EFFECT OF OSTEOARHTITIS ON KNEE MUSCLE ACTIVATION, STABILITY AND FORCE CONTROL

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Introduction

Osteoarthritis (OA) is the most common chronic condition among older adults. The development of OA is mechanically driven by altering the stabilising integrity of the joint [Herzog, 2006]. Since muscle action is the only dynamic regulator of joint stability, agerelated changes in neuromuscular function may provide insight as to why OA develops in the absence of a traumatic knee joint injury. This study investigated the muscle activation patterns of young healthy adults (YC), older healthy adults (OC), and adults with OA during a standing force control task.

Methods

A force matching protocol [Flaxman, 2012] was used to evaluate muscle activation patterns of 41 YC, 16 OC, and 19 OA. Subjects stood with their dominant foot fixed to a force platform and isometrically modulated ground reaction forces along the horizontal plane. With equal body weight on each leg, subjects generated 30% of their maximal force in 12 different directions, corresponding to various combinations of medial-lateral-anterior-posterior loads.

Electromyography (EMG) of 8 muscles that cross the knee joint, kinetics and kinematics were recorded. Processed EMG was normalised to previously recorded maximum voluntary isometric contraction and ensemble averaged into group means for each loading direction. Polar plots displayed muscle activation patterns, which were quantified with symmetry analyses, mean activation levels (X_{EMG}) , directions (Φ) , and specificity indices

(SI). Group differences were tested with independent T-tests at α <0.05.

Results

Similar muscle activation patterns were observed in all groups (i.e. symmetry and Φ) (Figure 1). However, in all muscles, the X_{EMG} was significantly greater in OC and OA compared to YC. OA displayed significantly greater X_{EMG} in the quadriceps and lower SI in rectus femoris and hamstrings than OC.

Discussion

Our results indicate that regardless of loading direction, both OC and OA groups have greater levels of muscle co-contraction than YC. This is suggested to be an adaptive response to age-related changes in muscle strength and force control [Rudolph, 2007]. Considering that individuals with OA have reduced muscle strength and force control compared to age-matched controls [Rudolph, 2007], it is suggested that the greater levels of activation and decreased specificity observed in our OA group is this "stiffening" response adapted to an extent that may expose the joint to detrimental loading conditions. Further investigation regarding age-related neuromuscular changes and their influence on the development of OA is warranted.

References

Flaxman et al, J Biomech, 45:2570-6, 2012. Herzog & Federico, Biomechan Model Mechanobiol 5: 64–81, 2006. Rudolph et al, Phys Ther, 87:1422-32, 2007.

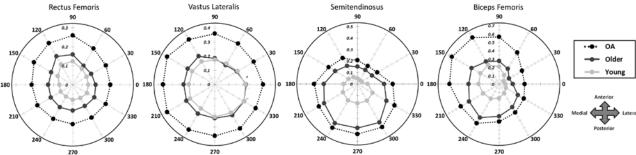


Figure 1: EMG polar plots of group mean activation patterns. Numbers along the circular trajectory represent loading location angle (°). Numbers along the radii represent normalised EMG magnitude.