two females and eight males; age: $24 \pm 4$ years; height: $177.3 \pm 5.1$ cm; mass: $76.2 \pm 13.6$ kg) with no previous lower extremity injury resulting in surgery; no injury to the lower extremity requiring treatment in the previous two years; and a measurable soleus H-reflex and muscle response (M-response) were recruited to volunteer for this study. The volunteers had no history of an adverse reaction to Xylocaine, neurological conditions, or blood disorders. Subjects were not currently taking any medication that could affect central nervous system function (i.e. antidepressants, barbiturates, narcotics, and sedatives). Additionally, female subjects were not taking oral contraceptives. All females who qualified for this study reported for testing during the early follicular phase of their menstrual cycle in attempt to standardize testing procedures, as the effect of hormone release on the H-reflex is unknown. Subjects were assessed for inclusion criteria via a pre-participation history questionnaire, a physical examination, and a reflex screening. Informed consent to participate in this study was obtained prior to subject participation. Human subject approval (#10322) was obtained from the Human Investigations Committee at the University of Virginia prior to beginning the study.

### 2.2. Instrumentation

H-reflex and M-response measurements were collected using surface electromyography (MP150, BIOPAC Systems Inc., Santa Barbara, CA). Signals were amplified (DA100B, BIOPAC Systems Inc.; gain 1000) from disposable, 10 mm pre-gelled Ag–AgCl electrodes (EL503, BIOPAC Systems Inc.). The EMG signal was band-pass filtered from 10 to 500 Hz and sampled at 1024 Hz with a common-mode rejection ratio of 110 dB. Reflex measurements were elicited using the BIOPAC stimulator module (STM100A, BIOPAC

Systems Inc.) with a 200-V (maximum) stimulus isolation adapter (STMISOC, BIOPAC Systems Inc.), 2 mm shielded disc electrode (EL254S, BIOPAC Systems Inc.) and a 7 cm dispersive pad.

### 2.3. Reflex screening

A screening session was held in the Sports Medicine/Athletic Training Research Laboratory for all volunteers prior to enrollment in the study. A general explanation of the study and its significance was given along with an explanation of the measurement protocol, the risks involved in participation were explained, and informed consent was obtained. H-reflex and M-response measurements were recorded in the soleus musculature to ensure subjects had readable measurements necessary for data collection. If readable measures were not obtained the subject was dismissed (all subjects had the needed measures and therefore none had to be dismissed). After the H-reflex and M-response were elicited the volunteers were randomly assigned to a testing order (effusion/control). Each subject was considered as a block, and the sequence of the conditions was assigned to each subject by way of a computer generated random permutation.

### 2.4. Subject preparation

Each subject was shaved, debrided, and cleaned with isopropyl alcohol over the soleus musculature for reception of the EMG electrodes. Recording electrodes were placed over the midline of the soleus and a ground electrode positioned on the medial malleolus. A cathode was placed over the tibial nerve and an anode positioned superior to the patella. Adhesive collars were applied to the cathode in order to maintain its position over the nerve for the duration of the data collection.

### 2.5. Testing procedures

#### 2.5.1. Effusion/control testing sessions

Volunteers were admitted into the General Clinical Research Center (GCRC) the evening prior to testing. Subjects were shaved over the sites for EMG electrode placement. Subjects were fed a standardized snack at 23:00, then fasted until the completion of the testing protocol. Subjects were then asked to prepare for bed and lights were turned off by 24:00. Subject were awoken at 05:50 and asked to void. Subjects then returned to bed and remained lying down until the completion of testing. Beginning at 06:30 baseline neurophysiological measures ($M_{max}$, H-reflex at 25% of $M_{max}$, and conditioned reflexes for pre- and post-synaptic inhibition) were recorded. Volunteers were then prepared for the knee injection. A 25 G 1 1/2″ needle was inserted and removed to mimic the puncture caused during the Xylocaine injection. This injection was performed to determine if the needle puncture itself or the pain that possibly resulted from the needle stick influenced our dependent measures. The Xylocaine injection was performed followed by the injection of the sterile saline. Neurophysiological measurements were recorded after the first needle insertion and after the Xylocaine injection. Measurements were taken again at 25 and 45 min post-saline injection. Upon completion of testing, the electrodes were removed, subjects were fed, and then discharged from the GCRC.

#### 2.5.1.2. Control

Subject admission to the GCRC was the same as described above. All measurements were time matched to the effusion condition. No injections (needle stick, Lidocaine, or saline) took place during this condition.

### 2.6. H-reflex and M-response procedures

Volunteers were in a semi-reclined position with the involved knee in approximately 15% of flexion and their ankle in a neutral position. Subjects were asked to place their hands to their sides and to keep their head facing forward [18,22].

A series of 1-ms [36] square wave pulses were delivered to each volunteer's tibial nerve in order to obtain the peak-to-peak amplitude of the H-reflex and M-response. Stimuli were given with 10 s rest intervals in between to ensure post-activation depression did not interfere with the H-reflex amplitude [37]. The stimulus intensity was set to elicit an H-reflex at 25% of the maximum M-response ($M_{max}$) and five measures were then recorded.

### 2.7. Paired reflex depression procedures

The method to elicit PRD (a measure of reflex activation history) in humans has been described in detail elsewhere [47]. Briefly, dual stimuli were delivered to the posterior tibial nerve 80 ms apart to evoke soleus H-reflexes while subjects lay supine (Fig. 1). Stimulus intensity was set to elicit H-reflexes at ~25% of $M_{max}$. The depression of the second H-reflex relative to the first (% depression) was used for data analysis. The decrease in the second reflex is referred to as PRD, and represents modulation of processes controlling rate dependent reflex depression and the influence of the reflex activation history [47]. Eight measures were recorded and averaged in order to improve the intra-session reliability of the protocol [9].

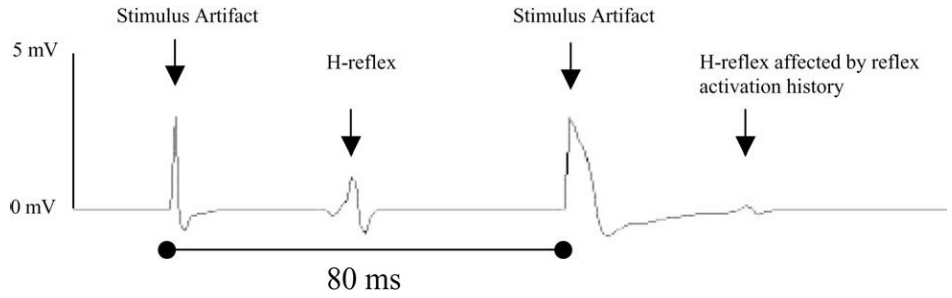


Fig. 1. Tracing of the paired reflex depression protocol. The stimuli to deliver the H-reflexes were set to the same intensity and delivered 80 ms apart. Note the depression in the second reflex compared to the first.

### 2.8. Recurrent inhibition procedures

Recurrent inhibition to soleus alpha motoneurons was brought about by a conditioning H-reflex (H1) and was estimated by a subsequent test H-reflex (H′). To elicit the conditioning H-reflex, stimulus 1 (S1) was adjusted to an intensity that evoked an H-reflex of approximately 25% of the $M_{max}$. Stimulus 2 (S2) was set at an intensity to evoke activity in all motor axons ($M_{max}$) and created the afferent volley resulting in H′. The inter-pulse interval was set at 10 ms [4,9]. When S2 is applied alone, an H-reflex will not appear due to its collision with the antidromic volley occurring in the motor axons. However, when S2 is preceded by S1 at a 10 ms inter-pulse interval, H1 collides with the antidromic volley, allowing H′ to pass (Fig. 2). H′ can only pass along the motor fibers in which the collision occurred, therefore the observed H′ is produced by only those motoneurons that give rise to H1. The percentage of recurrent inhibition was evaluated by comparing the amplitude of H′ to H1. Both reflexes were subjected to the same type of influences that may modify soleus monosynaptic reflex excitability, but only H′ could be influenced by recurrent inhibition evoked by the conditioning H-reflex discharge [4,9].

### 2.9. Joint effusion procedures

An area superolateral to the patella was cleaned with alcohol and povidone-iodine. While lying supine in bed, the subjects' lower limb was extended. Using a 25 G needle, a needle stick was made subcutaneously.

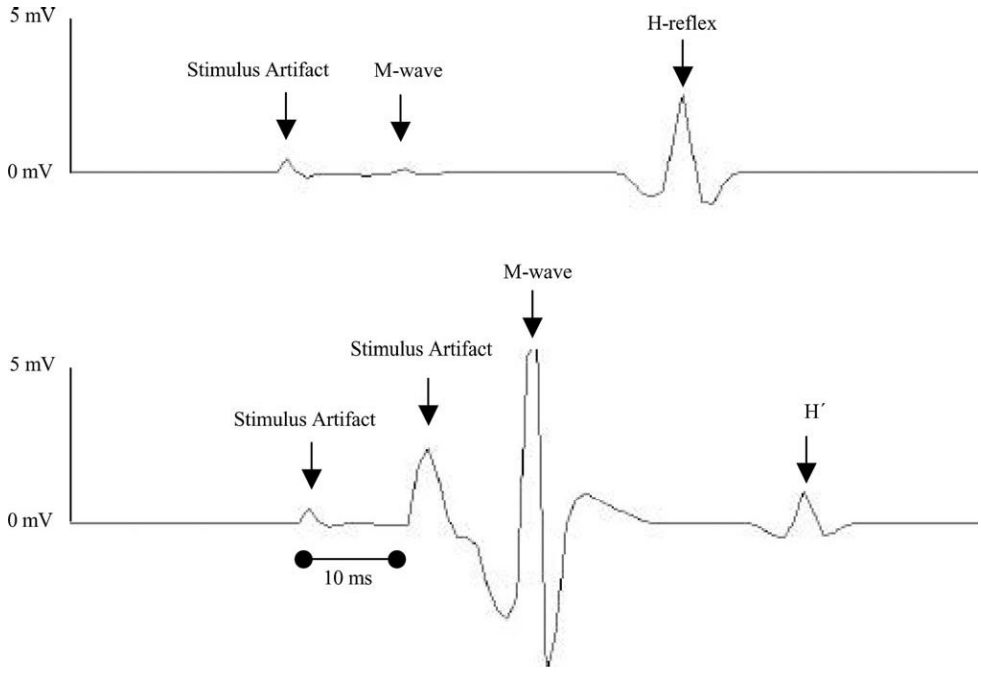


Fig. 2. Tracing of the recurrent inhibition protocol. Top tracing is S1 delivered alone eliciting an H-reflex at 25% of the entire MN pool. Bottom tracing S1 and S2 delivered together. H′ is seen in the tracing due to the H-reflex elicited by S1 and the antidromic impulses generated by S2 colliding and eliminating each other. Note the depression of the H-reflex in the bottom tracing compared to the H-reflex in the top tracing. The depression of the H-reflex in the bottom tracing is due to activation of Renshaw cells.

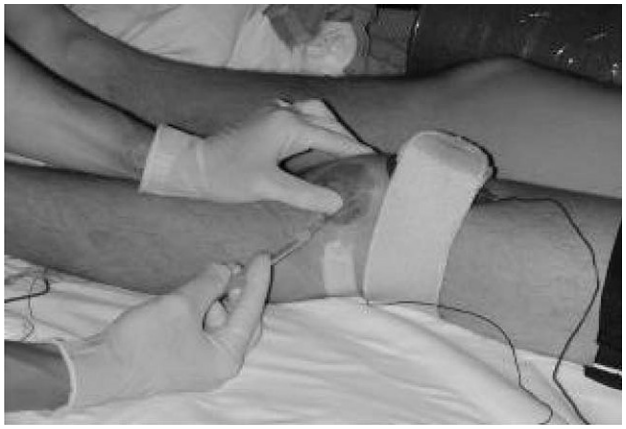

Fig. 3. Injection of Xylocaine into the subcutaneous tissue surrounding the knee.

This injection was necessary in order to evaluate the effect of a needle stick on our dependent measures. Using a sterile, disposable syringe with a 25 G 1 1/2″ needle, 3 ml of 1% Xylocaine was injected subcutaneously for anesthetic purposes (Fig. 3). When injecting the saline into the intra-articular space, the physician manually everted and moved the patella laterally using his free hand. Using a disposable syringe with a 21 G needle attached, 60 ml of saline were injected. The needle was advanced between the articular surface of the patellofemoral joint at the midpoint of the patella [28]. The injection of the saline resulted in a considerable effusion (Fig. 4). Sterile disposable gloves were worn during each injection for all subjects.

### 2.10. Statistical analysis

An a priori power analysis based on our previous work examining differences in the maximum soleus H-reflex prior to and after injection of saline into the knee joint revealed an average effect size of 1.77 mV and an average standard deviation of 1.38 mV. Using the value 1.38 mV as the estimate for measurement dispersion and assuming a sample size of 10 knees per group, we calculated a minimum detectable effect size of 1.71 mV as the smallest within-subject change that would lead one to reject the null hypothesis of no effect when the hypothesis rejection criterion is based on a statistical test with a two-sided type I error rate of 0.05 and a power of 0.80.

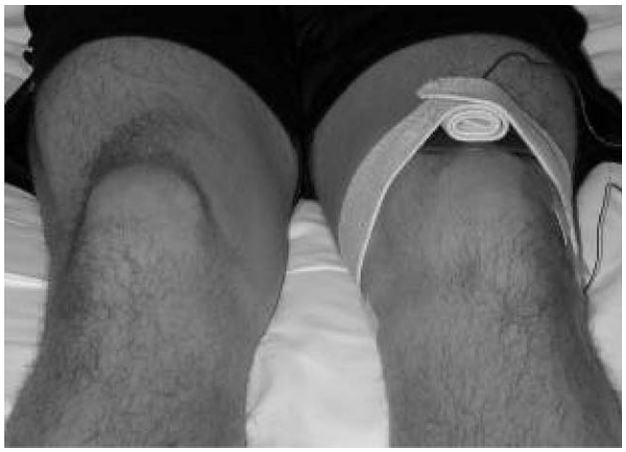

Fig. 4. Bilateral knee joint comparison following the saline injection. Note the effusion in the left knee when compared to the right.

A mixed effects model for repeated measures was used for each dependent variable. The model specifications included two independent variables: condition and measurement interval. Condition was modeled as the between subject factor, while measurement interval was modeled as a within subject factor. Measurement interval by condition interaction was also considered. The model parameters were estimated by restricted maximum likelihood. The covariance matrix was modeled in a spatial power forum. Comparisons of interactions were formulated by one degree of freedom contrasts between the groups mean. Multiple comparison type I error rate adjustment was based on Fisher's least significant difference criterion (FLSD) with a type I error rate of 0.05.

## 3. Results

### 3.1. Recurrent inhibition

A significant interaction was found between measurement interval and condition ($F_{3,54}= 12.79$; $P < 0.0001$). The conditioned reflex during the effusion condition increased from baseline ($20.3 \pm 21$) at 25 min ($24.5 \pm 22.5$; $P< 0.0001$) and 45 min ($25.4 \pm 22.2$; $P < 0.0001$) post-knee joint effusion (Fig. 5). The conditioned reflex post-needle stick and post-Xylocaine did not change from baseline ($P > 0.05$). Additionally, no difference was detected between 25 and 45 min post-knee joint effusion for the effusion condition ($P > 0.05$). No differences were noted between any of the measurement intervals during the control condition ($P > 0.05$).

### 3.2. Paired reflex depression

A significant measurement interval by condition interaction was found ($F_{3,54}= 26.49$; $P< 0.0001$). The conditioned reflex during the effusion condition increased from baseline ($17.1 \pm 13.1$ V) at 25 min ($24.5 \pm 15.1$; $P < 0.0001$) and 45 min ($29.4 \pm 19.6$; $P < 0.0001$) post-knee joint effusion (Fig. 6). The conditioned reflex post-needle stick and post-Xylocaine

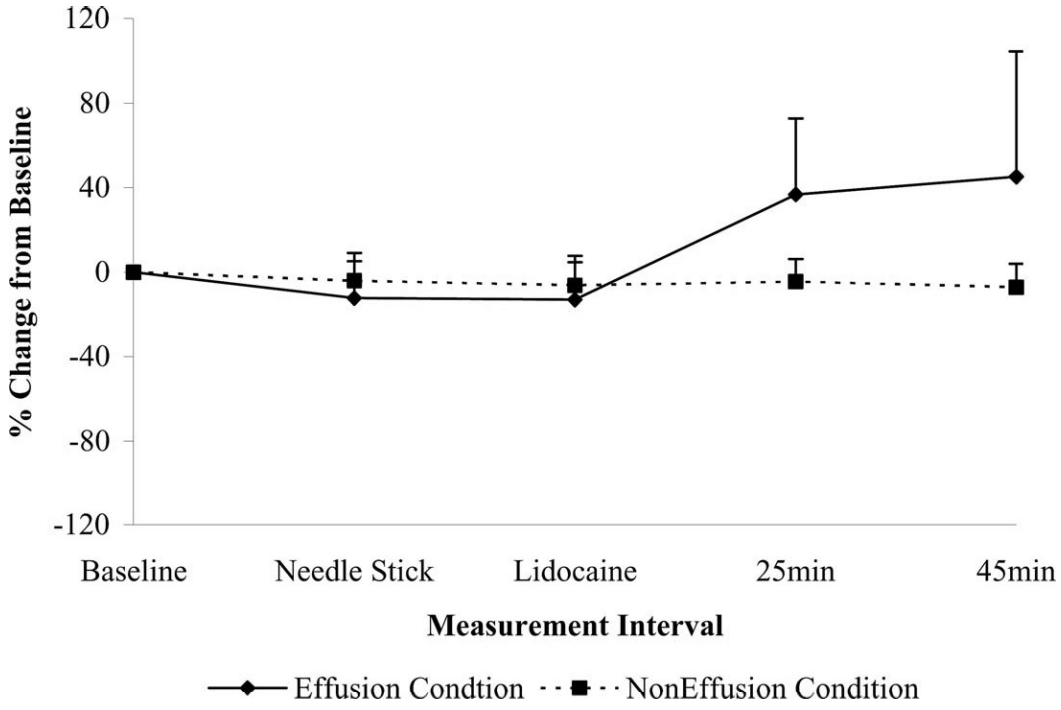


Fig. 5. Averaged conditioned H-reflex amplitude as a percentage of unconditioned reflex amplitude expressed as a percentage change from the baseline value. Values above 0 represent a decrease in recurrent inhibition and values below 0 represent an increase in recurrent inhibition (±SD).

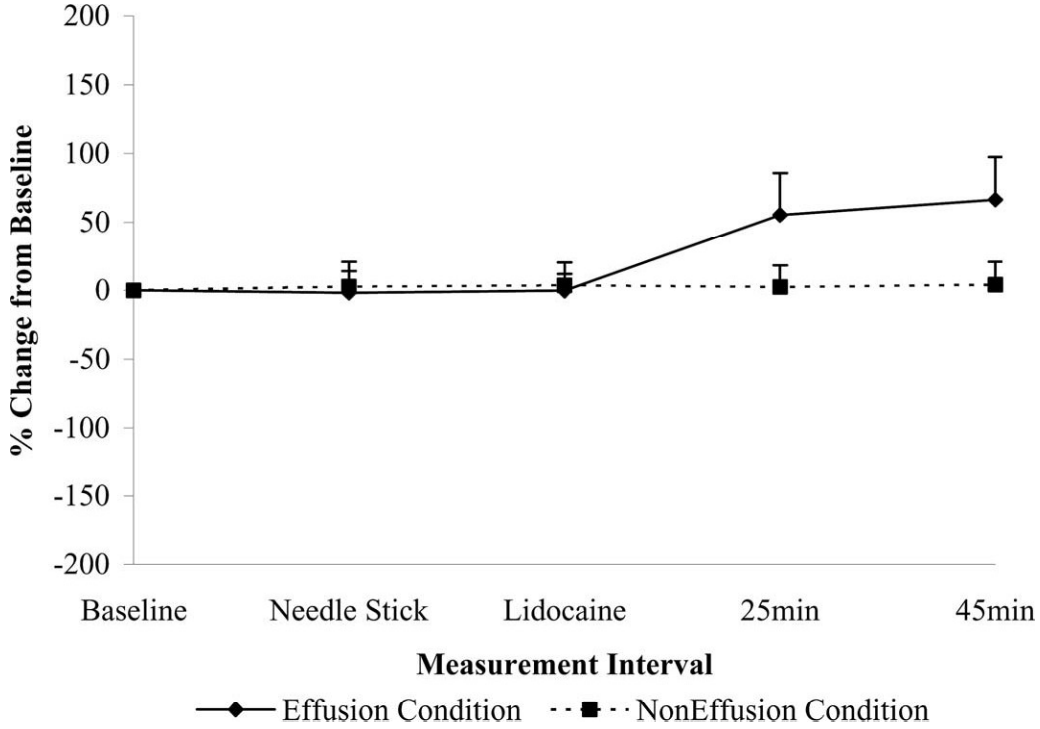


Fig. 6. Conditioned H-reflex as a percentage of unconditioned reflex expressed as a percentage change from the baseline value. Values above 0 represent a decrease in paired reflex depression and values below 0 represent an increase in paired reflex depression (±SD).

did not change from baseline. Additionally, no difference was detected between 25 and 45 min post-knee joint effusion for the effusion condition ($P > 0.05$). No differences were noted between any of

the measurement intervals during the control condition ($P > 0.05$).

## 4. Discussion

Knee effusion has been shown to result in soleus facilitation [19,21]. Our data suggest that this AMR is due to both pre- and post-synaptic spinal mechanisms. Specifically, Renshaw cells and potentially the interneurons responsible for pre-synaptic inhibition contribute to the response of the musculature surrounding the knee joint following injury. Our observations diverge from previous hypotheses that arthrogenic muscle inhibition (AMI)/AMR is due only to activation of the Ib interneuron [20,27,50].

Previous activation of the Ia–motoneuron synapse is thought to result in decreased transmitter release with subsequent afferent volleys [7]. Additionally, it has been proposed that modulation of reflex depression associated with previous reflex activation may allow for summation of spindle afferent feedback to contribute to an increased neural drive allowing for a facilitated motor output during movement [47]. This modulation of the reflex depression associated with reflex activation history has been hypothesized to act with other pre-synaptic mechanisms functioning to gate motoneuron excitability [45,47].

The amount of reflex depression was reduced following induction of the experimental effusion leading us to speculate that a pre-synaptic regulatory control normally acting to decrease reflex excitability was modulated accounting for the increased excitability of soleus motoneurons. However, the mechanism by which this pre-synaptic control acts remains unknown. Descending pathways are known to influence reflex activation history through spinal interneurons. Specifically, GABA-ergic interneurons responsible for pre-synaptic inhibition are influenced by descending controls [31,41,45]. Post-activation depression, previous activation of a muscle resulting in a decrease in the H-reflex, can be viewed as being similar to the PRD protocol (both are the result of previous activation of muscle) used in this study. The amplitude of the H-reflex has been found to return to 66% of its original amplitude within 400–500 ms which corresponds with the time course for classic pre-synaptic inhibition [10] and has lead investigators to speculate about its involvement in post-activation depression [48]. Further work is needed to determine the mechanism(s) by which both post-activation depression and reflex activation history are modulated.

Activation of quadriceps muscle afferents has been shown to lead to pre-synaptic inhibition in the soleus H-reflex [5,32]. The reflex inhibition present in the quadriceps musculature prevents quadriceps activation and likely reduces afferent activity arising from the muscle. Depressed afferent activity arising from the quadriceps muscle group may lead to the increased excitability of the soleus due to a release of pre-synaptic inhibition.

Under normal conditions reflex activity caused by afferent information is reduced by supraspinal systems allowing for efficient and precise control of movement [3]. Perhaps, when a joint effusion is present afferent traffic originating from Ruffini endings decreases the effectiveness of descending pathways. By "shutting off" supraspinal systems ultimately responsible for pre-synaptic control of motor output afferent impulses may summate and contribute to the increased motor output to the soleus muscle.

The decrease in pre-synaptic control was accompanied by reduced post-synaptic control to the soleus alpha motoneurons in the presence of an effusion. Recurrent inhibition of alpha motoneurons has been described as a gating mechanism functioning to reduce the sensitivity of alpha motoneurons to changes in their excitatory drive. Activation of Renshaw cells are also thought to control the frequency of firing of motor units [2,49]. The decrease in recurrent inhibition potentially allows for afferent feedback from the periphery to preferentially control the output of motoneurons. Descending pathways controlling Renshaw cells may become less active (similar to what was described above for the pre-synaptic mechanism) allowing for afferent feedback to be a major contributor to how the body responds to a perturbation or movement.

The fact that both pre- and post-synaptic inhibition is reduced to the soleus is likely a response by the central nervous system allowing more motoneurons to be available to the postural musculature in an attempt to compensate for decreased availability of motoneurons to the quadriceps muscle group. The decreased inhibition seen with both pre- and post-synaptic control mechanisms may allow for the triceps surae muscle group to make rapid postural adaptations during gait in an attempt to promote a stable standing posture despite the quadriceps weakness and knee joint instability. However, it should be mentioned that this "lack of" central control to the soleus muscle may possibly promote unstable and uncontrolled movement potentially causing more harm to the extremity. Available data examining the control of posture and gait following knee effusion suggest the former to be true. Static postural stability has been shown to improve following induction of a knee joint effusion [34]. A study evaluating walking gait in the presence of an effusion noted a quadriceps avoidance gait pattern in which subjects demonstrated a decrease in knee joint torque and quadriceps EMG, and an increase in hip extensor work and hamstring EMG [46]. Although they did not

evaluate the contribution of the lower leg musculature to this gait pattern, one may speculate that activity in the soleus musculature is enhanced assisting the hip joint and surrounding musculature in providing lower extremity support and allowing for propulsion during walking. How lower extremity joint kinematics and kinetics respond to perturbations when an effusion is present remains to be answered and requires future study. It should be noted that our measurements were taken while subjects were lying supine and the results may be different had they been performing a more functional movement. However, we speculate that soleus AMR would be mediated by both pre- and post-synaptic spinal mechanisms during movement. The amount of pre-synaptic vs. post-synaptic inhibition present during function may differ depending on the type of task (i.e. a power type movement compared to one that requires endurance) [8].

Future work should be conducted to determine the involvement of pre- and post-synaptic mechanisms in patients who have sustained knee damage. Studies examining the influence of therapeutic interventions on muscle activation following knee injury are also needed in order to aid clinicians in returning patients back to pre-injury status.

In conclusion, the AMR seen in the soleus musculature following joint effusion is regulated by both pre- and post-synaptic control mechanisms. These data provide valuable information about the spinal inhibitory mechanisms contributing to the response of the lower extremity musculature when a joint effusion is present.

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**Riann M. Palmieri** is an Assistant Professor and the Program Director of the Undergraduate Athletic Training Education Program at the University of Michigan. She also serves as the Director of the Neuromuscular Research Laboratory and the Co-director of the Human Neuromechanics Laboratory. Riann earned her B.S. in Athletic Training from California University of Pennsylvania, her M.S. in Athletic Training at Indiana State University, and her Ph.D. in Sports Medicine at the University of Virginia. Her research interests include the neuromuscular consequences of joint injury, adaptations to anterior cruciate ligament injury, and the role of sports injury in the development of degenerative joint disease.

**James A. Tom** is currently a clinical instructor in the Department of Orthopedic Surgery at Drexel University. He received his doctor of medicine degree in 1997 from the Albert Einstein College of Medicine, where he subsequently completed his residency training in Orthopedic Surgery.

**Jeffrey Edwards** is an Associate Professor in the Department of Physical Education and the Department of Athletic Training. His B.S. is from Indiana State University, his M.S. from Ball State University, and his Ph.D. was earned at Indiana University. His research interests include neuromuscular adaptations to injury or exercise, and measurement of human energy expenditure.

**Arthur Weltman** is a Professor in the Department of Human Services (School of Education) and in the Department of Medicine and is the Director of the Graduate Program in Exercise Physiology in his own department and Director of the Exercise Physiology Laboratory in the General Clinical Research Center (GCRC). Dr. Weltman is a Principal or Co-Investigator on a number of NIH and Industry supported grants. His current research interests include an examination of the relationship among exercise, body composition, regional distribution of body fat, strength, balance, function, and hormones.

**Ethan N. Saliba** is the Head Athletic Trainer, an Assistant Professor in the Departments of Human Services and Clinical Orthopedic Surgery, as well as an Adjunct Assistant Professor in the Department of Physical Medicine and Rehabilitation at the University of Virginia. He earned his B.S. in Physical Therapy at the University of Kansas, and his M.Ed. and Ph.D. in Sports Medicine at the University of Virginia.

**Danny J. Mistry** is currently a postdoctoral fellow in the center for complimentary and alternative medicine as well as an assistant professor in the Departments of Physical Medicine and Rehabilitation and Curry School of Education. He completed his residency training at the University of Virginia Health System—two years in Physical Medicine and Rehabilitation and five years in Internal Medicine, with three of these years as a research fellow in cardiovascular physiology. He received his doctor of medicine degree in 1986 from the University of Madras in Madras, India, following which he completed residency training at Breach Candy Hospital in Bombay, India. Dr. Mistry also holds a master's degree in exercise science from the University of Massachusetts and is a certified athletic trainer and massage therapist. His current research interests include the effects of various bioelectromagnetic therapeutic modalities on tissue healing.

**Christopher D. Ingersoll** is the Joe H. Gieck Professor of Sports Medicine, Director of the Graduate Programs in Sports Medicine/Athletic Training, and Adjunct Professor in the Department of Physical Medicine and Rehabilitation at the University of Virginia. He also directs the Sports Medicine/Athletic Training Research Laboratory and serves as the strength, balance and function consultant to the University of Virginia General Clinical Research Center. He earned his B.S. in Sports Medicine from Marietta College (OH), his M.A. in Athletic Training at Indiana State University, and his Ph.D. in Biomechanics at the University of Toledo. Ingersoll is a member of the National Athletic Trainers' Association and currently serves as Editor-in-Chief of the *Journal of Athletic Training*. He is also a fellow in the American College of Sports Medicine. He has authored or coauthored over 70 peer reviewed publications, 180 presentations at scientific meetings, and 40 invited presentations or symposia in the areas of Sports Medicine, Athletic Training and Neuroscience.