MECHANOMYOGRAPHIC ANALYSIS OF EXERCISE HYPERTROPHY - THE EFFECT ON MUSCLE CONTRACTILE PROPERTIES.

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This study investigated mechanomyographic (MMG) derived muscle changes during biceps brachii hypertrophy. Healthy participants (age: 23.73 ± 2.67, n=19) performed unilateral biceps curls for 8 weeks (non-dominant limb; treatment, dominant limb; control). Significant differences (p<0.05) were found between treatment and control limbs with treatment MVC initially declining (weeks 1-3), due to fatigue (decline in MPF), followed by improvement above baseline (weeks 6-8). Treatment muscle girth was greater than control (weeks 5-8), whilst MMG variables Dmax (maximal muscle displacement) (weeks 2, 7) and Vc (contraction velocity) (weeks 7, 8) declined. During de-loading, MVC and muscle girth (weeks 9, 10) remained higher, whilst Dmax and Vc both remained lower (week 9). MMG contractile property changes due to exercise induced hypertrophy suggest MMG has applicability as a diagnostic tool in rehabilitation.

KEY WORDS: Hypertrophy, Mechanomyography (MMG), Exercise.

INTRODUCTION: Many strength training regimes with the goal of lean muscle hypertrophy are employed by elite athletes in order to improve competitive performance (Brook, Wilkinson, Mitchell, Lund, Szewczyk, Greenhaff, Smith, & Atherton, 2015). Such regimes are also employed by injured athletes as a rehabilitative means before reintroduction to sport (Brook et al., 2015; Reuter, Proier, Imhoff & Lenich, 2016). Diagnosis of muscle contractile properties during hypertrophy currently has no definitive method. Recent evidence suggests that mechanomyographic (MMG) may prove to be an effective, non-invasive and cost efficient muscle performance quantification technique through analysis of physiological contractile properties such as muscular contraction time and velocity (Ibitoye, Hamzaid, Zuniga & Abdul Wahab, 2014). MMG has shown applications in evaluating muscular properties under voluntary and evoked muscle contraction and has been applied as a diagnostic tool to prevent musculoskeletal disorder, further demonstrating its promise in clinical practices (Ibitoye et al., 2014).Thus the aim of this study was to determine whether MMG was a viable form of detecting muscular hypertrophy in the biceps brachii from 8 weeks of resistance training, followed by a further 2 weeks of de-loading. The benefit of this study is the potential development of a cost effective muscle contractile property detection technique that may be implemented in clinical settings. Rehabilitative regimes for injured athletes are critical for effective return to activity. MMG provides the possibility of a quantitative measure to identify the success of such regimes.

METHODS: Ethical clearance for this study was obtained from the University of Queensland Medical Research Ethics Committee and informed consent was obtained from each participant. Male and female participants aged 18-30 (mean ± standard deviation, age: 23.73 ± 2.67, BMI 21.84 ± 2.45, n=19) with no history of biceps brachii injuries were recruited for a 10 week study. Baseline MMG, maximum voluntary contraction (MVC) and median power frequency (MPF) of electromyography (EMG) were taken in week 0 for both the dominant (control) and non-dominant (treatment) biceps brachii. Thickness ultrasound readings were also obtained in B-mode using a 7.5MHz ultrasound transducer probe (Mindray DP-50). All aforementioned measurements were taken at 50% total length of the biceps brachii, with MMG and MVC's conducted at 90° elbow flexion. Additional readings were taken at the end of each week throughout the 10 weeks. The intraclass correlation coefficient (ICC) of MMG's maximal muscle displacement (Dmax) parameter has been found to be good to excellent (ICC=0.86-0.96) whilst the ICC of muscle thickness ultrasound measurements has been found to be excellent (ICC=0.91-0.99), supporting the re-test reliability of both techniques

(Ditroilo, Smith, Fairweather & Hunter, 2013; Jenkins, Miller, Buckner, Cochrane, Bergstrom, Hill, Smith, Housh, & Cramer, 2015).

Thicknesses of ultrasounds were determined using ImageJ. Note: Week 4 ultrasounds are omitted due to conflicting scheduling with other ultrasound data not presented within this manuscript. The exercise protocol involved participants' using their non-dominant arm to bicep curl a dumbbell loaded to either 70%, 50% or 30% of that week's MVC recording (progressively lowering weight due to failure to maintain correct form with previous weight). A total of 9 sets of 12 repetitions were performed with 90 second rest intervals (5x weekly for weeks 0-8, de-loading in weeks 9-10). This loading strategy has been hypothesized as the ideal combination of mechanical tension and metabolic stress to maximize a hypertrophic response (Schoenfeld, 2010). Repeated measures two-way ANOVAs were conducted in Graph Pad Prism 6 (limb group x week) with protected fishers LSD post-hoc used to identify location of significance at $p < 0.05$.

RESULTS: During weeks 1-3, the treatment limb decreased in MVC against the control limb ($p < 0.05$) before increasing in weeks 6-8, whilst remaining higher in weeks 9-10 of de-loading ($p < 0.05$). MPF saw the treatment limb significantly less in output against the control arm for weeks 1-6 ($p < 0.05$). Thickness ultrasounds demonstrated treatment to be significantly thicker during weeks 5-8 and for weeks 9-10 of de-loading ($p < 0.05$). No significance was obtained for contraction time (Tc). MMG variable Dmax (maximal muscle displacement) of the treatment limb was significantly lower than the control for weeks 2 and 7 ($p < 0.05$), whilst Vc (contraction velocity) was slower for weeks 7 and 8 ($p < 0.05$). Both treatment Dmax and Vc were additionally lower in week 9 of de-loading.
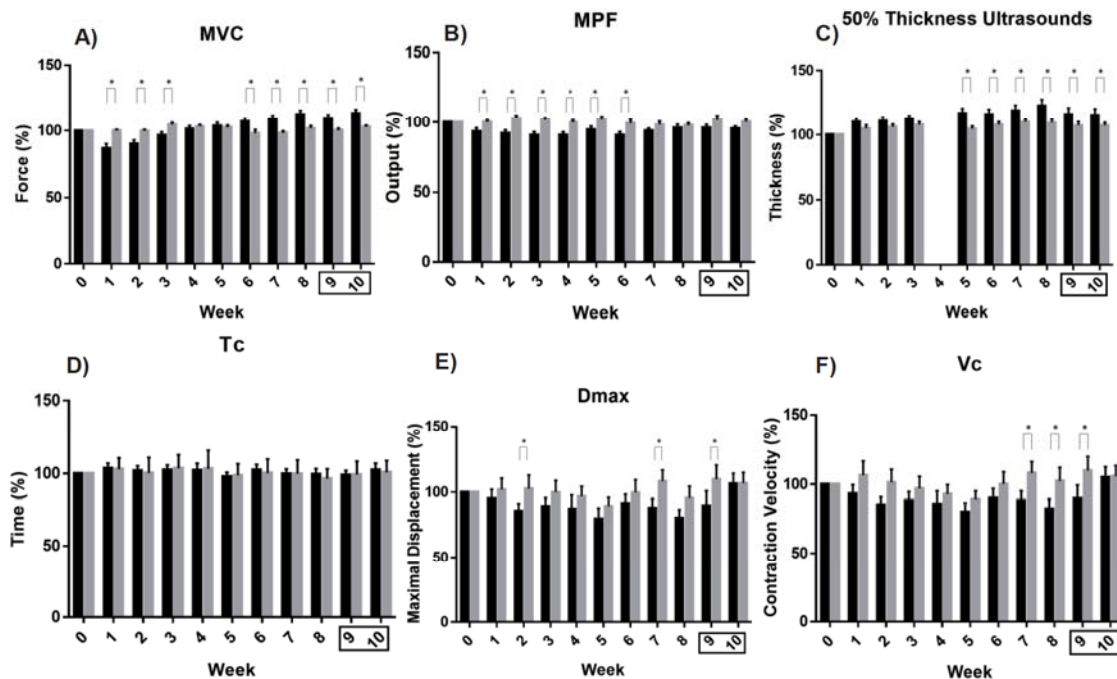

Figure 1: Study results. Mean (±SEM) treatment (left■) and control (right■) limb values each week for A) MVC B) MPF C) 50% thickness ultrasounds D) Tc E) Dmax and F) Vc. * indicates significant ($p < 0.05$) difference between limbs for each week. De-loading weeks 9 and 10 are outlined for each graph. Changes are as a percentage from week 0 baseline. Abbreviations: MVC; maximum voluntary contraction, MPF; median power frequency, Tc; contraction time, Dmax; maximal muscle displacement, Vc; contraction velocity, SEM; standard error mean.

DISCUSSION: MVC for weeks 1-3 saw the treatment limb significantly lower ($p < 0.05$) than the control limb (Figure 1A). This decline can be attributed to the unaccustomed nature of the treatment limb to the exercise protocol (Cheung, Hume & Maxwell, 2003). However, skeletal muscle rapidly adapts to its mechanical environment due to its plastic nature (Marcotte, West & Baar, 2015). Increased load across a muscle results in a compensatory increase in muscle size and strength (Marcotte, West & Baar, 2015). These adaptations to exercise are suggested to have occurred when treatment achieved higher MVCs than control ($p < 0.05$) in weeks 6-8 (Figure 1A). This perceived eventual adaptation of muscles to exercise is further supported by the MPF results of the EMG in which treatment limb demonstrated fatigue against the control limb for weeks 1-6 ($p < 0.05$) due to exercise (Figure 1B). However compensatory adaptation enables muscles to more efficiently use substrates for ATP production, providing the treatment limb fatigue resistance which was seen from week 7 onwards for the MPF results (Rockl, Hirshman, Brandauer, Fujii, Witters & Goodyear, 2007). During weeks 9 and 10 of de-loading, treatment MVC remained higher than control ($p < 0.05$) to indicate retention of strength. The MVC results were in line with muscle thickness ultrasounds (Figure 1C) in which treatment limb was significantly larger than control in week 8 ($p < 0.05$). The thickness ultrasounds demonstrated that the treatment limb was larger than control in week 5-8 ($p < 0.05$) of the hypertrophy phase, approximately in line with previous literature regarding the onset of hypertrophy (Ogasawara, Thiebaud, Loenneke, Loftin, & Abe, 2012). Weeks 9 and 10 of de-loading additionally demonstrated higher treatment thickness ultrasounds ($p < 0.05$), indicating no significant atrophy within the de-loading phase. The amplitude domain (Dmax) of recorded MMG signals are related to motor unit recruitment, with a linear relationship between the MMG amplitude and the work load of the contracting muscle (Al-mulla, Sepulveda & Colley, 2011). As such, these MMG parameters will change when skeletal muscle undergoes morphological changes and remodelling under phases of growth and wasting. During hypertrophy, contractile elements enlarge and the extracellular matrix expands (Vierck, O'reilly, Hossner, Antonio, Byrne, Bucci & Dodson, 2000). Additionally, there is an increase of sarcomeres and myofibrils added in parallel (Schoenfeld, 2010). These intrinsic changes within muscle in turn lead to altered contractile properties and recruitment patterns (Blaauw, Schiaffino & Reggiani, 2013). Dmax was significantly lower for treatment against control ($p < 0.05$) in weeks 2 and 7 of the hypertrophy phase (Figure 1E), suggesting intrinsic changes in the recruitment pattern of motor neurons to cause maximal muscle belly displacement. This change in neural control was reflected in the amplitude of the MMG waveform as a significantly diminished curve height. This lowered treatment Dmax was found in week 9 ($p < 0.05$) of the de-loading phase. Tc, which physiologically corresponds to completion time of the power stroke from the binding of actin and myosin during contraction, of the treatment limb saw no significant changes throughout the study (Figure 1D). However velocity of contraction (Vc), which physiologically corresponds to the rate of actin-myosin cross-bridge formation, did achieve significance (Figure 1F). As stated before, MMG parameters will change when skeletal muscle undergoes morphological changes and remodelling. This was evident in treatment Vc, which is reflective in the ascending slope of contraction in an MMG waveform. During weeks 7 and 8 of hypertrophy, the treatment limb was found to have a slower Vc than control ($p < 0.05$), suggesting pronounced modification of muscle architecture from hypertrophy. This slowed rate of contraction was also seen in week 9 ($p < 0.05$) of the de-loading phase. Physiologically, this demonstrates that alterations in fibre recruitment (Dmax) are somewhat coordinated with the velocity of actin-myosin cross-bridge formation (Vc) in order to maintain a stable time for the power stroke of contraction (Tc). The exact mechanisms on which this occurs remain to be elucidated; nevertheless MMG appeared to be able to identify these changes in contractile properties.

CONCLUSION: This investigation utilised mechanomyography (MMG) to non-invasively quantify changes in biceps brachii contractile properties following 8 weeks of exercise induced hypertrophy (rehabilitation) and 2 weeks of de-loading. The MVC and ultrasound

results appear to indicate that the treatment limb was successful in undergoing hypertrophy. Subsequently, it appears MMG is able to detect changes in muscle remodelling during a period of significant growth. While the exact mechanisms of remodelling remain unknown and require further studies, this investigation demonstrates MMG as a potentially viable technique to quantify these changes within a muscle as they occur. MMG therefore presents itself as a possible diagnostic and observational tool for muscle hypertrophy that may be applied within clinic upon further validation. The significance of this is the development of a diagnostic tool that may validate the efficacy of specific strength regimes with the intent of inducing hypertrophy, particularly for injured athletes desiring a return to activity.

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