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Title:

Effects of Football Simulated Fatigue on Neuromuscular Function and Whole-body Response to Disturbances in Balance

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Abstract

The effect of football specific fatigue on explosive neuromuscular performance and dynamic balance has received little attention in the literature despite the potential consequences for injury risk. This study aimed to investigate the effect of fatigue induced by simulated football match-play on maximal and explosive knee flexor (KF) and knee extensor (KE) torque, and thus the maximal and explosive KF/KE ratio, as well as the effect of fatigue induced by simulated football match-play on whole-body response to disturbances in balance. Fifteen male team sports players (mean ± SD: age 24.2±4.2 years; stature 1.79±0.09 m; body mass, 77.3±10.7 kg) underwent ~90 minutes of the modified Loughborough Intermittent Shuttle Test (LIST; fatiguing exercise condition) or seated rest (control condition) on separate days. Maximal and explosive isometric KF and KE voluntary torque (MVT/ EVT) were assessed pre and post condition. Maximal and explosive KF/KE ratios were calculated. Centre of mass (COM) response (displacement) to unexpected anterior and posterior platform perturbations were also assessed pre and post condition. Football simulated fatigue resulted in reduced KF (15%) and KE (12%) MVT ($p \leq 0.002$) but was not found to reduce EVT of either muscle group, or explosive KF/KE ratio. Football simulated fatigue resulted in impaired balance response (11% increase in COM displacement) to unexpected perturbation in the posterior ($p=0.002$) but not the anterior direction. Impaired response to dynamic disturbances in balance, rather than explosive torque or changes in muscle balance (H/Q ratios) may be a contributory factor towards increased injury risk in the latter portion of football games, and likely highlights the influence of fatigue on sensory/proprioceptive processes.

Keywords: Explosive strength, maximal strength, dynamic balance, injury risk.

Introduction

Football (soccer) participation includes a significant risk of musculoskeletal injury, including hamstring strain injuries (HSI), which are one of the most common injuries in football[1] and anterior cruciate ligament (ACL) injuries, which are also highly prevalent.[2] Considering the magnitude of economic and societal costs of sports injuries,[3] including the increased risk of osteoarthritis following an ACL injury,[4] identifying contributors to these injury mechanisms to assist in prevention strategies remains imperative. The majority of football injuries occur in the last 15 minutes of each half,[1,5] strongly implicating fatigue as a causative factor in sports injury. However, the influence of fatigue on neuromuscular function and joint stabilisation has not been fully elucidated.

The ratio of knee flexors to knee extensors strength, commonly referred to as the hamstrings-to-quadriceps ratio (H/Q ratio), has been proposed to influence the capacity for dynamic joint stabilization[6], influencing both ACL risk[7] and HSI risk.[8] Previous literature has demonstrated that fatigue can reduce the maximal H/Q ratio.[9,10] However, achieving maximal isometric torque can take >300 ms[11] and sporting injuries, such as ACL injuries, occur very rapidly (<50 ms from initial contact[12]). Thus, due to the limited time available in many injurious situations it is possible that explosive torque, defined as the ability to increase torque as quickly as possible from a low or resting level, rather than maximal torque, is particularly important for joint stabilisation and injury prevention. Furthermore, we previously found that explosive torque was more sensitive to fatigue than maximum torque during an isometric protocol,[13] perhaps due to greater decline in function of the type II fibres that are particularly important for a high rate of force development.[14]

Greater awareness of the likely importance of explosive torque has led to research on the explosive H/Q ratio,[15] with anecdotal evidence that a lower explosive H/Q ratio may predict ACL injury risk.[16] However, the effect of football specific fatigue on explosive H/Q ratio has received little attention. One preliminary study reported reduced explosive knee extension and flexion torque post football fatigue, but no

difference in the explosive H/Q ratio.[17] Therefore, further research is required to investigate the effects of football specific fatigue on explosive H/Q ratio.

An increased risk of injuries in the latter portions of a football game may also be explained by the effect of fatigue on the balance response to perturbations.[1,5] Specifically, fatigue may impair the dynamic postural control system and thus the ability to produce an appropriate muscular response to disturbances of balance, resulting in inferior dynamic joint stability.[18,19] Following football-specific activity, fatigue related reductions in balance performance have previously been found,[18,20] but only in relatively static tasks that do not replicate the unexpected, dynamic perturbations than can lead to sporting injuries.[12] Consequently, due to the paucity of research on the effect of football induced fatigue on the response to unexpected dynamic perturbations, further, more rigorous investigation is warranted. Furthermore, previous studies investigating the effect of football-specific fatigue on strength and balance responses has failed to utilise a control condition[9,17,18,20,21] and thus did not adequately isolate the effect of football induced fatigue.

Therefore, the current study aimed to investigate the effect of fatigue following simulated football activity on maximal and explosive hamstrings and quadriceps torque, and thus the maximal and explosive H/Q ratios, as well as the effect of football simulated fatigue on the response to dynamic disturbances in balance. It was hypothesised that fatigue induced by football match-play would reduce maximal and explosive hamstrings and quadriceps torque, with a greater reduction in explosive than maximal torque, and that fatigue would result in impaired balance response to unexpected dynamic disturbances in balance.

Methods

Participants

Fifteen healthy, active males (mean ± SD: age, 24.2 ± 4.2 years; stature, 1.79 ± 0.09 m; body mass, 77.3 ± 10.7 kg; estimated VO_2max, 51.2 ± 5.1 ml·kg·min^{-1}) provided written informed consent prior to their participation in the study, which was approved by the Loughborough University Ethical Advisory Committee. Participants had a BMI of ≤ 25 kg.m^{-2}, no history of traumatic lower limb injury (ACL rupture, fracture etc.) or

current musculoskeletal conditions. Body mass and stature were measured using a calibrated scale and stadiometer (Seca, Hamburg, Germany). All participants regularly participated in team sports (minimum twice per week) such as football and/or rugby. Participants were advised not to undertake any unaccustomed/strenuous physical activity for 36 hours prior to their laboratory visit and to arrive in a relaxed, fed and hydrated state.

Experimental Design

The study was a randomised, controlled, crossover design. Participants visited the laboratory on three occasions, separated by 5-10 days: a familiarisation session and two experimental sessions, consisting of an exercise and control condition in a randomised order. All sessions commenced between 8:00 am and 4:00 pm and at a consistent time for each participant. The familiarisation session involved participants completing a series of isometric muscle function tests of the knee flexors (KF) and knee extensors (KE) on two separate custom built isometric dynamometers; followed by familiarisation with dynamic disturbances in balance and finally, the multi-stage fitness test (MSFT[22]) to determine individual walking, jogging and cruising speeds for the exercise intervention. The exercise condition comprised a modified version of the Loughborough Intermittent Shuttle Test (LIST; see below), consisting of Part A up to 75 minutes, Part B from 75 minutes onwards involved three exhaustive bouts of exercise. Up to 750 ml of water could be consumed ad libitum. In the control condition, the LIST was replaced with a 90-minute period of seated rest. Both conditions (experimental sessions) involved identical pre and post intervention measurements, and with measurement order matched for both interventions, and measurement timings matched as closely as possible based on pilot work for the expected duration of the exhaustive bouts in Part B of the LIST. The pre measurements were: unanticipated balance perturbations (lasting 1.5 min), followed by KE and KF muscle function tests (randomly ordered with minimal rest between tests) both involving maximal and explosive voluntary contractions. Post testing occurred at equivalent times during the latter stages of both conditions (exercise and control). Specifically, during the exercise intervention perturbation testing was immediately after Part B exhaustive bout 1 (commencing at 78.5 $\pm$ 1.7 min), muscle function testing was after Part B exhaustive bout 2 and 3 (KE / KF in a randomised order matched to pre and

control, first muscle group test: commenced at 80.9 ± 1.1 min, second muscle group test: 83.5 ± 1.3 min). Isometric dynamometers were located by the side of the running track and the CAREN system (computer assisted rehabilitation environment, Motek Medical, Amsterdam, Netherlands) (for perturbation testing) was in an adjacent room. This ensured minimal transition time and recovery between exercise and testing (< 1 min from cessation of the exhaustive bouts and commencement of all tests, which then lasted ~1.5 min).

Modified LIST

Participants were fitted with a heart rate monitor and instructed to complete a self-selected 5-minute warm-up that would replicate their normal sporting behaviour prior to commencing the LIST protocol. The LIST is a shuttle run protocol (Figure 1) where participants are required to run between two lines, 20 m apart, at varying speeds dictated by an audio signal and based on speeds corresponding to their estimated VO_{2max}. The LIST was designed to replicate the activity patterns and the physiological demands of a football game in order to provide a state of fatigue that was comparable to that at the end of a football match.[23] The LIST has been found to be a valid and reliable fatigue protocol that closely replicates the activity profile, metabolic, physiological and neuromuscular demands of a football game.[23,24] The exercise periods involved: 3 x 20m walking, 1 x 20m maximal sprint, 4 s recovery, 3 x 20 m at a running speed equivalent to 55% VO_{2max} (jog), 3 x 20 m at a running speed corresponding to 95% VO_{2max} (cruise).[23,24] This pattern is replicated for each 15-minute block followed by a 1 minute recovery period. This pattern is described as Part A during of the LIST protocol. Maximum rate of oxygen consumption (VO_2max) was estimated from individual MSFT scores to calculate the jogging and cruising speeds, using an equation developed by Ramsbottom et al.[22] The different intensities of shuttles were dictated by a pre-recorded, modifiable audio track based on these results. In order to replicate a football match, a 15-minute half time rest period was provided following the third part A block. Part A continued for the first 75 minutes of exercise, before commencing part B: fatigue protocols 1, 2 and 3. Each part B fatigue protocol was designed to bring the participants to voluntary exhaustion in ~5 minutes. Part B was included to ensure the upper limits of football-specific fatigue were reached and in order to facilitate measurements after each three repeats of Part B. Part B

consisted of: 1 x 20m maximal sprint, 4 s recovery, 3 x 20 m at a running speed equivalent to 55% VO_{2max} (jog), 6 x 20 m at a running speed corresponding to 95% VO_{2max} (cruise) until participants could not keep time with audio signal or until voluntary exhaustion. Upon completion of the first part B block (fatigue protocol 1), balance perturbation testing was completed, following completion of the second part B block (fatigue protocol 2) one of the muscle function tests was completed and the remaining muscle function test was repeated following the final part B block (fatigue protocol 3). The intention was that all of the muscle function tests were completed close to 90 minutes, depending on each individual's time to exhaustion, to replicate the duration of a football match.

Average heart rate (HR) was recorded for each block using the heart rate monitor display (Polar T31; Polar Electro, Kempele, Finland). Sprint times were recorded using timing gates (Brower Timing Systems, Utah, USA) and average sprint times were calculated for each block. Rating of perceived exertion (RPE) was also taken following each block.

Muscle Function Measurements

Knee Extension: Participants were secured in a customised dynamometer with hip and knee joint angles of 140º and 150º respectively (where 180º is full extension). A knee angle of 150º was selected as the majority of ACL injuries occur at this knee angle.[12] Adjustable strapping across the pelvis and shoulders prevented extraneous movement. A strap, 40 mm width of reinforced canvas webbing, was placed proximal to the ankle (15% of tibial length above medial malleolus) and was in series with a calibrated S-beam strain gauge (Force Logic, Swallowfield, UK). The analogue force signal from the strain gauge was amplified (x370) and sampled at 2000 Hz using an external A/D converter (Micro 1401; CED, Cambridge, UK) interfaced with a personal computer running Spike 2 software (CED Ltd., Cambridge, UK). In offline analysis, force data were low-pass filtered at 500 Hz using a fourth-order zero-lag Butterworth filter, gravity corrected by subtracting baseline force, and multiplied by lever length (distance from ankle strap centre to knee joint space) to calculate torque.

Knee Flexion: Participants were secured in a prone position on a custom-built isometric dynamometer with a hip and knee joint angle of 140º and 150º, respectively.

Adjustable strapping was placed across the back, pelvis and knee in order to prevent extraneous movement. The distal tibia was placed in a metal brace and an ankle strap was securely fastened 2 cm proximal to the lateral malleolus. The brace was in series with an identical strain gauge as used for knee extension testing and positioned perpendicular to the tibia. The force signal was sampled in the same manner as the knee extension force with identical hardware, software and processing techniques utilised as for the knee extensors.

Electromyography (EMG)

Surface EMG signals were recorded from the superficial knee extensors (rectus femoris (RF), vastus medialis (VM) and vastus lateralis (VL)) and knee flexors (biceps femoris (BF) and semitendinosus (ST)) using a wireless EMG system (Delsys Trigno System, Boston, MA, USA). After preparation of the skin by shaving, light abrasion and cleaning with 70% ethanol, single differential Trigno Standard surface EMG sensors (Delsys Inc., Boston, MA) were attached over each muscle, parallel to the presumed orientation of the muscle fibres, and at standardised percentages of thigh length (measured from the knee joint space to the greater trochanter) above the superior border of the patella (VM 30%, VL 50%, RF 60%) or from the popliteal fossa (BF and ST 45%). The EMG signal was amplified at source (x 300; 20- to 450- Hz bandwidth) before further amplification (overall effective gain, x909) and sampled at 2000 Hz using the same A/D converter and PC software as the force data. For offline analysis, EMG was band pass filtered at 6-500Hz using a fourth-order zero-lag Butterworth filter, and corrected for the 48 ms delay inherent to the Trigno EMG system analogue output.

Protocol

Maximum Voluntary Isometric Contractions: Participants performed a series of warm-up contractions at ~50% and ~75% of perceived maximum voluntary torque (MVT). The warm-up protocol was repeated following the control session, but warm-up contractions were not required following the experimental condition. Following the warm-up, participants performed three maximum voluntary contractions (MVCs) each lasting 3s with >30 s rest between them. Participants were instructed to contract as hard as possible and motivated with a standardised script of strong verbal

encouragement and real time visual biofeedback of the force response during and after each contraction. MVT was defined as the greatest torque during any of the MVCs or explosive contractions (see below). Force values (N) were multiplied by individual lever lengths (m) to calculate maximal voluntary torque (MVT) values in N.m for the knee extensors (KE) MVT and knee flexors (KF) MVT. The H/Q MVT ratio, expressed as a percentage (%), was then calculated by dividing KF MVT by KE MVT and multiplying by 100. Maximal EMG amplitude was calculated for KE and KF by calculating the root mean square (RMS) EMG for a 500 ms epoch at MVT (250 ms either side), for each electrode site before averaging across the three quadriceps sites for quadriceps EMG at MVT ($QEMG_{MVT}$) and averaged across the two hamstrings sites for hamstrings EMG at MVT ($HEMG_{MVT}$).

Explosive Voluntary Isometric Contractions: Following conclusion of MVCs, participants completed 5 explosive contractions, each separated by 15s rest. Repetitions were kept as low as possible to minimize recovery time during the fatigue protocol. Participants were instructed to contract ‘as fast and as hard as possible’ for 1 s with the emphasis on ‘fast’. To provide biofeedback on their explosive performance the slope of the force time curve was displayed throughout the contractions, with the peak slope (maximal rate of force development, calculated over a 10 ms epoch) of their best attempt highlighted with an onscreen cursor. Contractions associated with obvious pretension or a counter movement were discarded, and another attempt made. A visual marker on screen depicted 80% of MVC during the explosive contractions and participants were instructed to achieve at least this level of force in order to ensure a forceful contraction. The explosive contractions were performed until 5 contractions met these criteria. RMS EMG of recording sensor was measured over three time windows: 0-50, 0-100 and 0-150 from EMG onset of the first agonist muscle to be activated before averaging all the recording from the quadriceps ($QEMG_{0-50}$, $QEMG_{0-100}$, $QEMG_{0-150}$) and hamstrings ($HEMG_{0-50}$, $HEMG_{0-100}$, $HEMG_{0-150}$), respectively.

During offline analysis, the three most suitable explosive contractions (no pretension, no countermovement, highest torque production) were chosen for analysis and all torque and EMG onsets were identified manually through visual identification using a systematic approach considered to have superior validity to automated methods.[11]

Torque and EMG signals were viewed on an *x* axis scale of 300 ms prior to the contraction and *y* axis scales of 0.68 Nm-0.05mV before zooming in to determine the final peak or trough before the signal deflected away from the envelope of the baseline noise.[11] Explosive torque of the knee flexors and extensors was measured at 50, 100 and 150 ms from EMG onset. Explosive H/Q ratio was calculated for each time point, using the same calculation as the H/Q MVT ratio. Calculating the explosive torque from EMG onset was chosen to include the inherent electromechanical delay (EMD), an important element of explosive neuromuscular performance.[15] EMD was determined as the time difference between EMG onset and force onset, with the longest time difference (EMD_{max}) being identified for the quadriceps ($QEMD_{max}$) and hamstrings ($HEMD_{max}$) and expressed in ms. Final values for all torque and EMG measurements were calculated by averaging values from the three selected contractions.

Perturbation Response Testing

All perturbation trials were completed on a CAREN system, which contains a large computer controlled mechanical platform that can independently perturb the support surfaces in each of the six degrees of freedom (Figure 2A). It has been found to be reliable for use in postural and balance research.[25] Prior to the perturbations fifty-seven spherical markers of 14 mm diameter were fixed to the subject (Figure 2B) to facilitate collection of kinematic data at 200 Hz from 15 body segments using nine T20 Vicon (Vicon, Oxford Metrics Group, UK) cameras.

The centre of mass (COM) was defined relative to the centre of the perturbing platform, which was defined as the mean position of four markers located at the corners of the force plates. Participants were instructed to stand upright on one leg on the CAREN platform with their hands by their side with the non-standing foot in a standardised position, which had the forefoot lightly touching the ankle of the standing leg, asked to try to remain stationary and not to take a step in response to the perturbation. Trials involved six perturbations in a randomised order; three posterior and three anterior. The perturbation commenced at random after participants had verbally confirmed they were stable in their standing position. The perturbations were controlled by a custom script written in the Motek Medical D-Flow software. Platform perturbations had a magnitude of 0.1 m and a maximum velocity of 0.35 $m.s^{-1}$. Motion analysis data were

analysed off-line. Data were low pass filtered with a fourth-order zero-lag Butterworth filter using a cut off frequency of 15 Hz. Initiation of perturbation was defined as when the velocity of platform markers exceeded 0.005 $m.s^{-1}$. Centre of mass (COM) displacement (over 5 ms time periods) values were obtained from the initiation of perturbation to 500 ms post perturbation using Visual3D software (C-motion, Germantown, MD, USA) with values at 100 ms intervals used for further analysis.

Statistical Analysis

Data are presented as mean ± SD. For all indices measured pre and post in both conditions (measures of maximal and explosive torque and EMG) the interaction effects of condition and time were analysed using a two-way repeated measures ANOVA (condition (control vs exercise) x time (pre vs post)). Within groups comparisons were investigated using a paired sample *t*-test. Indices measured only during the exercise condition (sprint times, HR and RPE) were analysed using a one-way repeated measures ANOVA, with a post-hoc Bonferroni correction to adjust for multiple pairwise comparisons. All statistical procedures were performed with IBM SPSS Statistics for Windows (Version 22.0, NY, USA, IBM Corp.) and statistical significance was accepted at $p < 0.05$.

Results

Physiological Variables during the Exercise Condition

Sprint performance, HR and RPE all changed during the exercise condition (ANOVA, $p \leq 0.002$). Mean sprint performance during the three exhaustive bouts (Part B) was slower and RPE was higher than all the prior 15-minute blocks (Part A) (ANOVA, $p \leq 0.005$; Table 1). HR was higher during the first exhaustive bout (Part B) than the prior 15-minute blocks (Part A) (ANOVA, $p \leq 0.02$; Table 1). No differences were found for any of the physiological variables between the exhaustive bouts of Part B (ANOVA, $p \geq 0.082$).

Maximum Voluntary Torque of the KE and KF

QMVT decreased following exercise (-12%; paired *t*-test $p = 0.002$; Figure 3A), but not control (-2%; paired *t*-test $p = 0.21$; Figure 3A), and consequently there was a condition x time interaction effect (ANOVA, $p = 0.017$). $QEMG_{MVT}$ also decreased following

exercise (-15%; paired *t*-test $p = 0.005$; Figure 3B) but not control (+2%; paired *t*-test $p = 0.63$, Figure 3B) and showed a condition x time interaction effect (ANOVA, $p = 0.004$).

HMVT decreased after exercise (-15%; paired *t*-test $p = 0.001$; Figure 3C), but not control (+1%, paired *t*-test $p = 0.80$; Figure 3C) and exhibited a condition x time interaction effect (ANOVA, $p = 0.013$). $HEMG_{MVT}$ decreased following both exercise (-25%; paired *t*-test $p = 0.004$; Figure 3D) and control (-16%; paired *t*-test $p < 0.001$; Figure 3D), without showing a condition x time interaction effect (ANOVA, $p = 0.178$). No within or between group differences were found for H/Q MVT ratio (condition x time interaction ANOVA, $p = 0.256$; Figure 3E).

Explosive Voluntary Torque of the KE and KF

There were no changes in KE or KF explosive torque for any time point during the explosive contractions for either exercise or control interventions (paired *t*-test $p \geq 0.101$), and consequently no condition x time interaction effects (ANOVA: KE $p \geq 0.578$, Figure 4A; KF, ANOVA $p \geq 0.062$, Figure 4B). Similarly, there was no condition x time interaction effect for explosive $QEMG_{0\text{-}50}$ and $QEMG_{50\text{-}100}$ (ANOVA, $p \geq 0.908$; Figure 4C). However, there was a small reduction in $QEMG_{100\text{-}150}$ following control (-8%; paired *t*-test $p = 0.001$) but not following exercise (paired *t*-test $p = 0.733$), and subsequently a condition x time interaction effect (ANOVA, $p = 0.024$). No condition x time interaction effects were found for explosive HEMG for any time point (ANOVA, $p \geq 0.072$). However, $HEMG_{50\text{-}100}$ (-12%; paired *t*-test P = 0.030; Figure 4D), and $HEMG_{100\text{-}150}$ (-18%; paired *t*-test $p = 0.010$; Figure 4D) decreased following exercise, but $HEMG_{0\text{-}50}$ did not change (-0%, paired *t*-test $p = 0.63$; Figure 4D). No differences were found in explosive HEMG following the control condition for any period ($p \geq 0.19$; Figure 4E).

Explosive H/Q ratio showed no condition x time interaction effect after 50 or 100 ms of contraction (ANOVA, $p \geq 0.515$; Figure 4E). However, consequent to a non-significant decrease following exercise (-6%, *t*-test $p = 0.269$) and an increase following control that approached significance (+6%, *t*-test $p = 0.053$) a condition x time interaction effect was found for explosive H/Q ratio at 150 ms (ANOVA, $p =$

0.029). No changes were found for quadriceps EMD (QEMD; ANOVA, p = 0.515; Control, pre: 19.8 ± 3.6 ms vs post: 21.1 ± 4.9 ms; Exercise, pre: 19.0 ± 5.9 ms vs post: 22.1 ± 6.3 ms) or hamstrings EMD (HEMD; ANOVA, p = 0.873; Control, pre: 29.7 ± 10.4 ms vs post: 27.8 ± 7.3 ms; Exercise, pre: 29.1 ± 8.3 ms vs post: 26.9 ± 6.1 ms).

R*esponse to Unexpected Mechanical Perturbations*

In response to unexpected anterior perturbations no differences were found for centre of mass displacement (COMD) relative to the base of support after the exercise or control condition at any time point ($p \geq 0.108$; Figure 5A), and consequently there were no condition x time interaction effects (ANOVA, $p \geq 0.085$). In response to unexpected posterior perturbations, COMD at the end of perturbation (500 ms) increased following exercise (+11%, paired t-test p = 0.04; Figure 5B) and decreased following control (-6%, paired t-test $p < 0.001$; Figure 5B), and consequently there was a condition x time interaction effect (ANOVA, p = 0.002) indicating less stability after exercise. No differences were found in COMD in response to unexpected posterior perturbations after the exercise or control condition for other time points (ANOVA, $p > 0.05$; Figure 5B).

Discussion

This study investigated the effect of fatigue induced by simulated football match-play on maximal and explosive KF and KE torque, and thus maximal and explosive H/Q ratio, as well as the response to whole-body disturbances in balance. In agreement with our hypothesis, there were modest reductions in maximal KF and KE torque following exercise, and the balance response to unexpected posterior perturbations was also impaired. Contrastingly, explosive KF and KE torque were not reduced following exercise, and there were no changes in the maximal or explosive H/Q ratio.

Following exercise, KE and KF MVT decreased by 12% and 15% respectively. Similar decreases in MVT for the KE and KF have been reported in the literature following simulated football fatigue protocols.[21,26,27] Larger decreases have been found in both muscle groups after localised exhaustive isokinetic (KE -23.5%, KF -24.4%[28]) and

isometric exercise (KE -42%[13]), which might be expected with localised activity involving a smaller muscle mass and more immediate post-exercise measurements due to no transition time between running track/pitch and dynamometer. Contrary to our hypothesis, no changes in KE or KF explosive torque occurred following the exercise condition at any time point during contraction. These findings are in contrast to previous research which found decreased KE and KF explosive torque following football specific fatigue[17,26] and following isokinetic[28] and isometric exercise.[13] Considering football simulated fatigue, one study involved treadmill running consisting of no changes of direction[17] and two studies measured EVT and MVT during the same contraction,[17,26] which may result in suboptimal measurements of both variables.[29] The differing fatigue protocols and different assessment methods may have contributed to these conflicting findings. The current study employed the LIST protocol that has been shown to closely replicate the activity profile, metabolic, physiological and neuromuscular demands of a football game,[23,24] enhancing ecological validity of the current results to a football situation. Nonetheless, our study deliberately employed the more strenuous LIST Part B protocol (indicated by higher HR, RPE and slower sprint times during Part B compared to Part A) in the later stages of the exercise, as an attempt to generate fatigue towards the upper boundary of football fatigue and therefore it was surprising that explosive torque was not compromised.

The greater sensitivity of MVT than explosive torque to fatigue is perhaps surprising given the importance of type II fibres for a high rate of force development[14] and this was contrary to our hypothesis. It is possible that after fatiguing exercise MVCs are compromised to a greater degree because they involve sustained maximum torque contractions (~3 s) which would be expected to be more metabolically demanding than brief explosive contractions up to a sub-maximum torque (80%MVT; <1 s and with measures of explosive torque in the first 150 ms). The higher reliability of MVT (CV_W 3.3%) compared to EVT (CV_W 5.1-16.6%)[30] may in part explain the greater sensitivity of MVT to fatigue. It is also possible that any positive effects due to elevated muscle temperature[31] and potentiation[32] post-exercise might have masked any decrement in explosive torque due to fatigue, although the capacity of these mechanisms to increase explosive torque remains controversial.[33]

The MVT H/Q ratio did not change after football simulation exercise in agreement with some previous studies,[17,26] but not others that found a decrease in MVT H/Q ratio.[9,10] The studies that found a decrease utilised isokinetic measurements, which due to their dynamic nature are more susceptible to fatigue than isometric contractions.[34] Furthermore, the isometric measurements in the current study, whilst facilitating the measurement of maximum and explosive torque, are clearly not specific to the dynamic nature of football and this may have limited the relevance of our testing procedure. Additionally, no differences were found for explosive H/Q ratio after the exercise condition for any time point during contraction. The only previous football simulated fatigue study also found no alteration in explosive isometric H/Q ratio following fatigue.[17]

The lack of decrease in KE or KF explosive torque, or explosive H/Q ratio, suggests that explosive strength variables are not part of the explanation or mechanism for the increased occurrence of injury in the latter periods of football games.[1,5] Various other physiological or biomechanical factors have been shown to deteriorate consequent to fatigue and may contribute to increased injury risk, including increased knee valgus on landing, altered cutting technique[35,36] and reduced joint positional awareness.[37] Previous literature has found a reduction in jump height following simulated football match-play,[21,38] indicating a reduction in the ability to generate force rapidly. Isometric explosive torque measures within the first 150 ms of contraction in the current study vs more prolonged stretch-shortening cycle contractions (700-1100 ms)[39] may explain these divergent findings.

Quadriceps and hamstrings EMG_{MVT} were reduced following exercise. This substantiates previous studies that demonstrated reduced maximal quadriceps[21,27,40] and hamstrings[28,40] activation during maximum contractions post fatigue. This may, in part, be due to changes in neuromuscular transmission at the sarcolemma as a consequence of fatigue.[41] However, no differences were found for EMD or explosive EMG of either muscle group after exercise. In contrast reduced explosive QEMG and HEMG have been reported post football related fatigue.[26] Thorlund et al.'s[26] protocol involved kicking, tackling and sideways cutting which may have resulted in a more

fatiguing stimulus to the quadriceps and hamstrings than observed in the current purely running based protocol. EMG recordings in the current study were not normalised for the electrical recording conditions (e.g. evoked maximal m-wave, Mmax) in order to promptly assess function in the fatigued state. Therefore, any changes in the electrical recording conditions (e.g. perspiration or temperature) during the exercise condition may have influenced the findings. However, a recent study found no change in Mmax following simulated football match-play.[38]

Football simulated fatigue resulted in impaired balance response to unexpected perturbation in the posterior direction but not the anterior direction. The dynamic postural control system, may be impaired during posterior perturbations producing a limited muscular response following football related fatigue, potentially resulting in inferior protective function for dynamic joint stability.[18,19] Previously football fatigue has been found to reduce balance performance during relatively static tasks,[18,20] but not been examined during dynamic perturbations. Consequently, the current findings show for the first time that football induced fatigue impairs dynamic balance during posterior perturbations. Changes following fatigue only occurred for COMD following posterior displacement and only at the end of the perturbation. No changes were observed for any earlier time point, suggesting that these changes might not be applicable to a sporting environment where injuries appear to occur in a much more rapid time period (<50 ms[12]). Posterior platform perturbations result in an anterior displacement of the COM relative to the base of support, requiring corrective response from the posterior musculature, including the hamstrings.[42] More distal musculature, such as the soleus, gastrocnemius or tibialis anterior may have larger effects on these postural responses to perturbation.[43] Future research should include neuromuscular assessment of these distal muscle groups following football as these muscle groups have been largely ignored in previous similar studies.

This study improved on methodological limitations of previous similar research by including a control condition and minimising potential recovery time between the exercise condition and post testing (< 1 minute to starting the test and only 1.5 minutes

duration). Furthermore, individuals in the current study were tested at a knee angle applicable to sporting injury (150º)[12] increasing the application of findings to a potential sports injury scenario. However, the constraint of minimising recovery time precluded EMG normalisation, and using controlled isometric conditions, important for robust explosive torque measurements, may have limited the ecological validity (e.g. dynamic contractions) of the findings.

In conclusion, football specific fatigue resulted in modest reductions in MVT of the KE and KF, but no change in H/Q MVT ratio. Whilst there was increased COM displacement following posterior perturbations, this was not the case for anterior perturbations, potentially contributing towards reduced joint stability post fatigue. However, explosive performance of both the KF and KE remained unchanged implying that alterations in explosive neuromuscular performance may not be a contributory factor towards the increased injury risk in the latter portion of football games. These results appear to highlight the role of sensory and proprioceptive aspects of movement, inherent in balance response, as potential mechanisms for the increase in injury risk towards the end of match-play rather than explosive motor performance.

Clinical Perspective

Impaired response to unexpected disturbances in balance, rather than explosive torque or changes in muscle balance (H/Q ratios) may be a contributory factor towards increased injury risk in the latter portion of football games. This likely highlights the influence of fatigue on sensory/proprioceptive processes, inherent in dynamic sporting balance performance. This could inform clinicians to improve proprioceptive processes in a fatigued state for injury prevention.

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Table 1. Physiological variables during the exercise condition (LIST protocol). Data are mean ± SD, n = 15. *****Significant difference to time periods 15-75 mins ($P < 0.05$)

Exercise Block	Sprint Times (s)	RPE	HR (b.min^{-1})
0-15 mins (Part A)	2.53 ± 0.10	14 ± 2	161 ± 13
15-30 mins (Part A)	2.56 ± 0.11	15 ± 2	167 ± 11
30-45 mins (Part A)	2.58 ± 0.10	16 ± 2	167 ± 11
45-60 mins (Part A)	2.59 ± 0.12	14 ± 2	160 ± 12
60-75 mins (Part A)	2.58 ± 0.11	16 ± 2	164 ± 12
Fatigue 1 (Part B)	2.93 ± 0.19*****	19 ± 2*****	176 ± 8*****
Fatigue 2 (Part B)	2.99 ± 0.21*****	18 ± 2*****	171 ± 9
Fatigue 3 (Part B)	2.98 ± 0.30*****	19 ± 1*****	172 ± 9

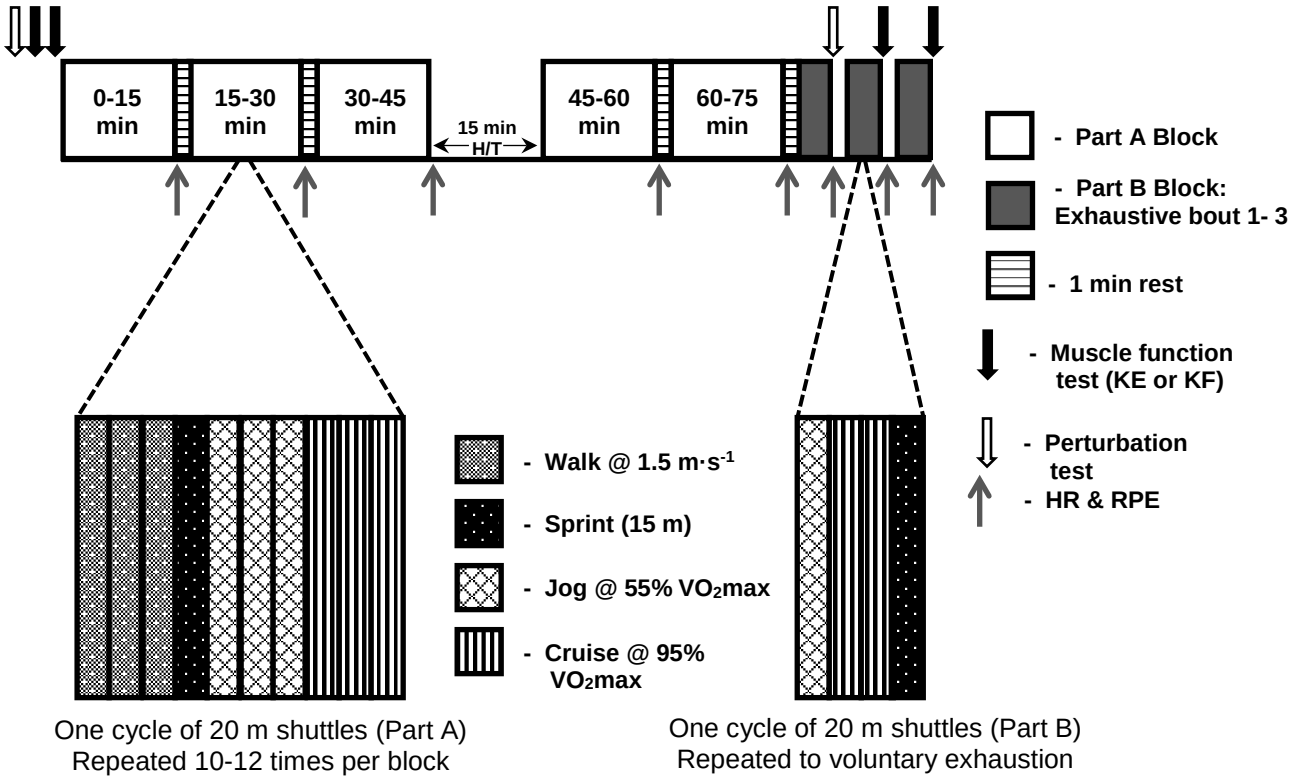

Figure 1. The modified LIST protocol.

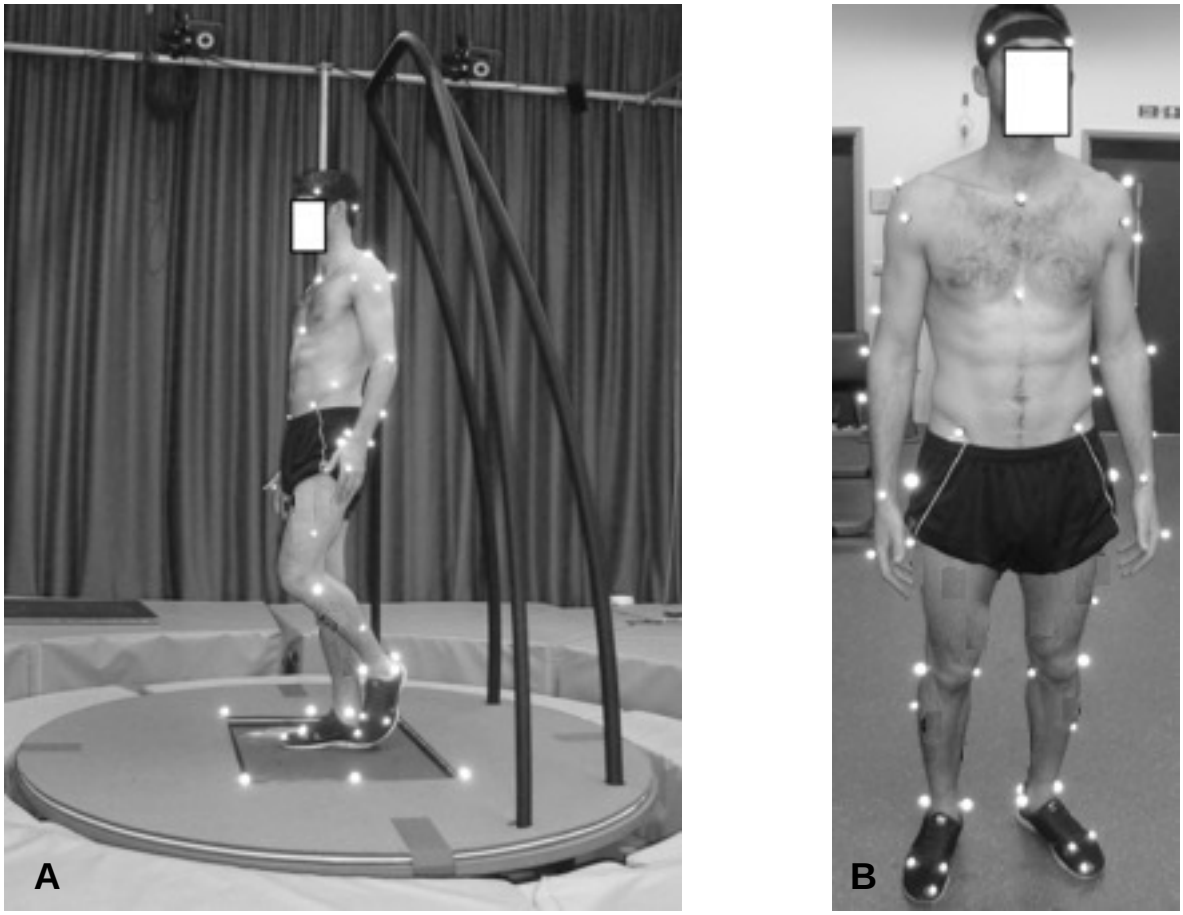

Figure 2. Perturbation testing on the CAREN machine (**A**) employing a fifty- seven marker set (**B**).

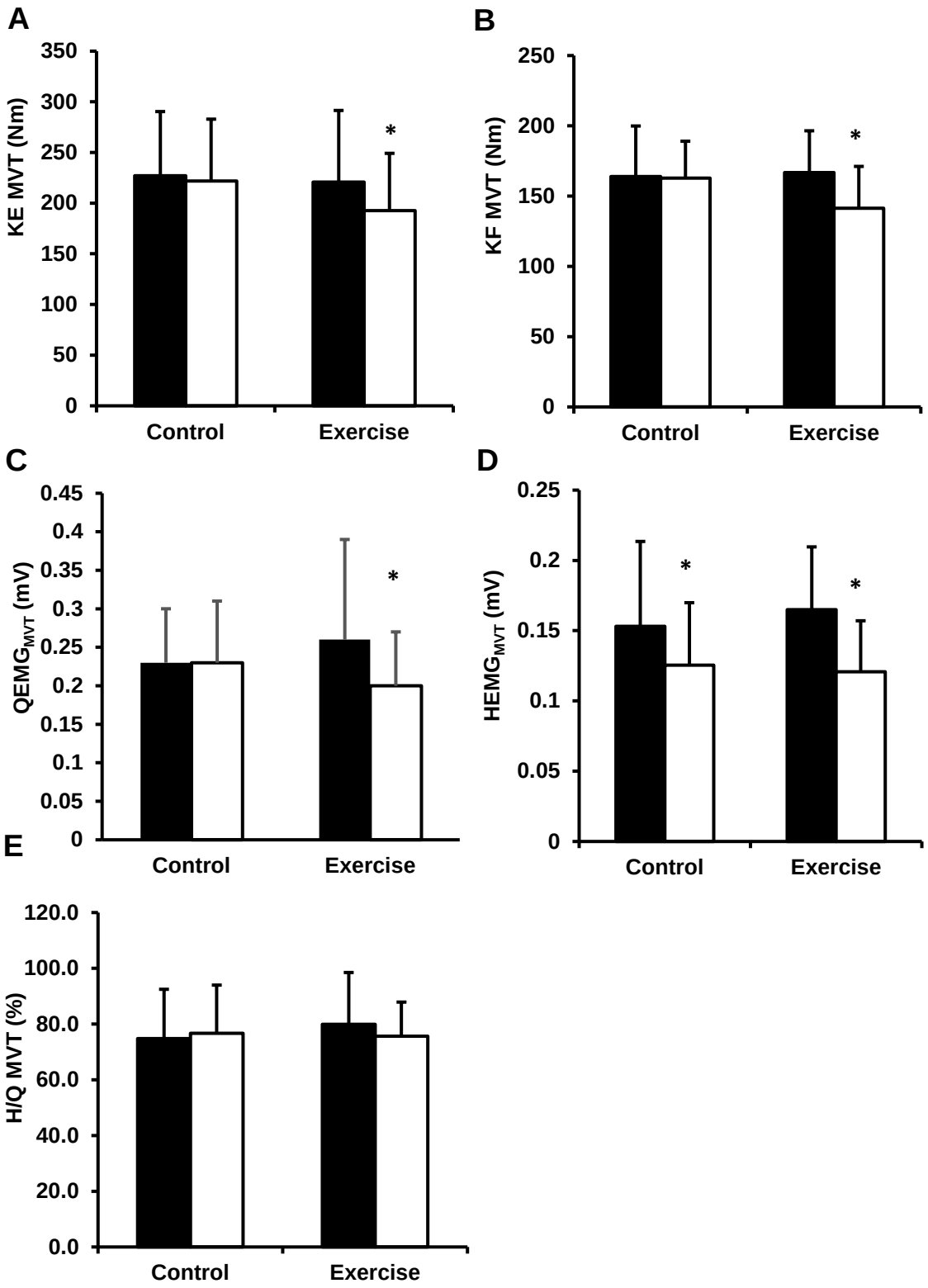

Figure 3. Knee extension (KE) maximum voluntary torque (MVT) (**A**), knee flexion (KF) MVT (**B**) and the H/Q MVT ratio (**E**). Quadriceps EMG ($QEMG_{MVT}$) (**C**) and hamstrings EMG ($HEMG_{MVT}$) (**D**) amplitude at MVT are also shown. Pre (black bars) and post (white bars) for control and exercise conditions. Data are mean ± SD (n = 15). Significant differences from pre to post condition indicated by *, $P < 0.05$

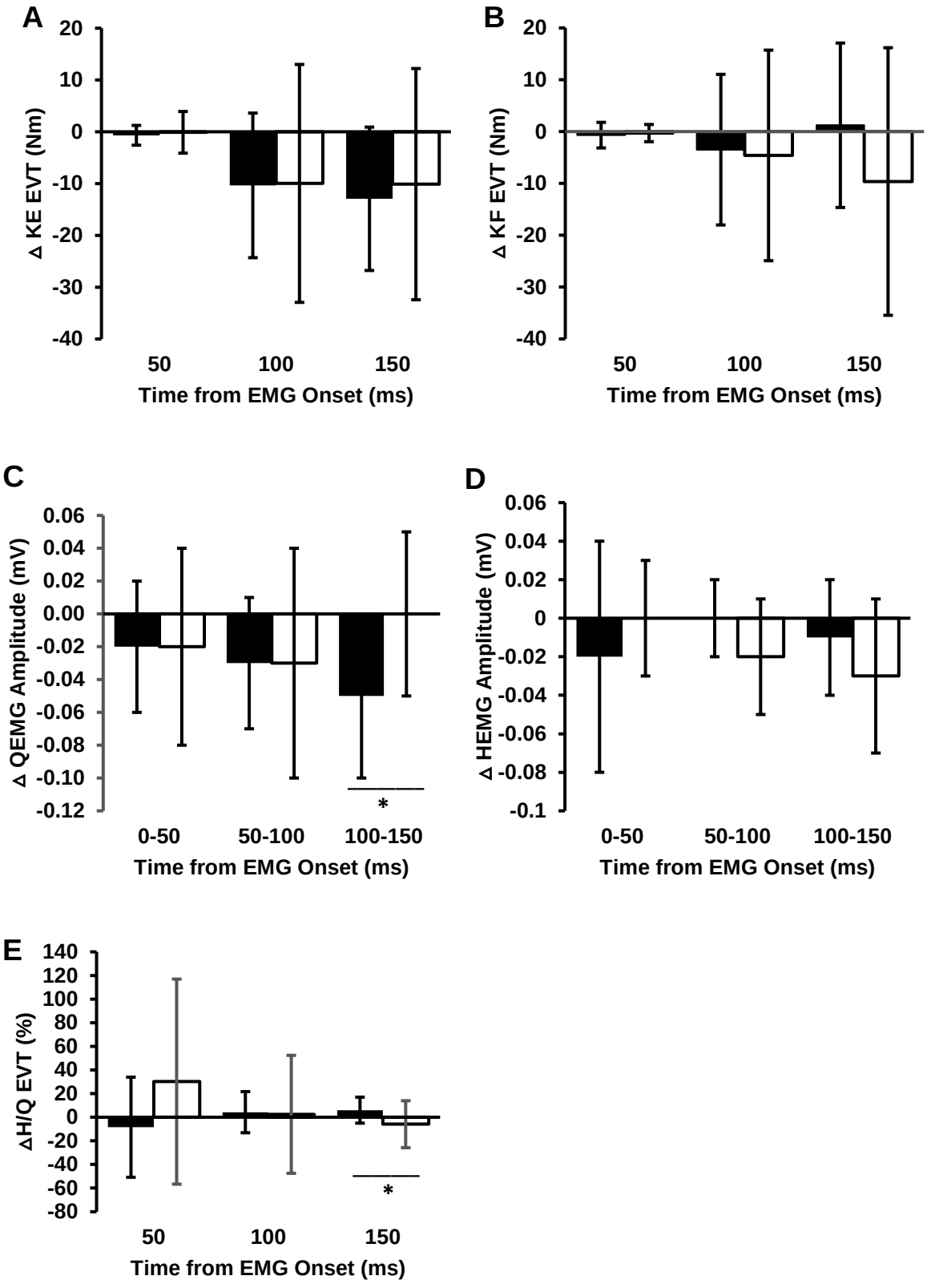

Figure 4. Changes in explosive KE (**A**) and KF (**B**) torque, explosive H/Q ratio (**C**), explosive quadriceps EMG (**D**) and explosive hamstrings EMG (**E**) during explosive voluntary contractions. Data are mean values ± SD (n = 15). Control: black bars, Exercise: white bars. Significant interaction effects indicated by *, $P < 0.05$.

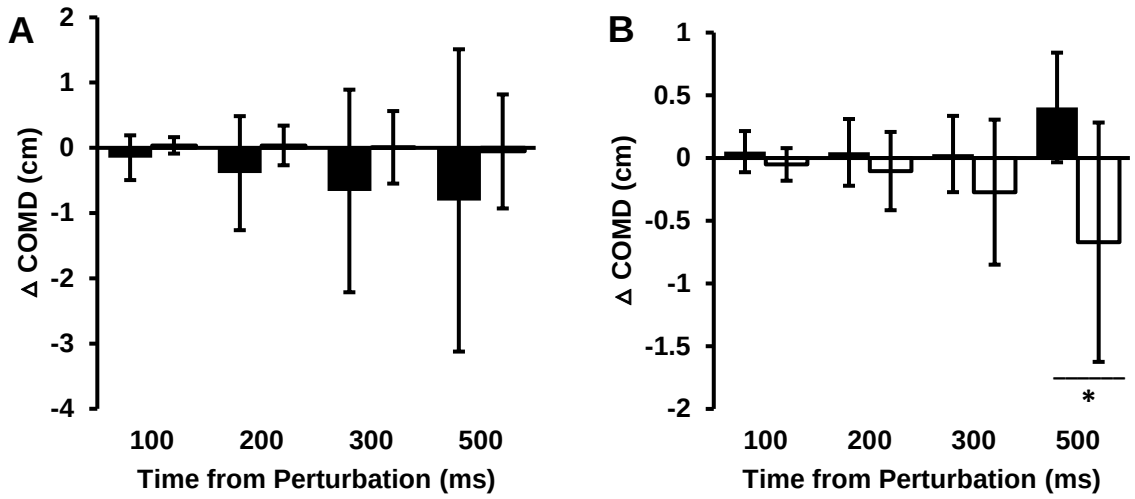

Figure 5. Changes in Centre of mass (COM) displacement following anterior (**A**) and posterior (**B**) perturbations. Data are mean values ± SD (n = 15). Control: black bars, Exercise: white bars. Significant interaction effects indicated by *, $p < 0.05$.