

# AN INVESTIGATION INTO THE EFFECTS OF A SIMULATED EFFUSION IN HEALTHY SUBJECTS ON KNEE KINEMATICS AND LOWER LIMB MUSCLE ACTIVITY DURING A SINGLE LEG DROP LANDING

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Arthrogenic muscle inhibition (AMI) is defined as an ongoing reflex inhibition of the musculature surrounding a joint following distension or damage to the structures of that joint [Hopkins and Ingersoll, 2000]. AMI following joint injury may affect movement and muscle recruitment which may impair rehabilitation and delay the return to activity. Knee angular displacement and velocity as well as lower limb EMG were measured in the period 250 milliseconds pre initial contact to 250 milliseconds post initial contact during a single leg drop jump in 8 healthy subjects before and after a simulated knee joint effusion of 60 millilitres. Repeated measures ANOVA and post hoc testing revealed no statistically significant differences in pre and post effusion in knee kinematic or lower limb EMG measures undertaken. A simulated knee effusion did not result in significant alterations to knee joint mechanics or lower limb muscle activation patterns during a single leg drop landing. The mechanism by which an effusion affects motor control during functional and dynamic weight bearing tasks warrants further investigation.

**KEY WORDS:** drop landing, knee, effusion, kinematics, EMG

**INTRODUCTION:** Arthrogenic muscle inhibition (AMI) is defined as an ongoing reflex inhibition of the musculature surrounding a joint following distension or damage to the structures of that joint (*Hopkins and Ingersoll. 2000*). It is the natural response of the body to injury which may result in inadequate neuromuscular control in functional activities and a delay in the return to activity. Long term effects of inactivity following injury, potentially caused by AMI, can adversely affect muscles, bones, ligaments, and neural activity. Inhibition of the quadriceps is most likely the cause of strength loss, atrophy, and deficits in neuromuscular control after knee injury (*Hopkins et al. 2001*). The presence of an effusion can lead to persistent quadriceps muscle weakness, resulting in knee instability as the capacity of the muscle group to respond to external loads generated by functional activity is compromised. Insufficient control and strength at the knee as a result of AMI can lead predispose an individual to reinjury as well as to the development of chronic degenerative conditions. An individual's post injury or pathological state cannot establish whether a change in movement or muscle recruitment patterns is a direct result of knee pathology or contributed to its etiology. The effusion model has the advantage of nullifying other factors of injury, pain and inflammation which are difficult to quantify. Recent investigations have assessed the effects of a simulated effusion on a range of measurements at the knee joint including muscle strength, postural control, proprioception and quadriceps H-reflex. However few studies have quantified the impact of this type of effusion on high speed dynamic weight bearing activity. Therefore the aim of this research was to quantify the effects of a simulated effusion on knee movement patterns and lower limb muscle activity in the period pre and post IC during a single leg drop landing in a sole testing session. An understanding of the influence that an effusion may have on this type of dynamic function could assist therapists in understanding the myriad of abnormalities associated with neuromuscular control due to injury and in the rehabilitation from lower limb injuries.

**METHODS: Data Collection:** Eight physically active subjects who gave informed consent participated in this study (24.6 years  $\pm$  4.3, 174.1 cm  $\pm$  0.1, 70.6 kg  $\pm$  12.5). Subjects stood on a 35 centimetre high platform adjacent to a rectangular force plate embedded in the laboratory floor with the test leg relaxed and non-weight bearing. The subject then used the contralateral leg to propel him/herself from the platform and stick the landing when impacting

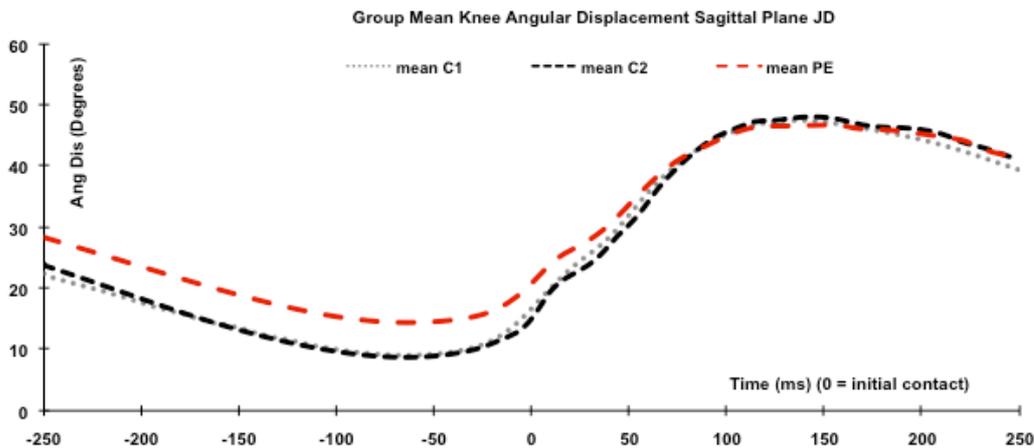
on the force plate. The motion analysis system (CODA, Charnwood Dynamics Ltd, Leicestershire, UK), force plate and EMG were manually triggered to simultaneously begin recording as the subject was given a verbal command to land on the platform. Surface EMG activity from the vastus medialis (VM), vastus lateralis (VL), biceps femoris (BF) and soleus (SOL) muscles were recorded on all subjects during the drop landing. Electrodes were placed on specific sites in accordance with the SENIAM research groups recommendations. Data was recorded on a Biopac MP100A (Biopac Systems Inc. Santa Barbara, CA, USA). In order to address potential measurement error in data recording, data was recorded in three measurement intervals throughout the testing session, twice prior to the effusion, Control 1 (C1) and Control 2 (C2), and once following the effusion, Post Effusion (PE). Following the control testing sessions, 2 ml of 2% Lidocaine was injected subcutaneously lateral to the knee joint line for anaesthetic purposes and 60 ml of saline solution (0.9% w/v Sodium Chloride Intravenous Infusion) was subsequently injected into the knee joint capsule. A ballotable patella test and an effusion wave test were performed to ensure that the effusion was within the knee joint.

**Data Analysis:** Sagittal and coronal knee angular displacement and velocity at initial contact (IC), and peak/trough values during the 250 ms period prior to and following IC were identified for five drop landing trials for each subject. IC during the single leg drop jump was identified using the vertical component of the ground reaction force (GRF) using 15 Newtons (N) as a threshold for detection of impact. Forceplate data was collected at 200 Hz sampling rate. EMG signals were amplified (gain 300) and sampled at a rate of 1000 Hz. They were subsequently band pass filtered (Blackman 61 dB) at 20 Hz (low) and 500 Hz (high). The data was then full wave rectified and average over a 15 ms moving window. The selected time periods were the period 250 ms pre to cover the initial EMG pre activation prior to ground contact and 250 ms post ground contact as this corresponded with the initial weight acceptance phase. Statistical analysis was carried out using SPSS for Windows (Version 12.0.1; SPSS Inc, Chicago, IL, USA). We used a general linear model three factor repeated measures analysis of variance to analyse differences in kinematic/EMG variables at each of the test intervals. In each case the dependent variable was the kinematic/EMG variable in question and the independent variables were test interval (C1, C2 and PE). Post hoc paired t-tests were then carried out to test for differences in variables between individual pairs of test intervals (C1vC2, C2vPE, C1vPE). The alpha level was set at 0.05. Due to the potential for multiple comparison errors in the analysis, we used a Bonferroni adjustment to re-calculate the P value for the repeated measures and post hoc t-tests with adjusted critical P values of 0.001 and 0.002 respectively at a 95% confidence level for kinematics. Similarly, Bonferroni adjustments were also used for EMG calculations with adjusted critical P values of 0.002 and 0.003 at a 95% confidence level for the repeated measures and post hoc tests respectively.

**RESULTS:** Repeated measures ANOVA and post hoc testing revealed no statistically significant differences in pre and post effusion in knee kinematic (Fig 1) or lower limb EMG (Table 1) measures undertaken in 8 healthy subjects during a single leg drop landing task.

**Table 1: EMG activity during the 250ms pre IC and 250ms post IC drop landing**

Muscle	Variable	Measurement Interval			Repeated Measures ANOVA Level of Significance
		C1	C2	PE	
Vastus Medialis	250ms to IC	23.83 (6.93)	20.25 (4.60)	17.92 (6.57)	0.04
	IC to 250ms	60.24 (10.25)	58.40 (11.00)	49.31 (19.30)	0.09
Vastus Lateralis	250ms to IC	22.98 (4.43)	19.17 (5.28)	18.06 (6.65)	0.05
	IC to 250ms	60.91 (10.02)	59.37 (7.33)	49.95 (13.60)	0.15
Biceps Femoris	250ms to IC	27.20 (12.26)	26.24 (16.8)	19.13 (17.70)	0.07
	IC to 250ms	44.58 (16.21)	36.82 (12.96)	26.85 (10.43)	0.08
Soleus	250ms to IC	23.77 (7.04)	19.76 (5.79)	20.58 (8.15)	0.25
	IC to 250ms	40.96 (8.77)	39.23 (8.28)	38.40 (9.90)	0.63



**Fig 1: Knee angular displacement sagittal plane during the 250ms pre IC and 250ms post IC**

**DISCUSSION:** We hypothesised that there would be a significant decrease in quadriceps muscle activity based on previous research (Torry *et al.* 2000 and 2005, Palmieri Smith *et al.* 2007). These muscles have a crucial role in stabilising the knee joint when landing from a jump as their eccentric contraction assists in dissipating large forces generated during this task. A decrease in their recruitment capability could reduce stability of the knee joint during a dynamic task which may predispose a patient to initial or repeated injury. The principal finding in this study was that a 60 ml simulated effusion of the knee joint does not result in alterations to knee kinematics or lower limb muscle activity in healthy subjects during single leg drop landing. The only other similar study in this area was conducted by Palmieri-Smith *et al.* (2007) which used two levels of effusion (30 ml and 60 ml) and measured sagittal plane kinematics and lower limb EMG in the period 250 ms post IC. The results of that study demonstrated a decrease in knee flexion position at landing using an effusion of 60 ml, as well as a reduction in vastus medialis and vastus lateralis muscle activity between the high and low level effusions. The authors concluded that a reduction in muscle activity altered landing mechanics and therefore larger forces to be transferred through the knee as

evidenced by an increased ground reaction force. The present study did not observe similar sagittal plane kinematics or EMG results. The lack of correlation between the findings of these two studies may be due to methodological differences, for example the methodology of the EMG analysis. EMG is normalised in studies to allow comparisons between conditions and/or subjects. The *Palmieri-Smith et al (2007)* study normalised their EMG by comparing their recording to that from a maximum voluntary isometric contraction [MVIC] and expressing the EMG as a percentage of maximal contraction. We choose to normalise our EMG data to mean or peak of an ensemble average as *Yang and Winter (1984)* have reported that normalising EMG with respect to MVIC is less reliable than normalising relative to a sub maximal contraction as well as been reported as have large inter subject variability. The disparity between the two studies may also have been due to the measurement protocol used in our investigation. Our data collection was conducted in a single testing session whereas the *Palmieri-Smith et al (2007)* study was conducted at four separate testing sessions on different days. In a reliability study, *Monaghan et al (2006)* observed limits of agreement of over eight degrees in sagittal plane gait variables during gait between testing sessions on different days. These limits may increase in a task such as drop landing as the patterns of movement are not as robust as that of gait. Coupled with this, in order to address the issue of test-retest variance that is inherent in kinematic studies we examined measures in three separate measurement intervals. We could have arrived at a different set of conclusions to those presented here had we employed a straightforward test-effusion-retest model and if we had not applied the Bonferroni adjusted p-values to our analysis to account for multiple comparisons. In particular a number of the EMG findings would have either bordered on or displayed statistical significance.

**CONCLUSION:** Despite these levels of effusion having an effect on patients in a clinical setting, they may not be sufficient to elicit major changes in movement patterns and muscle function during high velocity cyclical tasks in healthy subjects following a simulated effusion. Allied with this, the presence of a long term effusion with associated inflammation and pain in acute or chronically injured patients with adaptations to learned movement and muscle recruitment patterns may be responsible for the loss of proprioception and muscle inhibition as observed in clinical situations.

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