European Journal of Sport Sciences
Vol 2 | Issue 6 | December 2023
ISSN 2796-0048

RESEARCH ARTICLE

Do Between-limb Strength Differences in the Elbow Flexors Affect Their Neuromuscular and Strength Adaptations to Short-term Strength Training?

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ABSTRACT

***Purpose*: The present study quantified between-limb responses to strength training in the stronger (STR) versus weaker (WEA) elbow flexors in participants with symmetric (SYM) or asymmetric (ASY) 1-RM strength.**

***Methods*: Neural, hypertrophic, and strength adaptations to 4 weeks ($3 \cdot wk^{-1}$) of unilateral elbow flexion training of both arms were examined in 24 participants (6 men and 6 women in each group) who had not undertaken strength training in the past 12 months. Changes in one-repetition maximum load (1-RM strength), isokinetic ($20°\cdot s^{-1}$ and $210°\cdot s^{-1}$), and isometric (MVIC) strength and rate of force development (RFD), as well as muscle activation (EMG; normalised to maximum M-wave amplitude) and size (CSA_{Flexor}), were measured. Transcranial magnetic stimulation was used to assess motor-evoked potential amplitude (MEP) and cortical silent period duration (cSP).**

***Results*: Following training, significant increases in 1-RM strength (SYM, STR: $\Delta = 2.4 \pm 1.1$ kg, WEA: $\Delta = 1.9 \pm 1.1$ kg; ASY, STR: $\Delta = 1.8 \pm 0.7$ kg, WEA: $\Delta = 1.7 \pm 0.7$ kg) and CSA_{Flexor} (SYM, STR: $\Delta = 107 \pm 98$ mm^2, WEA: $\Delta = 121 \pm 64$ mm^2; ASY, STR: $\Delta = 108 \pm 73$ mm^2, WEA: $\Delta = 105 \pm 65$ mm^2) were observed, although they were not different between arms in either group. Increases in isokinetic strength were also detected, but only in STR (SYM: $20°\cdot s^{-1}$, $210°\cdot s^{-1}$; ASY: $20°\cdot s^{-1}$). No statistical changes were detected in MVIC, RFD, EMG, MEP, or cSP. Correlation analyses demonstrated both similarities and differences in the between-limb responses when comparing the groups.**

***Conclusion*: Between-limb responses to strength training in participants with and without strength symmetry were similar, although isokinetic strength increases were only observed in the stronger arms. However, the correlation results suggest that individual differences exist between the groups, signalling that between-limb responses can differ in individuals with and without strength asymmetry.**

Keywords: Between-limb, elbow flexors, muscle size, strength.

Submitted: November 08, 2023

Published: December 29, 2023

10.24018/ejsport.2023.2.6.117

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1. Introduction

Muscular strength improvements in response to strength training result from both neural and muscular adaptations (Moritani & deVries, 1979; Narici *et al*., 1989). Although these responses are likely triggered simultaneously at the commencement of a strength training programme, neural adaptations such as improvements in motor unit recruitment and rate coding are considered to predominate during the initial stages of strength gain (Del Vecchio *et al*., 2019), while hypertrophic adaptations are likely to be more important after several weeks of training in previously untrained muscles (Moritani & deVries, 1979; Narici *et al*., 1996). While these responses are traditionally

considered to occur in untrained individuals, muscles that have already encountered loading and thus have already undergone hypertrophy and increased activation might have less reserve for change (Häkkinen *et al*., 1987). Although it is common to classify individuals as being more trained than others for these reasons, an interesting consideration is that the stronger limb, which likely participates in high-force activities more often than the weaker limb, might be considered to be more trained in individuals who have not undertaken a deliberate exercise programme. As studies sometimes describe responses to strength training after applying different interventions to each upper or lower limb or using the contralateral limb as a control (Jones & Rutherford, 1987; Seger *et al*., 1998), it is important to understand the training-induced responses in lateralised muscles (same muscles on opposite sides of the body) to ensure appropriate conclusions are drawn.

Most studies investigating lateralised responses have defined limbs based on their dominance using handedness inventories (Philpott *et al*., 2015; Tan, 1989). Whilst this classification is reasonable, it might be more appropriate to define limbs based on their strength (i.e., stronger vs weaker) when investigating strength training-induced responses. Although it is easy to assume that the preferential use of limb results in the dominant limb being stronger than the non-dominant limb, strength differences may also result from differential specialisation factors (Sainburg, 2005; Tan, 1989), which may predispose individuals to be stronger in a particular limb. For instance, the dominant limb appears specialised in performing tasks requiring trajectory and torque control, whereas the non-dominant limb appears specialised in performing tasks requiring positional control (Healey *et al*., 1986). As a consequence, each limb may have unique abilities that enable them to be more effective in performing certain tasks (Healey *et al*., 1986). Therefore, when addressing the training-induced changes, if baseline strength differences are caused by incidental activities, it could be expected that the stronger limb will have less scope for further neuromuscular change due to a *ceiling effect*. However, if baseline strength differences are caused by differential specialisation factors, both limbs may generate specialised adaptative responses, with the stronger and weaker limbs possibly displaying greater improvements in dynamic and static strength, respectively. As a consequence, the between-limb training-induced changes might provide insight into the mechanisms that influence the baseline strength differences. In saying that, baseline strength differences may have little impact on neuromuscular adaptations, and thus, both limbs respond to the same extent following strength training.

Surprisingly, few studies have compared the changes between limbs after each has been trained unilaterally using the same relative loads. While some data exist comparing the changes in muscle strength and size in lower-limb muscles (Baroni *et al*., 2016), including in older women (≥60 years old; Nunes *et al*., 2022), it is not clear whether the same outcomes are observed in upper-limb muscles. Lower limbs often simultaneously participate in many activities of daily living, such as standing from a chair, climbing stairs, and walking and running, which may reduce between-limb differences (Abdelmohsen, 2019). By contrast, with the exception of bilateral load carriage, many activities in the upper limbs, such as holding an object, grasping, twisting, and throwing, are often performed with the preferred limb (Oldfield, 1971). Thus, a greater limb disparity may be detected in upper-body muscles (Stoll *et al*., 2000). If so, asymmetric training responses may be more likely in upper-body muscles than lower-body muscles. Interestingly, Moritani and deVries (1979) observed that hypertrophy predominantly contributed to strength changes later in the weaker (6 weeks) than stronger (two weeks) elbow flexors following isoinertial strength training, possibly indicating that strength increases in the weaker arm were associated with neural adaptations for a longer period of the training. Recently, Carvalho and Barroso (2020) reported that baseline 1-RM strength and muscle thickness were the same between the elbow flexors, and that they adapted similarly to the same relative isoinertial training stimulus. However, this study did not investigate the impact of baseline strength differences nor examine early phase neural adaptations such as changes in the motor evoked potential (MEP) (Griffin & Cafarelli, 2007), cortical silent period (cSP) (Latella *et al*., 2012) or other force-generating characteristics.

Given the above, the purpose of the present study was to quantify the adaptive responses in both the stronger and weaker elbow flexors in individuals with symmetric (SYM) or asymmetric (ASY) 1-RM strength following a short period (four weeks) of unilateral strength training of both arms matched for relative load. It was hypothesised that increases in MEP and electromyogram (EMG) amplitudes and a decrease in cSP duration would be greater in the weaker than the stronger elbow flexors in ASY, and that greater strength increases would also be detected in the weaker arm. For SYM, it was hypothesised that similar strength adaptations would be detected in both arms.

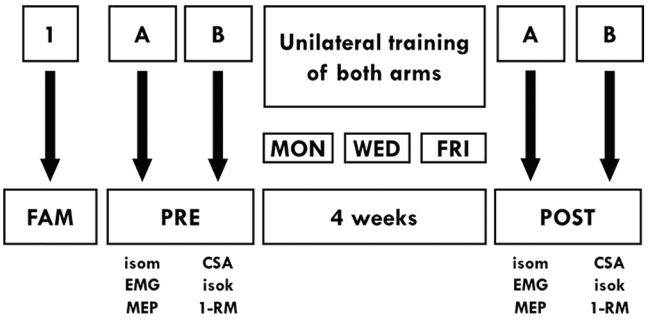

Fig. 1. Experimental approach. FAM: familiarisation; PRE: pre-training; POST: post-training; isom: isometric contraction; EMG: electromyography; MEP: motor evoked potential; CSA: muscle cross-sectional area; isok: isokinetic contraction; 1-RM: 1-repetition maximum.

2. Methods

2.1. Participants

Twenty-four participants (males: n = 12, age: 23 ± 6, height: 1.7 ± 0.5 m, body mass: 70.9 ± 10.9 kg; females: n = 12, age: 25 ± 6 y, height: 1.7 ± 1.0 m, body mass: 72.4 ± 16.7 kg) who had not undertaken strength training for at least 12 months prior to the study volunteered for the present study. This particular cohort was recruited to improve the likelihood of detecting both neural and hypertrophic adaptations. Participants had not experienced any upper body musculoskeletal injuries within the last three years, were not taking any anti-inflammatory, anabolic, or anti-depressant medications, and were not identified as being at-risk according to transcranial magnetic stimulation (TMS) safety checklist (Rossi *et al*., 2011; Rossini *et al*., 2015). Participants were required to abstain from any high-intensity upper-body activities and were instructed to maintain their current diet for the duration of the study. Additionally, all participants were instructed not to drink caffeinated or alcoholic drinks for 6 and 12 hours before each testing session, respectively. The University Human Research Ethics Committee approved this study, and all participants provided written informed consent before entering the study (#16826).

2.2. Experimental Approach

Each participant attended 17 sessions throughout the 6-week study: one familiarisation, four testing and 12 training sessions. During the first visit, the participants were extensively familiarised with the isometric, isokinetic, and isoinertial strength, as well as TMS testing procedures. The pre-training testing sessions (PRE) were conducted on the second and third visits, separated by at least two days. Isometric strength, EMG amplitudes during isometric contractions, and responses to TMS were examined in the first testing session (PRE_A), while muscle cross-sectional area (CSA) and both isokinetic and isoinertial strength were assessed in the second testing session (PRE_B). Participants then completed four weeks of unilateral free-weight elbow flexor training on both arms 3 times a week. The length of the training period was selected in order to compare the early phase adaptative responses between arms. The post-training testing sessions (POST) were completed over the following week at least three ($POST_A$) and six ($POST_B$) days after the cessation of training and at least two days apart. Fig. 1 shows the experimental approach to the study.

2.3. Experimental Procedures

2.3.1. Pre- and Post-training Testing Sessions A

Within sessions PRE_A and $POST_A$, isometric strength and muscle activation (via EMG) and then M-wave and TMS testing were conducted with the same set-up for one arm and then repeated for the other arm. The testing order for each arm was randomised at PRE and then repeated at POST. First, each participant was seated on an isokinetic dynamometer (Biodex System 4 Pro, Shirley, NY) with the elbow (left or right) supported at 90° and aligned with the axis of rotation of the lever arm. Rather than the participant holding onto the handle of the elbow flexion attachment, the force was produced against the handle at the wrist with the forearm supinated (secured to the end of the handle with an inelastic cord) to minimise the effect of grip-associated muscle activity on TMS-based measurement outcomes (Hasegaw *et al*., 2001; Hess *et al*., 1987). The dynamometer, chair, and handle configurations were noted during the familiarisation session and standardised for both PRE_A and $POST_A$ sessions. Surface electrodes (Ag–AgCl) were placed over the mid-belly of the biceps brachii and its distal tendon, with the reference electrode placed on the humeral lateral epicondyle (Blue Sensor N-00-S, 28 mm^2, Ambu, Ballerup, Denmark). When assessing maximal voluntary isometric contraction (MVIC) torque and rate of force development (RFD), the participant was instructed to pull upwards on the handle of

the dynamometer *"as fast and then as hard as possible"* without pretension (Van Cutsem & Duchateau, 2005; Viitasalo, 1982) or countermovement (Kamimura *et al*., 2009). Trials were discarded if observable EMG or a countermovement (negative force deflection) greater than ~0.5 N was detected (Blazevich *et al*., 2009; Tillin *et al*., 2010). Testing consisted of three 3-s MVICs with 1 min of passive rest between trials. However, if the peak torque of the third trial was >5% greater than the earlier trials, more MVIC attempts (no more than three) were provided until there was a plateau or drop in force output. This procedure ensured that maximum force capacity was expressed. The participant then performed three additional trials with the instruction to contract *"as fast as possible,"* although their peak torque had to reach at least 80% of their MVIC torque. A horizontal guideline on a large television screen in front of the participant and a buzzer activated when torque reached the 80% MVIC torque level (LabChart software function, ADInstruments, New South Wales, Australia) provided real-time feedback to the participant.

Following the isometric strength tests, cathode and anode electrodes (White Sensor 4560M, 79 mm^2, Ambu, Ballerup, Denmark) were placed over Erb's point in the supraclavicular fossa and the acromion process, respectively. To obtain the maximal M-wave amplitude (M_{max}), electrical stimuli of 200-μs duration were delivered using a constant-current stimulator (DS7AH, Digitimer, Welwyn Garden City, UK) to Erb's point to evoke resting M-waves in the biceps brachii. Stimulation intensity was increased until there was no further increase in M-wave amplitude. Stimulation intensity was set 20% above this intensity, and the mean amplitude (mV) from two stimuli delivered ~8 s apart was used in analyses.

Finally, corticospinal responses were assessed by applying TMS to the upper limb motor areas of the contralateral cerebral hemisphere using a Magstim 200^2 stimulator (Magstim Co, Dyfed, UK) and a 90-mm figure-of-8 coil held tangential to the skull to induce a posterior-anterior current. For consistency of coil placement, the participant wore a tight-fitting cap with a latitude-longitude matrix positioned about the nasion-inion and interaural lines. After locating the hotspot of the biceps brachii, active motor threshold (AMT) was determined with the participant contracting to a target of 5% of peak muscle activity (EMG_{peak}) (root mean squared [RMS] EMG signal was provided as visual feedback). AMT was defined as the minimum stimulator output intensity (SO) that generated a motor evoked potential (MEP) that was 2 SD above the background EMG and was determined using maximum-likelihood parameter estimation by sequential testing (PEST) (Awiszus & Borckardt, 2011). Finally, 10 single-pulse stimuli were delivered approximately every 10 s during 5% EMG_{peak} contraction at block-randomised stimulator intensities of 120% and 150% of AMT.

2.3.2. *Pre- and Post-training Testing Sessions B*

In sessions PRE_B and $POST_B$, CSA and both elbow flexor isokinetic and isoinertial strength were assessed in both arms. First, whole upper arm CSA was measured at a single location using peripheral quantitative computed tomography (pQCT; XCT-3000; Stratec Medizintechnik, Pforzheim, Germany). Details of the procedures and equipment have been reported previously (Rowe *et al*., 2018). Briefly, the participant rested supine with a single arm placed in an abducted position through the pQCT gantry. A customised mount enabled an acrylic holder to support the participant's arm. A scout view scan was then performed slightly distal to the radial head, and a reference line was positioned at the proximal endplate of the radial head. A single-slice CT scan was performed at 33% of the humeral segment length. The segment length was estimated as 0.186 × standing height (measured at PRE). Quality assurance scans were performed using the manufacturer's standard phantom each morning before testing and with the cone phantom every 30 d.

Following the CSA assessment, the participant was seated on the isokinetic dynamometer (as described above) to assess maximal unilateral isokinetic elbow flexion strength in both arms. Strength was assessed at angular velocities of 210°·s^{-1} and then 20°·s^{-1} through a 90° range of motion starting with the elbow slightly bent (~10°: vertical lever arm = 0°). The limb test order was randomised at PRE and then repeated at POST. On this occasion, the participant held the elbow attachment handle without the elbow support (described above) and positioned their elbow underneath their shoulder while lightly contacting the chair backrest. The lateral epicondyle was aligned to the rotational axis of the lever arm; a laser pointer was used to ensure alignment. To allow the lever arm, handle attachment, and participant's arm to move alongside the chair without obstruction, the dynamometer head was rotated by 5°. The resultant set-up was recorded during the familiarisation and subsequently used in PRE_B and $POST_B$. Three single concentric elbow flexion trials separated by 1 min of passive rest for each velocity were completed with the highest peak torque (Nm) for each velocity used for data analysis.

Finally, the maximal unilateral isoinertial elbow flexion strength of both arms was assessed using a one-repetition maximum (1-RM) test. The limb test order was randomised at PRE and repeated at POST. The participant was seated at an adjustable preacher curl bench (Sorinex, South Carolina,

Fig. 2. The elbow flexion testing and training technique with the involved arm resting on the adjustable preacher curl bench and the uninvolved arm behind the participant's back.

USA) with the bench angled at 10° (vertical = 0°). Fig. 2 depicts the adjustable preacher curl bench and the technique used during 1-RM strength testing (and training).

The test arm rested on the bench, holding a dumbbell, and the non-test arm was positioned behind the back. Starting from elbow flexion, the participant lowered the dumbbell to near-maximal elbow extension before returning to the starting position without making detectible body compensatory movements. Two minutes after each successful lift, a heavier load was attempted until a repetition could not be completed. The 1-RM load was determined to be the nearest 0.5 kg. The arm that lifted the heaviest load was recorded as 'stronger'; if there was no difference between arms for 1-RM strength, the participant was asked which arm they would use to throw a ball as far as possible, and the selected arm was noted as 'stronger'.

2.4. Data Analysis

During isometric strength testing, torque and EMG signals (BioAmp EMG system; PowerLab, ADInstruments, New South Wales, Australia) were recorded using a laptop computer running LabChart software (version 8.1.9, ADInstruments, New South Wales, Australia) using a 16-bit analogue-to-digital converter sampling at 2,000 Hz (PowerLab 16/35, ADInstruments, New South Wales, Australia). The torque signals were smoothed with an 18-Hz low-pass filter (linear-phase finite impulse response [FIR] filter), whereas the EMG signals were filtered with a 20-500 Hz band-pass FIR filter (gain = 1,000, transition width = 4 Hz, input impedance = 200 MΩ, common-mode rejection ratio ≥ 85 dB at 1–60 Hz). The MVIC trial that generated the highest peak torque (Nm) and the trial, either MVIC or 80% of MVIC, that generated the greatest RFD ($Nm \cdot s^{-1}$) (from torque onset to 50 ms) were used in subsequent analyses. MVIC torque was accepted as the highest reading of the filtered torque-time record, while RFD was calculated as the slope of the filtered torque-time curve from torque onset to 50 ms (RFD_{50}) as well as from 100 ms to 200 ms ($RFD_{100–200}$) after torque onset. RFD was also normalised to each participant's MVIC torque (i.e., $RFD_{50/MVIC}$, $RFD_{100–200/MVIC}$). Torque onset was determined manually as the inflection before an increase in torque of >0.5 Nm (Tillin *et al.*, 2010).

To quantify muscle activation, the RMS from EMG onset to both 40 ms (EMG_{40}) and 100 ms (EMG_{100}) were calculated from the best RFD trial, while EMG_{peak} was determined as the RMS within a 500-ms window before peak torque from the best MVIC trial; all values were then normalised to M_{max} ($\%M_{max}$). EMG onset was determined manually by selecting a small section (~500 ms) around the noticeable change in the rectified EMG signal, zooming until the y-axis scale was ~0.1 mV, and then a marker identifying the onset was positioned at the earliest detectable change in the EMG signal (Tillin *et al.*, 2010). For TMS and M_{max} testing, the EMG signals were also sampled at 2,000 Hz; however, no other filtering was imposed (gain = 1,000 and 500, respectively). The average MEP peak-to-peak amplitude ($\%M_{max}$) at each stimulus intensity (120% and 150% of AMT) was calculated. cSP was then quantified as the time (or duration) from stimulus artefact to the reoccurrence of an ongoing EMG signal (Damron *et al.*, 2008; Kimberley *et al.*, 2009). That is, the end of the cSP was identified as

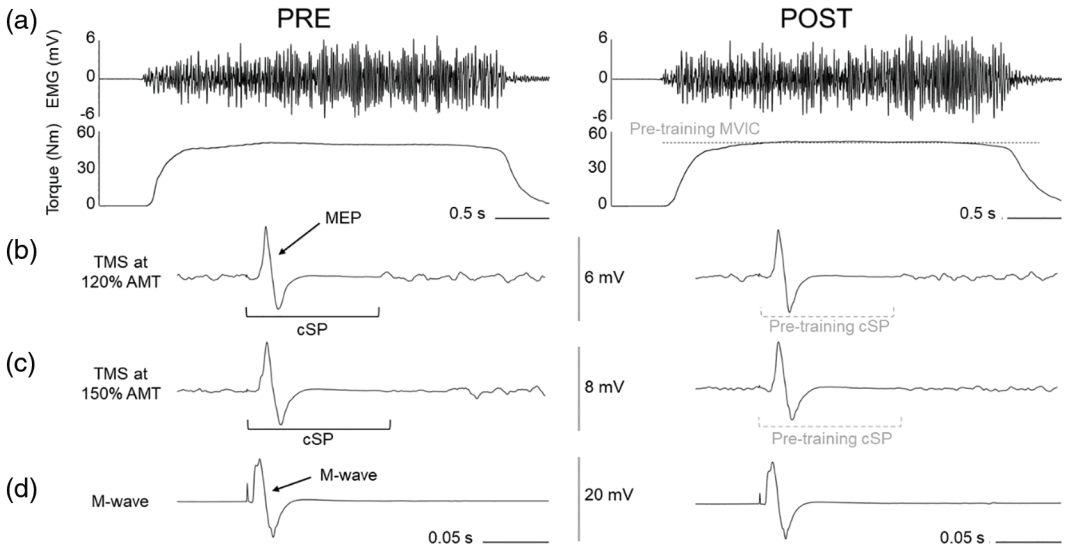

Fig. 3. Representative traces from one participant, showing the changes in elbow flexor torque and volitional muscle activity (EMG) (a), MEP amplitudes and cSP duration (b & c), and M-wave amplitude (d) with training.

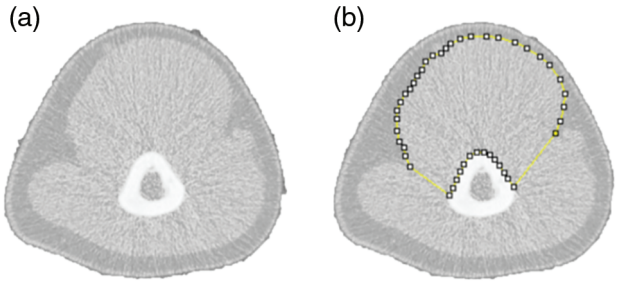

Fig. 4. The same pQCT image of the upper arm. a) Whole-limb image; b) Image with ImageJ outline demonstrating the technique used to isolate the elbow flexor muscle group to determine CSA_{Flexor}.

the time when the rectified EMG signal returned to 50% of pre-stimulus EMG (calculated from 100 ms before stimulation). The cSP at each stimulus intensity was quantified as the average time (s). The pre-stimulus EMG (RMS) 100 ms before TMS was also reported (%M_{max}). Fig. 3 shows a representative example of one participant's isometric strength (torque) and other test results following training.

Finally, all pQCT images were generated with an installed macro in the Stratec software that used edge detection and threshold techniques to differentiate the tissues (Rowe *et al*., 2018; Schiferl, 2017). To ensure that CSA was determined from pQCT images only for the trained elbow flexors without the inclusion of the untrained elbow extensors, elbow flexor CSA (CSA_{Flexor}) (mm^2) was calculated by outlining the muscle group with the polygon tool in ImageJ software (National Institutes of Health, Maryland, USA) (Schneider *et al*., 2012). However, it is possible a small section of the outline included the elbow extensors as the boundary between muscle groups is difficult to observe in pQCT images. Due to the location of the single-slice CT scan, the predominant muscles captured within the outline were the biceps brachii and brachialis. Example images are provided in Fig. 4.

2.5. Strength Training

The strength training protocol consisted of four weeks of unilateral elbow flexion training of both arms 3 times a week (Fig. 1). The participant completed two warm-up sets of unilateral dumbbell preacher curls on each upper arm consisting of 15 and 12 repetitions at 50% and 60% of their PRE 1-RM, respectively, before training using 3 sets of 8–12 repetitions with a load equivalent to 75% of PRE 1-RM. A 2-minute passive rest was given between sets. The load was continually adjusted to ensure that the participant was able to complete the final lift within the proposed repetition range (i.e., training was performed to failure in each set). A metronome was used to provide timing to the participant, with 3 s and 2 s devoted to eccentric and concentric phases, respectively.

2.6. Statistical Analysis

The normality of data was confirmed using Shapiro-Wilk testing and quantile-quantile (Q-Q) plots and transformed (rank-based inverse transformation) in the event of non-normality (Templeton, 2011). Data parameters that required transformation were 1-RM, isokinetic ($210°{\cdot}s^{-1}$ and $20°{\cdot}s^{-1}$), MVIC, RFD, EMG, and MEP amplitudes. Three-way (arm [stronger vs weaker], time [PRE vs

TABLE I: The Loads Lifted (kg) and Repetitions Completed During Strength Training of the Stronger and Weaker Arms in the Symmetry and Asymmetry Strength Groups

		Mean load per set (kg)			Repetitions per set (n)		
		Sessions 1–4	Sessions 5–8	Sessions 9–12	Set 1	Set 2	Set 3
Symmetric strength	Stronger arm	8.5 (3.4)	9.3 (3.3)	10.0 (3.4)	9.9 (0.9)	8.6 (0.6)	8.6 (0.7)
	Weaker arm	8.4 (3.3)	9.2 (3.3)	9.8 (3.4)	9.7 (0.9)	8.4 (0.6)	8.3 (0.7)
Asymmetric strength	Stronger arm	7.9 (2.8)	8.8 (2.7)	9.5 (2.8)	9.6 (1.1)	8.2 (0.7)	7.9 (0.6)
	Weaker arm	7.0 (2.4)	7.9 (2.5)	8.6 (2.6)	9.3 (1.0)	8.1 (0.6)	8.0 (0.4)

Note: The numbers in the table refer to the mean (SD) values.

POST], and strength symmetry-asymmetry [symmetric vs asymmetric]) repeated measures factorial MANOVAs (RFD, early EMG amplitude, and pre-stimulus EMG amplitude) and ANOVAs (all other dependent variables [as well as training loads]) were used to compare the responses to strength training. Participants were assigned to those with symmetric (SYM) or asymmetric (ASY) 1-RM strength of more than 0.5 kg. Pairwise comparisons (with Holm-Bonferroni Sequential adjustment) were reported when significant main and interaction effects were detected. Between-limb effect sizes (Hedges' g) for the changes in dependent variables are reported and interpreted as trivial ($g < 0.2$), small ($0.2 \leq g < 0.5$), moderate ($0.5 \leq g < 0.8$), and large ($g \geq 0.8$). Additionally, to demonstrate the between-limb relationships in the dependent variable changes (POST–PRE), Pearson's product-moment correlations (r) with 95% confidence intervals (CI) using bias-corrected accelerated bootstrapping were computed. Significant relationships were reported when the 95% CI did not cross 0.00. The alpha level was set at 0.05 for all other analyses, and data are presented as mean ± SD. Statistical computations were performed using a statistical analysis program (SPSS, Version 25.0; Chicago, Illinois, United States).

3. Results

3.1. Training Loads of the Strength Training

A summary of the loads lifted (kg) and repetitions completed during the 4-week strength training programme is presented in Table I. In the SYM strength group, loads for the stronger arm increased from 8.5 ± 3.4 kg to 10.0 ± 3.4 kg, and for the weaker arm, increased from 8.4 ± 3.3 kg to 9.8 ± 3.4 kg. In the ASY strength group, the stronger arm loads increased from 7.9 ± 2.8 kg to 9.5 ± 2.8 kg, and for the weaker arm from 7.0 ± 2.4 kg to 8.6 ± 2.6 kg. There were significant arm ($p < 0.001$) and time ($p < 0.001$) effects as well as an arm × strength symmetry-asymmetry interaction effect ($p = 0.005$), with the stronger arm training with greater absolute loads than the weaker arm in the ASY strength group. When assessing the relative training loads lifted (%1-RM strength: PRE or POST), the stronger arm increased from 77.2 ± 2.8 % to 80.4 ± 5.5 %, while the weaker arm increased from 75.7 ± 4.1 % to 82.3 ± 3.5 % in the SYM strength group. In the ASY strength group, the relative loads for the stronger arm increased from 76.5 ± 2.3 % to 84.3 ± 4.1 %, and for the weaker arm increased from 75.7 ± 2.5 % to 85.2 ± 4.1 %. There were significant time ($p < 0.001$), time × strength symmetry-asymmetry ($p = 0.047$), and arm × time ($p = 0.036$) interactions. Post-hoc analyses determined that the relative loads lifted in SYM ($p = 0.001$) and ASY ($p < 0.001$) strength groups increased, while an increase was also detected in both the stronger ($p < 0.001$) and weaker ($p < 0.001$) arms.

3.2. 1-RM Strength

When a three-way ANOVA assessed SYM and ASY strength effects (as well as arm and time effects), significant arm ($p < 0.001$), time ($p < 0.001$) and arm × strength symmetry-asymmetry interaction effects were observed ($p < 0.001$) (Table II). Follow-up testing revealed that the stronger arm was significantly stronger than the weaker arm in the ASY strength group ($p < 0.001$). Moreover, both arms in the SYM and ASY strength groups increased 1-RM strength following the training period (SYM, STR: $\Delta = 2.4 \pm 1.1$ kg, $p < 0.001$, WEA: $\Delta = 1.9 \pm 1.1$ kg, $p < 0.001$; ASY, STR: $\Delta = 1.8 \pm 0.7$ kg, $p = 0.004$, WEA: $\Delta = 1.7 \pm 0.7$ kg, $p < 0.001$) (Fig. 5a), with small and trivial effect sizes for the between-limb changes that favoured the stronger arm (SYM: $g = 0.45$, ASY: $g = 0.14$).

3.3. Source:Isokinetic Peak Torque at 20°·s^{-1} and 210°·s^{-1}

Isokinetic peak torques (Nm) at 20°·s^{-1} and 210°·s^{-1} did not differ between arms or strength symmetry-asymmetry groups, and there were no interaction effects. However, there were significant time effects (Table II). For isokinetic peak torque at 20°·s^{-1}, significant increases were only noted for the stronger arm of each strength group (SYM: $\Delta = 5.2 \pm 3.2$ Nm, $p = 0.008$; ASY: $\Delta = 2.3 \pm 1.6$ Nm,

TABLE II: Summary of the Results

	Stronger				Weaker										
	Symmetric		Asymmetric		Symmetric		Asymmetric								
	PRE	POST	PRE	POST	PRE	POST	PRE	POST	Arm	Time	Sym	Arm × Time	Arm × Sym	Sym × Time	A × T × S
1-RM strength (kg)	10.5 (3.8)	12.9 (4.5)	10.2 (3.6)	12.0 (3.8)	10.3 (3.7)	12.2 (4.3)	8.9 (3.3)	10.6 (3.5)	**$F_{(1,22)}$ = 32.047 p < 0.001**	**$F_{(1,22)}$ = 40.787 p < 0.001**	$F_{(1,22)}$ = 0.880 p = 0.358	$F_{(1,22)}$ = 2.530 p = 0.126	**$F_{(1,22)}$ = 17.427 p < 0.001**	$F_{(1,22)}$ = 0.808 p = 0.378	$F_{(1,22)}$ = 1.989 p = 0.172
Isokinetic peak torque 20°·s^{-1} (Nm)	33.6 (12.6)	38.8 (15.4)	30.9 (12.9)	33.3 (12.9)	34.3 (13.5)	36.9 (14.8)	29.1 (11.8)	31.8 (11.6)	$F_{(1,22)}$ = 1.789 p = 0.195	**$F_{(1,22)}$ = 22.897 p < 0.001**	$F_{(1,22)}$ = 1.185 p = 0.288	$F_{(1,22)}$ = 0.957 p = 0.339	$F_{(1,22)}$ = 0.274 p = 0.606	$F_{(1,22)}$ = 0.039 p = 0.845	$F_{(1,22)}$ = 0.607 p = 0.444
Isokinetic peak torque 210°·s^{-1} (Nm)	24.7 (10.7)	29.2 (12.7)	23.8 (10.9)	25.2 (9.7)	25.4 (10.7)	27.8 (12.2)	21.9 (9.8)	23.6 (10.0)	$F_{(1,22)}$ = 1.878 p = 0.184	**$F_{(1,22)}$ = 14.143 p = 0.001**	$F_{(1,22)}$ = 1.120 p = 301	$F_{(1,22)}$ = 0.890 p = 0.356	$F_{(1,22)}$ = 2.861 p = 0.105	$F_{(1,22)}$ = 1.581 p = 0.222	$F_{(1,22)}$ = 1.961 p = 0.175
MVIC torque (Nm)	53.5 (19.8)	55.4 (17.2)	46.4 (15.9)	47.9 (16.4)	52.0 (18.5)	52.9 (15.9)	41.7 (12.6)	44.5 (13.8)	**$F_{(1,22)}$ = 7.435 p = 0.012**	$F_{(1,22)}$ = 3.662 p = 0.069	$F_{(1,22)}$ = 1.849 p = 0.188	$F_{(1,22)}$ = 0.009 p = 0.927	$F_{(1,22)}$ = 0.024 p = 0.879	$F_{(1,22)}$ = 0.003 p = 0.957	$F_{(1,22)}$ = 0.667 p = 0.423
EMG_{peak} (%M_{max})	10.9 (2.9)	12.1 (3.1)	10.9 (2.9)	10.7 (2.5)	11.5 (4.1)	11.2 (3.5)	9.2 (3.3)	9.7 (2.1)	$F_{(1,22)}$ = 1.414 p = 0.247	$F_{(1,22)}$ = 0.791 p = 0.383	$F_{(1,22)}$ = 1.295 p = 0.267	$F_{(1,22)}$ = 0.397 p = 0.535	$F_{(1,22)}$ = 1.417 p = 0.247	$F_{(1,22)}$ = 0.231 p = 0.636	**$F_{(1,22)}$ = 5.220 p = 0.032**
RFD (Nm·s^{-1})				see Table IV					$F_{(2.21)}$ = 1.338 p = 0.284	$F_{(2,21)}$ = 2.471 p = 0.109	$F_{(2,21)}$ = 0.308 p = 0.738	$F_{(2,21)}$ = 1.590 p = 0.228	$F_{(2,21)}$ = 0.210 p = 0.813	$F_{(2,21)}$ = 0.919 p = 0.414	$F_{(2,21)}$ = 0.217 p = 0.807
RFD_{MVIC} (%MVIC·s^{-1})				see Table IV					$F_{(2.21)}$ = 0.857 p = 0.439	$F_{(2,21)}$ = 0.253 p = 0.779	$F_{(2,21)}$ = 0.184 p = 0.833	$F_{(2,21)}$ = 1.693 p = 0.208	$F_{(2,21)}$ = 0.388 p = 0.683	$F_{(2,21)}$ = 0.039 p = 0.962	$F_{(2,21)}$ = 0.923 p = 0.413
EMG_{40} (%M_{max})	2.7 (1.0)	2.3 (1.4)	2.3 (2.0)	2.2 (1.3)	1.7 (1.1)	2.7 (1.9)	2.0 (1.3)	2.2 (1.1)	$F_{(2,21)}$ = 1.032 p = 0.374	$F_{(2,21)}$ = 0.727 p = 0.495	$F_{(2,21)}$ = 1.365 p = 0.277	$F_{(2,21)}$ = 3.053 p = 0.069	$F_{(2,21)}$ = 0.682 p = 0.516	$F_{(2,21)}$ = 0.965 p = 0.397	$F_{(2,21)}$ = 2.845 p = 0.081
EMG_{100} (%M_{max})	4.1 (2.0)	4.2 (1.9)	3.3 (2.0)	3.6 (1.9)	3.4 (1.9)	4.7 (2.0)	3.9 (1.8)	3.3 (1.0)							

Note: Results summary, including mean (SD) and statistical main effects, for dependent variables measured before (PRE) and after (POST) unilateral strength training of both stronger and weaker arms in the symmetric and asymmetric strength groups. A: Arm; T: Time; Sym: Symmetry; S: Symmetry.

$p = 0.033$) (Fig. 5b), with small effect sizes for the between-limb change scores (SYM: $g = 0.41$, ASY: $g = 0.39$). For isokinetic peak torque at 210°·s^{-1}, significant increases were detected only in the stronger arm of the SYM strength group (SYM: $\Delta = 4.5 \pm 4.0$ Nm, $p = 0.016$) (Fig. 5c), with small and trivial effect sizes calculated (SYM: $g = 0.41$, ASY: $g = -0.12$).

3.4. Maximal Voluntary Isometric Contraction (MVIC) Torque and Peak EMG (EMG_{peak})

Significant effects of the arm ($p = 0.012$) were observed for MVIC (Nm), but no time ($p = 0.069$), strength group ($p = 0.188$), or interaction effects were detected (Table II and Fig. 6a). Based on the follow-up testing, the stronger arm produced a greater MVIC torque than the weaker arm (Table II). There were also small effect sizes for the between-limb MVIC changes, with greater changes in the stronger and weaker arms for the SYM and ASY strength groups, respectively (SYM: $g = 0.24$, ASY: $g = -0.34$). For EMG_{peak} (%M_{max}), which was assessed in the MVIC trial, there were no arm ($p = 0.247$), time ($p = 0.383$), strength group ($p = 0.267$) or interaction effects (Table II and Fig. 6b). Moderate and small effect sizes were calculated for the between-limb EMG_{peak} changes that favoured the stronger and weaker arms in the SYM and ASY strength groups, respectively (SYM: $g = 0.63$, ASY: $g = -0.36$).

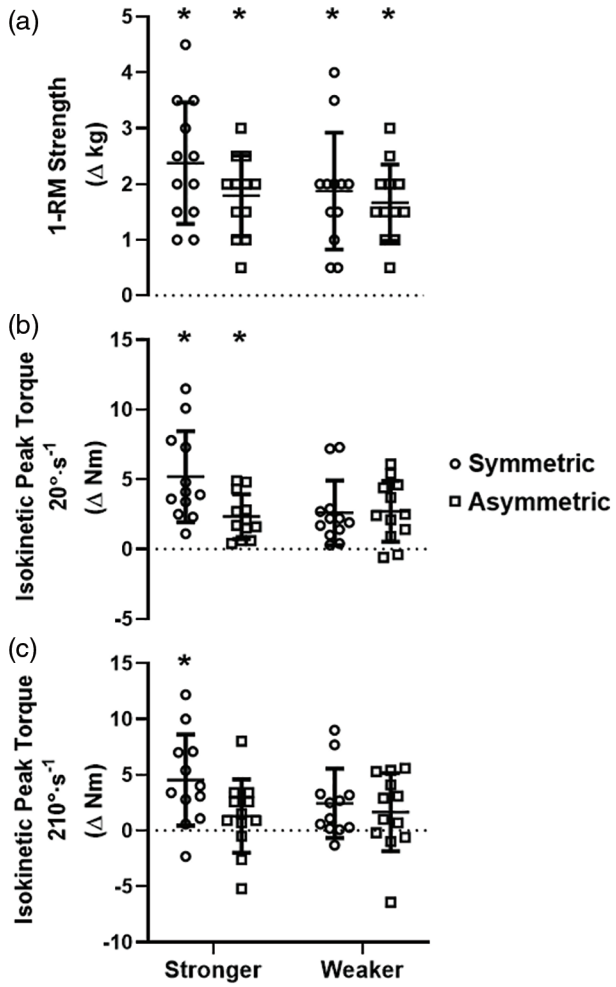

Fig. 5. Change score results for a) 1-RM strength, b) isokinetic peak torque at $20°{\cdot}s^{-1}$ and c) isokinetic peak torque at $210°{\cdot}s^{-1}$ in the stronger and weaker elbow flexors of both groups following unilateral strength training of both arms. ∗Significant change from PRE, $p < 0.05$. Horizontal lines are group means with standard deviations (SD).

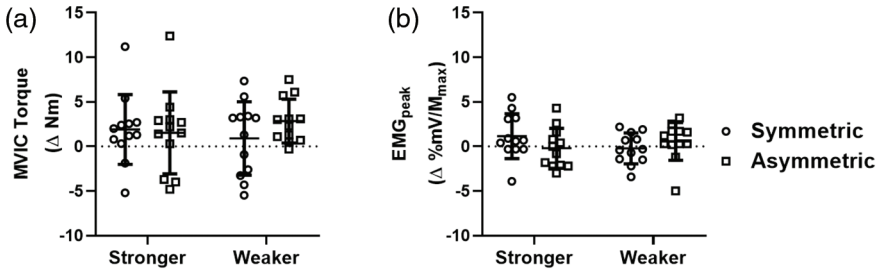

Fig. 6. The change score results for a) MVIC torque and b) EMG_{peak} in the stronger and weaker elbow flexors of both groups following unilateral strength training of both arms. Horizontal lines are group means with standard deviations (SD).

3.5. Rate of Force Development (RFD and RFD $_{MVIC}$) and Early EMG Amplitude (EMG $_{40}$ and EMG $_{100}$)

For RFD ($Nm{\cdot}s^{-1}$), no arm ($p = 0.284$), time ($p = 0.109$), strength group ($p = 0.738$) or interaction effects were detected (Table II). Similarly, no arm ($p = 0.439$), time ($p = 0.779$), strength group ($p = 0.833$) or interaction effects were observed for normalised RFD (%MVIC·s^{-1}) (see Table IV for RFD results). Trivial and small between-limb effect sizes were calculated for changes in RFD_{50} and normalised RFD_{50} (RFD_{50}: SYM: $g = -0.18$, ASY: $g = 0.37$; normalised RFD_{50}: SYM: $g = -0.21$, ASY: $g = 0.43$), while small and moderate effect sizes were calculated for the changes $RFD_{100–200}$ and normalised $RFD_{100–200}$ ($RFD_{100–200}$: SYM: $g = -0.63$, ASY: $g = -0.44$; normalised $RFD_{100–200}$: SYM: $g = -0.76$, ASY: $g = -0.52$). Assessed within the RFD trial, EMG amplitudes measured to early time points (EMG_{40} and EMG_{100}; $\%M_{max}$) did not vary between arms ($p = 0.374$) or strength groups ($p = 0.277$) and did not change following training ($p = 0.495$). No interaction effects were detected (Table II). In the SYM strength group, the magnitude of the between-limb changes in early EMG

TABLE III: Summary of the Results

	Stronger				Weaker										
	Symmetric		Asymmetric		Symmetric		Asymmetric								
	PRE	POST	PRE	POST	PRE	POST	PRE	POST	Arm	Time	Sym	Arm × Time	Arm × Sym	Sym × Time	A × T × S
AMT (SO)	39.7 (7.1)	41.6 (4.6)	40.1 (8.1)	43.2 (7.4)	42.9 (5.7)	43.5 (6.0)	43.4 (8.9)	42.6 (8.6)	$F_{(1,22)} = 3.243$ $p = 0.085$	$F_{(1,22)} = 3.303$ $p = 0.083$	$F_{(1,22)} = 0.027$ $p = 0.872$	$F_{(1,22)} = 2.805$ $p = 0.108$	$F_{(1,22)} = 0.300$ $p = 0.589$	$F_{(1,22)} = 0.004$ $p = 0.951$	$F_{(1,22)} = 0.657$ $p = 0.426$
MEP 120% AMT (%M_{max})	8.9 (4.0)	11.0 (5.3)	12.9 (9.6)	14.8 (7.6)	11.0 (5.2)	12.3 (6.0)	14.3 (7.6)	12.6 (6.0)	$F_{(1,22)} = 0.017$ $p = 0.897$	$F_{(1,22)} = 1.260$ $p = 0.274$	$F_{(1,22)} = 1.672$ $p = 0.209$	$F_{(1,22)} = 2.744$ $p = 0.112$	$F_{(1,22)} = 0.276$ $p = 0.604$	$F_{(1,22)} = 1.084$ $p = 0.309$	$F_{(1,22)} = 0.474$ $p = 0.499$
MEP 150% AMT (%M_{max})	17.2 (4.9)	20.1 (8.9)	24.2 (10.1)	26.1 (12.1)	21.2 (12.2)	21.3 (7.6)	26.7 (13.8)	25.7 (12.5)	$F_{(1,22)} = 1.173$ $p = 0.291$	$F_{(1,22)} = 1.014$ $p = 0.325$	$F_{(1,22)} = 2.503$ $p = 0.128$	$F_{(1,22)} = 1.609$ $p = 0.218$	$F_{(1,22)} = 0.209$ $p = 0.652$	$F_{(1,22)} = 0.826$ $p = 0.373$	$F_{(1,22)} = 0.628$ $p = 0.437$
cSP 120% AMT (s)	0.085 (0.021)	0.091 (0.020)	0.092 (0.021)	0.095 (0.025)	0.101 (0.029)	0.099 (0.027)	0.107 (0.034)	0.099 (0.024)	**$F_{(1,22)} = 8.156$ $p = 0.009$**	$F_{(1,22)} = 0.015$ $p = 0.904$	$F_{(1,22)} = 0.232$ $p = 0.635$	$F_{(1,22)} = 1.530$ $p = 0.229$	$F_{(1,22)} = 0.056$ $p = 0.816$	$F_{(1,22)} = 0.617$ $p = 0.441$	$F_{(1,22)} = 0.042$ $p = 0.839$
cSP 150% AMT (s)	0.118 (0.018)	0.115 (0.025)	0.121 (0.023)	0.127 (0.019)	0.125 (0.023)	0.124 (0.028)	0.137 (0.021)	0.129 (0.021)	**$F_{(1,22)} = 6.257$ $p = 0.020$**	$F_{(1,22)} = 0.272$ $p = 0.607$	$F_{(1,22)} = 1.012$ $p = 0.325$	$F_{(1,22)} = 1.512$ $p = 0.232$	$F_{(1,22)} = 0.012$ $p = 0.915$	$F_{(1,22)} = 0.020$ $p = 0.889$	$F_{(1,22)} = 3.206$ $p = 0.087$
pre-stimulus EMG (%M_{max})	0.5 (0.1)	0.6 (0.2)	0.7 (0.5)	0.6 (0.4)	0.5 (0.2)	0.5 (0.2)	0.4 (0.1)	0.5 (0.1)	$F_{(3,20)} = 1.960$ $p = 0.153$	$F_{(3,20)} = 0.387$ $p = 0.763$	$F_{(3,20)} = 1.097$ $p = 0.374$	$F_{(3,20)} = 0.861$ $p = 0.477$	$F_{(3,20)} = 0.900$ $p = 0.459$	$F_{(3,20)} = 0.916$ $p = 0.451$	$F_{(3,20)} = 2.327$ $p = 0.105$
M_{max} (mV)	17.1 (5.9)	17.2 (6.1)	14.8 (5.2)	15.2 (4.9)	17.8 (6.7)	18.1 (7.2)	14.6 (4.0)	15.0 (5.9)	$F_{(1,22)} = 0.254$ $p = 0.619$	$F_{(1,22)} = 0.641$ $p = 0.432$	$F_{(1,22)} = 1.290$ $p = 0.268$	$F_{(1,22)} = 0.027$ $p = 0.871$	$F_{(1,22)} = 0.792$ $p = 0.383$	$F_{(1,22)} = 0.033$ $p = 0.857$	$F_{(1,22)} = 0.044$ $p = 0.836$
CSA_{Flexor} (mm^2)	1752 (551)	1859 (603)	1575 (491)	1683 (501)	1702 (557)	1823 (593)	1462 (480)	1568 (509)	**$F_{(1,22)} = 16.795$ $p < 0.001$**	**$F_{(1,22)} = 56.766$ $p < 0.001$**	$F_{(1,22)} = 0.942$ $p = 0.342$	$F_{(1,22)} = 0.286$ $p = 0.598$	$F_{(1,22)} = 3.445$ $p = 0.077$	$F_{(1,22)} = 0.063$ $p = 0.804$	$F_{(1,22)} = 0.577$ $p = 0.455$

Notes: Results summary, including mean (SD) and statistical main effects, for dependent variables measured before (PRE) and after (POST) unilateral strength training of both stronger and weaker arms in the symmetric and asymmetric strength groups. A: Arm; T: Time; S: Symmetry; Sym: Symmetry.

amplitude was large and favoured the weaker arm (EMG_{40}: $g = -0.84$, EMG_{100}: $g = -0.86$), while the magnitudes were small and moderate in the ASY strength group (EMG_{40}: $g = -0.21$, EMG_{100}: $g = 0.56$).

3.6. Pre-stimulus EMG before MEP Generation and M-wave Amplitude (M_{max})

No arm ($p = 0.153$), time ($p = 0.763$), strength group ($p = 0.374$) or interaction effects were observed for pre-stimulus EMG (%M_{max}) for all TMS trials (AMT, 120% and 150% of AMT). Similarly, no arm ($p = 0.619$), time ($p = 0.432$), strength group ($p = 0.268$) or interaction effects were detected for M_{max} (mV) (Table III). Trivial between-limb differences were calculated for the changes in M_{max} (SYM: $g = -0.09$, ASY: $g = 0.05$).

3.7. Active Motor Threshold (AMT), Motor Evoked Potential (MEP) Amplitude at 120% and 150% of AMT, Cortical Silent Period (cSP) Duration at 120% and 150% of AMT

Analysis of AMT (SO) demonstrated no arm ($p = 0.085$), time ($p = 0.083$), strength group ($p = 0.872$) or interaction effects (Table III). There were small and moderate effect sizes detected for the between-limb changes in AMT in the SYM and ASY strength groups, respectively (SYM: $g = 0.28$,

TABLE IV: Absolute and Normalised (SD) Rates of Force Development (RFD) in Stronger and Weaker Arms Before (PRE) and After (POST) Unilateral Strength Training of Both Arms in Individuals with Symmetric and Asymmetric Strength

		Symmetric				Asymmetric			
		0–50 ms	Δ	100–200 ms	Δ	0–50 ms	Δ	100–200 ms	Δ
Stronger Arm									
Absolute ($Nm \cdot s^{-1}$)	PRE	53.2 (17.4)		179.0 (63.6)		48.4 (29.3)		161.3 (60.5)	
	POST	57.4 (18.9)	4.1 (21.0)	178.3 (70.6)	−0.6 (35.9)	51.3 (22.6)	2.9 (39.5)	165.0 (70.9)	3.8 (41.0)
Normalised (%MVIC·s^{-1})	PRE	115.9 (63.0)		337.6 (43.3)		110.6 (83.7)		349.2 (80.9)	
	POST	113.1 (54.6)	−2.8 (44.7)	318.4 (59.4)	−19.2 (58.2)	123.8 (74.7)	13.3 (103.5)	338.1 (63.9)	−11.1 (71.0)
Weaker Arm									
Absolute ($Nm \cdot s^{-1}$)	PRE	50.6 (25.0)		162.4 (91.3)		60.8 (40.1)		134.5 (52.0)	
	POST	59.3 (11.5)	8.6 (26.9)	191.1 (84.2)	28.8 (53.0)	48.6 (17.4)	−12.1 (38.2)	157.2 (41.3)	22.6 (41.9)
Normalised (%MVIC·s^{-1})	PRE	114.4 (68.2)		307.3 (111.6)		147.0 (89.2)		324.2 (98.6)	
	POST	123.8 (51.3)	9.4 (64.0)	353.6 (62.1)	46.3 (102.5)	119.0 (56.9)	−28.1 (83.4)	357.8 (46.5)	33.6 (92.7)

Note: Change scores (Δ) are also shown. Mean (SD).

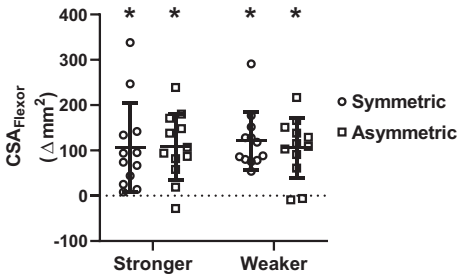

Fig. 7. The change score results for CSA_{Flexor} in the stronger and weaker arms of both groups following unilateral strength training of both arms. ∗Significant change from PRE, $p < 0.05$. Horizontal lines are group means with standard deviations (SD).

ASY: $g = 0.68$). Similarly, MEPs at 120% and 150% of AMT (%M_{max}) did not differ between arms or strength groups and did not change with training (Table III). Small and moderate between-limb effect sizes were calculated for the changes in MEP at 120% of AMT (SYM: $g = 0.14$, ASY: $g = 0.59$), and small effects were calculated for the changes in MEP at 150% (SYM: $g = 0.31$, ASY: $g = 0.38$). In addition, cSPs at 120% and 150% of AMT (s) were unchanged following training, but a between arm difference was detected (120%: $p = 0.009$; 150%: $p = 0.020$) (Table III) with the weaker arm showing a greater cSP at both TMS intensities. The magnitude of the between-limb differences were small for the changes in cSP at 120% of AMT (SYM: $g = 0.31$, ASY: $g = 0.38$) while they were trivial and large for the changes in cSP at 150% in the SYM and ASY strength groups, respectively (SYM: $g = -0.09$, ASY: $g = 0.94$).

3.8. Elbow Flexor Muscle Cross-sectional Area (CSA_{Flexor})

Significant arm ($p = 0.022$) and time ($p = 0.003$) effects were observed, but no strength group ($p = 0.269$) or interaction effects were detected for CSA_{Flexor} (mm^2) (Table III). Post-hoc testing demonstrated that the stronger arm had a larger CSA_{Flexor} than the weaker arm. In addition, significant increases were observed in the stronger (SYM: $\Delta = 107 \pm 98$ mm^2, $p = 0.003$; ASY: $\Delta = 107 \pm 73$ mm^2, $p < 0.001$) and weaker (SYM: $\Delta = 121 \pm 64$ mm^2, $p < 0.001$; ASY: $\Delta = 105 \pm 65$ mm^2, $p < 0.001$) arms in both strength groups following training (Fig. 7). Trivial effect sizes were determined for the between-limb CSA_{Flexor} changes in both strength groups (SYM: $g = -0.16$; ASY: $g = 0.03$).

TABLE V: Between-limb Relationships for Changes in Specific Dependent Variables in the Strength Symmetry-Asymmetry Groups Following Unilateral Strength Training of Both Arms (*r* [CI])

	Symmetric strength	Asymmetric strength
1-RM	0.881 (0.676, 0.987)	0.766 (0.367, 0.950)
$20°{\cdot}s^{-1}$	0.514 (−0.158, 0.918)	0.328 (−0.146, 0.735)
$210°{\cdot}s^{-1}$	0.600 (0.045, 0.905)	0.492 (−0.388, 0.783)
MVIC	0.342 (−0.562, 0.950)	−0.488 (−0.823, −0.096)
EMG_{peak}	−0.186 (−0.601, 0.249)	0.288 (−0.469, 0.846)
RFD_{50}	−0.642 (−0.867, 0.133)	0.409 (−0.185, 0.830)
$RFD_{100–200}$	−0.307 (−0.712, 0.395)	0.068 (−0.673, 0.647)
$RFD_{50/MVIC}$	−0.531 (−0.905, 0.210)	0.510 (0.171, 0.795)
$RFD_{100–200/MVIC}$	−0.148 (−0.628, 0.400)	0.021 (−0.870, 0.785)
EMG_{40}	0.233 (−0.293, 0.729)	−0.310 (−0.763, 0.467)
EMG_{100}	−0.162 (−0.711, 0.432)	−0.379 (−0.852, 0.793)
AMT	−0.291 (−0.781, 0.340)	0.192 (−0.298, 0.661)
MEP 120%	0.307 (−0.528, 0.755)	−0.345 (−0.815, 0.341)
MEP 150%	−0.138 (−0.904, 0.896)	−0.377 (−0.831, 0.296)
cSP 120%	−0.586 (−0.901, 0.337)	0.342 (−0.291, 0.810)
cSP 150%	0.292 (−0.288, 0.808)	0.270 (−0.573,0.748)
M_{max}	0.192 (−0.298, 0.661)	0.239 (−0.734, 0.798)
CSA_{Flexor}	0.919 (0.568, 0.982)	0.617 (0.249, 0.911)

Note: Significance was determined when a 95% confidence interval (CI) did not cross 0.00.

3.9. Between-limb Correlations for Changes in Specific Dependent Variables in the Strength Symmetry-Asymmetry Groups Following Unilateral Strength Training of Both Arms

In both strength groups, between-limb relationships were detected for the changes in 1-RM strength and CSA_{Flexor}, indicating that the stronger and weaker elbow flexors responded similarly. However, there was a positive relationship for the between-limb changes in isokinetic peak torque at $210°{\cdot}s^{-1}$ in the SYM strength group, and negative and positive relationships for between-limb changes in MVIC torque and $RFD_{50/MVIC}$ in the ASY strength group, respectively (Table V). These results indicate that individual differences exist between the groups, suggesting that between-limb responses can differ.

4. Discussion

Although there were baseline between-limb 1-RM strength differences between the strength groups, the main finding of the present study was that the stronger and weaker elbow flexors in both groups responded similarly to the same relative training stimulus. Specifically, there were similar improvements in 1-RM strength and CSA_{Flexor} in both arms, while only the stronger arm increased isokinetic peak torque at $20°{\cdot}s^{-1}$ in both groups. The only adaptive response that differed between the strength groups was an improvement in isokinetic peak torque at $210°{\cdot}s^{-1}$ for the stronger arm in the SYM strength group following strength training. Consequently, between-limb strength differences in individuals who had not undertaken strength training for at least 12 months do not appear to substantially affect the short-term (4-week) training responses in the elbow flexors. However, the fact that significant relationships were detected for the between-limb changes in isokinetic peak torque at $210°{\cdot}s^{-1}$ in the SYM strength group and in MVIC torque and early-phase rate of force development ($RFD_{50/MVIC}$) in the ASY strength group, signal that individual differences exist between the groups, especially in non-specific strength responses to the training modality.

Maximal strength, when measured as the 1-RM load, improved more than both slow- and fast-velocity isokinetic strength. By contrast, isometric strength (MVIC torque and RFD) and muscle activity (EMG) measured in isometric tests did not change in either group. Thus, a specificity of adaptation was observed with the greatest improvement in the free-weight (isoinertial) test. The lack of change in EMG with training suggests that the recruitment and firing of motor units, which predominantly influence the EMG amplitude (Farina *et al*., 2004), were not detected during isometric testing. This outcome was unexpected as muscle activation was hypothesised to improve following training, especially in the weaker arm in the ASY strength group. However, EMG was measured during isometric tests, and no increase in isometric strength was observed. In future studies, measurement of EMG during isoinertial and isokinetic tests might provide better insight into the potential mechanisms underpinning the strength gains following isoinertial strength training as movement pattern-specific responses are commonly reported (Baker *et al*., 1994; Sale *et al*., 1992). Thus, the specificity principle may not only influence maximal strength adaptations but also the ability to detect muscle activity

changes. Another reason why RFD and EMG measured in the early rise in force did not change may be the lack of intent for fast force production (i.e., load acceleration) during the training; it has been well demonstrated that training with the intent for fast force production is a key to RFD improvements (Behm & Sale, 1993; Del Vecchio *et al*., 2019; Van Cutsem *et al*., 1998). Likewise, there were no performance changes in late-phase RFD. Since MVIC torque is a primary determinant of late-phase RFD (Andersen & Aagaard, 2006), the present findings are consistent with the unchanged MVIC torque.

As the strength training period was restricted to 4 weeks in the present study, it was expected that muscle size changes would be relatively small (Abe *et al*., 2000), although elbow flexor muscle size has recently been shown to increase following 6 weeks of isoinertial strength training (Carvalho & Barroso, 2020). In the present study, the stronger and weaker elbow flexors in both groups showed relatively large increases in muscle size (stronger arm: SYM = 5.9%, ASY = 7.3%; weaker arm: SYM = 7.3%, ASY = 7.3%). As exercise-induced muscle swelling can significantly influence the reliability of muscle size measurements for several days following unaccustomed exercise (Rowe *et al*., 2018), muscle size was measured 6–7 days after the final training session. This provides confidence that the extent of the change reflects true muscle fiber hypertrophy rather than muscle swelling. Although previous studies have reported CSA increases following short periods of strength training (DeFreitas *et al*., 2011), the use of elbow flexors in the present study might explain the relatively large increases observed. Based on previous data, strength training-induced hypertrophy can be significantly greater in the elbow flexors (+22%) than the knee extensors (+4%) in response to 3 months of machine-based strength training (Welle *et al*., 1996). A possible reason for this difference is the consistently higher muscle activation observed in the elbow flexors (Behm *et al*., 2002), providing a potentially greater and more uniform stimulus for hypertrophy (Wakahara *et al*., 2013). Regardless of the reasons, increases in muscle size were similar between arms in each group, so the presence of differences in between-limb strength before training did not appear to affect hypertrophic responses, at least in the 4-week period of training used in the present study.

The sole neurophysiological difference detected in the present study was cSP, with a longer cSP in the weaker arm. cSP represents the influence of cortical and spinal inhibitory mechanisms affecting α-motoneurone excitability (Säisänen *et al*., 2008; Yacyshyn *et al*., 2016). Intracortical inhibition, mediated by γ-aminobutyric acid B ($GABA_b$) receptors, is thought to predominantly influence its duration. $GABA_b$-mediated inhibition results in the inhibition of descending output from the motor cortex and, therefore, a brief period of disfacilitation (removal of excitatory input) of the spinal α-motoneurones (Werhahn *et al*., 1999). The longer cSP found in the weaker arm is consistent with previous findings of a longer cSP in the non-dominant hand (Priori *et al*., 1999), with the researchers reporting that voluntary contractions of the non-dominant limb require greater cortical control. However, as cSP at both TMS intensities was not different between the strength groups, the findings did not influence the baseline strength differences.

Despite the between-limb difference in cSP, there was no difference in MEP amplitude between limbs, nor were there detectible MEP and cSP changes induced by the training. MEP size reflects the excitability of the primary motor cortex, functionality of the corticospinal tract, and conduction efficiency along the peripheral motor pathway (Hallett, 2000). An increase in MEP following training might suggest corticospinal projections to α-motoneurones are strengthened, possibly supporting increases in force production (Kidgell *et al*., 2017). However, the lack of change in both MEPs and cSPs indicates there was little change in the excitability of neuronal cells to strength training. As no changes in MVIC torque and EMG_{peak} were also observed, i.e., no change in the descending drive to the muscle during the maximal isometric testing, a lack of change in MEPs and cSPs may not be unexpected. However, the lack of changes may have also resulted from the potential incompatibility of the testing and training methods. Although MEPs and cSPs were measured with the muscles active, during which afferent signalling might be more similar to that evoked during strength training (i.e., compared to resting conditions), they were both measured during low-force isometric contractions, whereas the training was performed using high-force dynamic exercises. It is possible that changes might have been detected if the testing conditions were more similar to the training conditions, and this should be considered in future research. Interestingly, increases in MEP amplitudes during isometric testing have been reported in previously untrained participants who performed isoinertial strength training paced by a metronome, while increases were also observed after skill-based training but not self-paced strength training (Leung *et al*., 2017). These findings suggest that MEPs may reflect changes in motor learning rather than being a physiological adaptation that contributes strongly to changes in force production.

While the results indicate the between-limb responses to strength training are similar in individuals with SYM and ASY strength, the between-limb correlation data suggest that adaptation variation may occur in some individuals. Although between-limb changes in 1-RM strength and CSA_{Flexor} were

positively correlated in both groups, indicating the stronger and weaker arms respond similarly no matter the baseline strength difference, the between-limb changes in isokinetic and isometric strength were only correlated in SYM and ASY strength groups, respectively. Specifically, individuals with SYM strength were better able to improve fast-velocity isokinetic strength in both arms, while individuals with ASY strength were better able to improve early-phase RFD in both arms. A negative correlation was also detected in the ASY strength group for the between-limb changes in MVIC. While correlation analysis only allows comparison of the relationships among the groups, a possible explanation for the results is that individuals with SYM strength are better able to generate strength adaptations more similar to the training modality (isoinertial training and isokinetic strength changes) as opposed to individuals with ASY strength (isoinertial training and isometric strength changes). The negative correlation observed in the ASY strength group for between-limb changes in MVIC also suggests that individuals may have generated specialised strength adaptations following training. Future research should explore the theory that specialised adaptations contribute to strength changes in lateralised muscles following strength training. Whatever the reason, the between-limb correlation data suggests that individual differences exist between the groups, especially when assessing strength responses non-specific to the training modality.

In conclusion, based on the present results, the hypertrophic and strength adaptations after a 4-week training period were similar between arms following strength training of the elbow flexors despite there being between-limb 1-RM strength differences in the strength groups. Specifically, similar between-limb increases in 1-RM strength and CSA_{Flexor} were observed, while only the stronger arm improved isokinetic peak torque at $20°\cdot s^{-1}$ in both groups. The only adaptative response that differed between the groups was the stronger arm in the SYM group improving isokinetic peak torque at $210°\cdot s^{-1}$ following strength training. Thus, the assumption that the elbow flexors of both arms respond similarly to short-term strength training appears to be justified. Of interest, no changes in MVIC torque, RFD, muscle activity (either peak or EMG in the early phase of a rapid contraction), MEP, or cSP were observed, possibly indicating that isoinertial training with free weights may not lead to detectable neural or strength adaptations in isometric tasks; thus, a movement pattern (contraction mode)-specific training effect may occur. Lastly, the comparison of the between-limb relationships suggests that training responses in individuals with SYM or ASY strength can differ, especially when assessing strength responses non-specific to the training modality.

Conflict of Interest

The authors declare that they do not have any conflict of interest.

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