

MECHANISMS UNDERLYING REDUCTIONS IN KNEE EXTENSION STRENGTH IN KNEE OSTEOARTHRITIS

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INTRODUCTION

Quadriceps weakness (inferred from knee extension strength) is a common symptom in individuals with knee osteoarthritis (OA) (Hurley et al, 1998). Knee OA patients have deficiencies of 10-60% during maximal voluntary isometric extension (MVIE) compared with healthy age matched controls (Pap and Machner, 2004). Three factors thought to be responsible for this are 1) disuse atrophy, 2) quadriceps muscle inhibition (QMI), and 3) antagonist muscle co-contraction (ACC).

QMI in knee OA has been measured using the burst super-imposition technique (a train of pulses) or the twitch interpolation technique (a single pulse). The former method is preferred when examining QMI at high force levels (Stackhouse et al, 2000). With sufficient practice, motivation, feedback and rest knee OA patients showed no difference to controls in voluntary activation levels, and disuse atrophy was suggested as the likely cause of quadriceps weakness (Lewek et al, 2004). However, they did not measure ACC and, therefore, could not eliminate this as a cause for the apparent quadriceps weakness.

Knee muscle co-contraction occurs to improve movement efficiency, protect joints (Kellis, 1998) and decrease anterior shear forces (Kingma et al, 2004). Small amounts of ACC (10%) occur in MVIE trials of healthy adults (Busse et al, 2006). Since knee OA patients increase ACC during walking, possibly due to pain (Hubley-Kozey et al 2008), it is possible that abnormal ACC levels

could decrease apparent knee extension strength in knee OA patients.

Therefore the purpose of this study was to examine the quadriceps strength, QMI and ACC during MVIE of knee OA patients.

METHODS AND PROCEDURES

Fifty-two patients (age 65.5 ± 7.6 years, height 1.70 ± 0.09 m, mass 81.4 ± 14.2 kg) previously diagnosed with OA and twenty-seven controls (age 64.9 ± 5.3 years, height 1.70 ± 0.09 m, mass 71.2 ± 13.2 kg) were recruited for this study. Exclusion criteria included previous ankle, knee, hip or back injuries and neurological conditions affecting motor function. This study was approved by the University of Western Australia Ethics Review Board.

Electromyographic (EMG) data were recorded from the rectus femoris (RF), vastus lateralis (VL), vastus medialis (VM), biceps femoris (BF) and semimembranosus (SM) using a tripolar configuration and an inter-electrode distance of 25 mm.

Strength was measured using a Biodex Isokinetic dynamometer with the knee and hip flexed to 90° . After familiarization subjects performed three maximum isometric trials of the quadriceps and hamstring muscle groups, each lasting approximately 4 seconds followed by a 2 minute rest period. Verbal encouragement and visual feedback was provided. The strongest measured trial was used for analysis.

QMI was measured with the use of a burst superimposition technique in which a supramaximal burst of electrical stimulation was superimposed on a MVIE trial. QMI values were calculated using central activation ratio (CAR), the ratio of volitional force and peak force with stimulus. All data were sampled simultaneously at 2000Hz.

Differences between patients and controls for each variable were examined using unpaired t-tests, significance was set at $p < 0.01$.

RESULTS

Normalised (for body mass) knee extension strength of the OA group was significantly lower $1.50 \pm 0.62 \text{ Nm.kg}^{-1}$ than the control group $1.90 \pm 0.48 \text{ Nm.kg}^{-1}$ ($p = 0.005$).

The mean CAR values were 0.942 ± 0.041 and 0.952 ± 0.033 for the OA and control groups respectively and no significant difference existed between the groups ($p = 0.267$).

The co-contraction ratio of the quadriceps to hamstrings was 0.78 ± 0.12 for OA and 0.74 ± 0.17 for controls and was also not significantly different ($p = 0.205$).

DISCUSSION

These results demonstrate reduced MVIE strength in the absence of excessive ACC or QMI in OA patients compared with age matched controls.

Knee OA patients in this study had 22% less quadriceps strength compared with age matched controls. These findings are in agreement with previous finding of 24% strength reductions in OA patients compared to controls (Lewek et al, 2004).

The average voluntary quadriceps activation of healthy older controls in MVIE has been reported at 93% (Newham et al, 2001 &

Lewek et al, 2004). Both OA patients and controls in the current study had 94% activation, signifying healthy activation levels.

ACC of the hamstrings during MVIE trials has been reported between 12- 20% in young healthy subjects (Kubo et al, 2004) and up to 40% in healthy older women (Macaluso et al, 2002). ACC did not differ between the controls and patients in this study with values of 26% and 22% respectively, and were well within the normal values for healthy adults.

SUMMARY

The reductions in quadriceps strength seen in knee OA patients are not due to QMI or ACC, leaving disuse atrophy as a likely cause for quadriceps weakness in knee OA patients.

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