## AN INVESTIGATION INTO THE EFFECTS OF A SIMULATED EFFUSION IN HEALTHY SUBJECTS ON KNEE KINEMATICS AND MUSCLE ACTIVITY DURING JOGGING AND RUNNING.

<sup>1</sup>Garrett Coughlan, <sup>2</sup>Rod Mc Loughlin, <sup>1</sup>Ulrik Mc Carthy Persson and <sup>1</sup>Brian Caulfield

<sup>1</sup> School of Physiotherapy and Performance Science, University College Dublin, Ireland.

<sup>2</sup> O'Neill's Sports Injury Clinic, University College Dublin, Ireland; email: <u>garrett.coughlan@ucd.ie</u>

## **INTRODUCTION**

The aim of this study was to measure the effects of a high level effusion on dynamic, clinically and functionally applicable activities. Knee joint injuries are commonly associated with activities that demand high levels of stability during dynamic movement. Depending on the mechanism and type of injury, a resultant effusion distends the knee joint capsule resulting in arthrogenic muscle inhibition, which may lead to weakness and atrophy in the surrounding musculature [1]. The majority of the research to date on the effects of knee effusion has used a simulated effusion model to evaluate knee function of healthy subjects in static, non-functional positions.

# **METHODS**

12 physically active subjects were recruited from the local university population for the purpose of this study. All procedures, including gait analysis, EMG and knee effusion protocol, were carried out in the university motion capture laboratory. Data were 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). Ten minutes quiet rest was allowed between intervals.

Kinematic data were collected at a sampling rate of 200Hz for 20 seconds at treadmill velocities of 8kmh<sup>-1</sup> and 12kmh<sup>-1</sup> using an active marker based motion capture system (CODA). Surface EMG activity from the vastus medialis (VM), vastus lateralis (VL), biceps femoris (BF) and soleus (SOL) muscles were simultaneously recorded on all subjects during gait tasks on a Biopac MP100A and analysed using its associated Acq Knowledge software. The activity was gathered using preamplified electrodes which were placed on specific sites in SENIAM accordance with the research groups' recommendations [2]. Following the completion of the two initial control trials, C1 and C2, the subjects underwent a simulated knee effusion procedure. 2ml of 2% Lidocaine was injected subcutaneously lateral to the knee joint line for anaesthetic purposes. Thereafter 60ml of saline solution (0.9% w/v Sodium Chloride Intravenous Infusion) was injected into the knee joint capsule. The same physician conducted all injection procedures.

Statistical analysis was carried out using SPSS for Windows. We used a general linear model three factor repeated measures analysis of variance to analyse differences in kinematic and 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 kinematic 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, we used a modified Bonferroni adjustment as described by Hochberg [3], to re-calculate the P value for the repeated measures and post hoc t-tests.

#### **RESULTS AND DISCUSSION**

Repeated measures ANOVA revealed a statistically significant difference (P < 0.004) with a decrease in peak knee flexion in the period 250 milliseconds post heelstrike (HS) at 8kmh<sup>-1</sup> (C1 = 39.25° (6.17), C2 = 37.47° (5.82), PE = 34.41° (5.88). Pairwise post hoc comparisons revealed that the only comparison to reach the level of significance was that of a decrease in peak knee flexion 250 milliseconds post HS at 8kmh<sup>-1</sup> during C1 versus that post effusion (P<0.001). No other significant differences were found in a range of variables at velocities of 8kmh<sup>-1</sup> and 12kmh<sup>-1</sup>. This period is referred to as the initial load bearing response during the stance phase and serves to reduce the impact on the lower limb and smooth the centre of mass displacement during weight transfer, thereby reducing energy expenditure and forces on the knee joint [4].

The principal finding in this study was that minimal changes occurred in knee joint angular displacement and velocity during treadmill jogging and running following a simulated knee effusion of 60ml. However, no significant inhibition of the quadriceps muscles occurred in our study at this time. In contrast to previous studies [5,6], this investigation found that both VM and VL activity increased in the period 250 ms post HS between the C1 and PE intervals, (VM C1 = 58.47 (6.36), PE = 62.45%ms (5.17), VL C1 = 54.17%ms (9.85), PE = 60.62%ms (6.34)) although these results were not significant. This may be an attempt by the knee musculature to increase joint stabilisation to absorb full limb support and to protect the passive joint structures from harmful forces that may be increased by an effusion.

### CONCLUSION

We observed minimal significant changes in sagittal and coronal plane kinematics and lower limb EMG activity during treadmill jogging and running. However we cannot rule out the possibility that prolonged presence of an effusion as well as an inflammatory component may alter movement control about a joint through ongoing changes in feedback from joint structures. Future studies should be conducted with a larger sample size. This study has implications for rehabilitation of knee injuries in that it may be possible for these patients to return to functional activity and to participate in more dynamic activities at an earlier stage in the rehabilitation process.

# **ACKNOWLEDGEMENTS:**

University College Dublin Seed Funding Grant 2008

## **REFERENCES:**

- 1. Palmieri R, et al., J Electromyogr Kinesiol. 14:631-40, 2004
- 2. SENIAM Roessingh Research and Development, 1999
- 3. Hochberg Y Biometrika. 75:800-802, 1988
- 4. Lucareli PR, et al., Clinics. 61:295-300, 2006
- 5. Torry MR, et al., J. Sports Sci. Med. 4:1-8, 2000
- 6. Torry MR, et al., Clin. Biomech. 15:147-159, 2005