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Research Notes

Is the rate of force development scaling factor associated with the rate of agonist muscle activation scaling factor and the level of agonist-antagonist coactivation?

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HIGHLIGHTS

- Neuromuscular quickness is evaluated by the rate of force development scaling factor (RFD-SF).
- The RFD-SF is associated with the rate of agonist muscle activation scaling factor (RAgMA-SF).
- The RFD-SF is not associated with the agonist-antagonist coactivation (I_{COA}).
- The RAgMA-SF may also be used as a measure of neuromuscular quickness.

ABBREVIATIONS

ADLs	Activities of daily living
BF	Biceps femoris
BFP	Brief force pulse
EMG	Electromyography
I_{COA}	Coactivation index
IMFT	Isometric maximum force test
RAgMA-SF	Rate of agonist muscle activation scaling factor
REMGF	Rate of EMG activity development
RFD	Rate of force development
RFD-SF	Rate of force development scaling factor
RMS	Root mean square
VL	Vastus lateralis

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BACKGROUND: Rate of force development scaling factor (RFD-SF) is used to assess neuromuscular quickness across submaximal levels. It has been suggested that such metric would be associated with the magnitude of the agonist muscle activation and with the level of simultaneous antagonist muscle group activation, i.e., agonist-antagonist coactivation.
AIM: To examine the associations between 1) the RFD-SF and the rate of agonist muscle activation scaling factor (RAgMA-SF) and 2) the RFD-SF and the agonist-antagonist coactivation.
METHOD: Eleven healthy and physically active subjects (7 men and 4 women, aged between 18 and 22 years) performed maximal strength tests of the knee extensors and flexors and a test that involved the production of brief submaximal force pulses of the knee extensors. The force exerted was recorded by a load cell and the electromyographic activities of the vastus lateralis and biceps femoris muscles were obtained during the tests. In the brief force pulses test, individuals were asked to produce around 120 submaximal force pulses that ranged between 20 to 80% of their maximum force value, while trying to produce each force pulse as fast as possible. The values of RFD-SF, RAgMA-SF of the vastus lateralis muscle, and a coactivation index (I_{COA}) were computed.
RESULTS: The RFD-SF is strongly associated with the RAgMA-SF (r=0.74; p=0.009) but not with the I_{COA} (r=0.29; p=0.38).
CONCLUSION: The RFD-SF is directly associated with the rate of agonist muscle activation and is not affected by the degree of coactivation between agonist and antagonist muscles.

KEYWORDS: Muscle power | Neuromuscular quickness | Electromyography | Motor control | Cocontraction | Coordination

INTRODUCTION

The performance of sports and activities of daily living (ADLs) relies on a person's ability to generate quick muscle force, known as neuromuscular quickness, at different intensities. Although maximal neuromuscular quickness is crucial for athletic performance [1,2], many ADLs may involve fast movements at submaximal force levels [3]. Recently, a new measure called the rate of force development scaling factor (RFD-SF) has been introduced to estimate neuromuscular quickness at submaximal levels [4].

To determine the RFD-SF of a muscle group, individuals complete an isometric brief force pulse (BFP) experimental protocol. This protocol involves generating brief force pulses as quickly as possible within a submaximal force range (e.g., 20-80% of the maximum muscle group force [4–6]). The RFD-SF is calculated as the slope of the relationship between the peak values of the force pulses produced and their corresponding rate of force development [4]. Previous studies have demonstrated that the RFD-SF is a sensitive measure for detecting neuromuscular system impairments [5,7,8]. It has been observed that older adults exhibit lower RFD-SF compared to younger individuals [5,7], and individuals with multiple sclerosis [6,9,10] or Parkinson's disease [8] have lower RFD-SF compared to healthy controls.

RFD-SF has been suggested to have a direct relationship with both central (i.e., motor unit recruitment and firing rate) and peripheral factors (i.e., muscle fiber type) of the agonist muscle group responsible for force generation [4]. Additionally, it is speculated that RFD-SF is influenced by the level of coactivation between agonist and antagonist muscles [7,11]. This study is the first investigating the relationship between RFD-SF and the rate of agonist muscle activation scaling factor (RAgMA-SF), and between RFD-SF and the level of agonist-antagonist coactivation. We hypothesized that there would be a moderate to strong positive association between RFD-SF and RAgMA-SF, and a weak to moderate negative association between RFD-SF and agonist-antagonist coactivation.

METHODS

Participants

Eleven healthy individuals (7 men and 4 women) aged 18 to 22 years, who regularly participated in physical activities for at least three sessions of 30 minutes per week, were recruited for the study. The participants had an average height of 177 cm (standard deviation=9) and weighed 71 kg (12.2). The study protocol was approved by the institutional review board of Rowan University and conducted in accordance with the Declaration of Helsinki. Prior to the test, participants were instructed to abstain from exercising their lower limbs for two days.

Apparatus and subjects' positioning

A customized instrumented knee extensor chair was used in this experiment (Fig 1A). A load cell (SM-500, Interface Inc.) was attached to the chair and to the participants dominant leg just above the ankle joint. Bipolar surface electrodes (Bagnoli™, Delsys, Boston, MA, USA) were placed on the muscle bellies of the vastus lateralis (VL) and short head biceps femoris (BF) following the SENIAM recommendations [12].

The subjects sat on the chair with tightly wrapped straps around their thighs and hips on the tested side (dominant) to prevent compensatory movement. They assumed a position with hips flexed at approximately 90° and knees at approximately 70° of flexion from full extension. In this position (Figure 1A), the participants underwent two protocols described below.

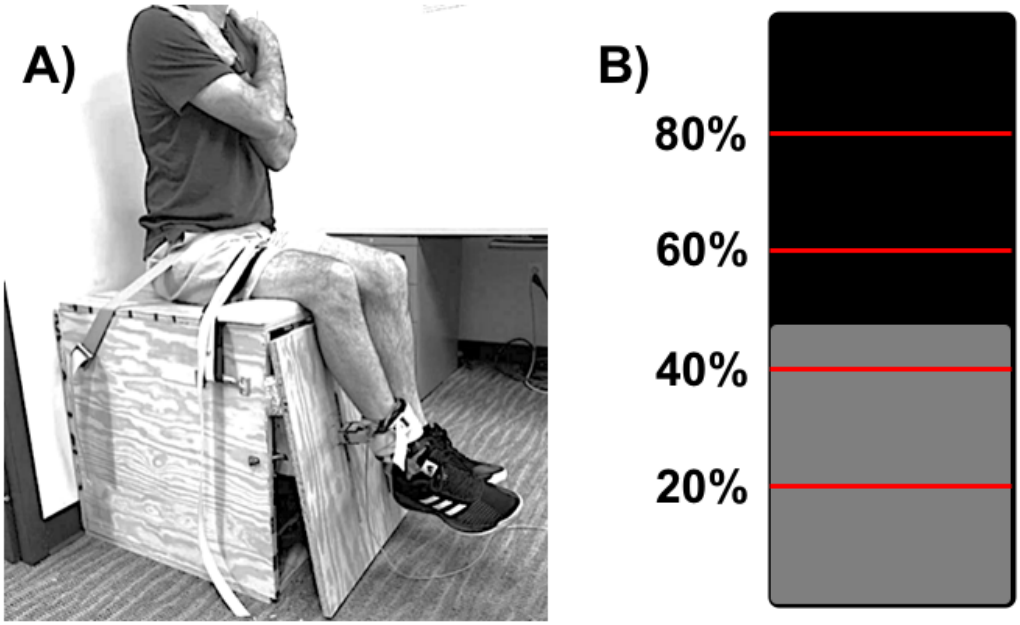

Figure 1. (A) Experimental setup depicting the subject's positioning during the test. (B) Vertical graph (tank graph with black background) presented to the subject during the brief force pulse protocol, illustrating the applied force level (dynamic gray rectangle), accompanied by horizontal red lines denoting force percentages (in % of the subject's maximum isometric force) and the corresponding ranges within which the force pulses were to be generated.

Isometric maximum force test (IMFT) protocol

For the isometric maximal strength test of the knee extensors and flexors, the instruction was to "kick as hard as possible" and "pull back as hard as possible", respectively. Participants performed three trials of knee extension and flexion, with each trial lasting 4 seconds and a 60-second rest interval between trials. The highest knee extensor strength value obtained was used to establish the target strength values for the BFP protocol.

BFP protocol

Participants were instructed to rapidly generate force pulses using their knee extensors at submaximal levels, isometrically "kicking and relaxing as fast as possible." A vertical bar graph shown on a computer monitor displayed the real-time force exerted by the subject. (Fig 1B). The force was represented as the percentage of the subject's maximum isometric force. The graph featured four red horizontal lines indicating 20, 40, 60, and 80% of the maximum recorded force, serving as reference points for three force ranges: small (20-40%), medium (40-60%), and large (60-80%). These predetermined ranges were employed as cues to guide participants in generating force pulses of specific magnitudes. For instance, upon receiving the command "small," participants were expected to produce a pulse falling within the small range. The same principle applied to the "medium" and "large" commands as well. Four trials, each lasting 40 seconds, were conducted, with participants performing 30 force pulses in each trial, resulting in a total of 120 force pulses per subject. In each trial, participants completed 10 pulses within each of the target ranges.

Data processing

Custom-written LabView routines were utilized for data recording and analyses. Force and electromyographic signals were synchronized, A/D transformed and recorded at 1000 Hz (NI USB-6341, 16 bits, NI). The force signal underwent filtering using a fourth-order low-pass Butterworth filter with a cutoff frequency of 50 Hz. The rate of force development (RFD) time-series was calculated and filtered using a similar filter with a cutoff frequency of 5 Hz, as it demonstrated the highest reliability for RFD-SF calculation[13]. The force peaks (corresponding to each force pulse) and the respective RFD peaks were plotted as a scatter plot, and a best-fit line was drawn to determine the slope, which represented the RFD-SF.

The VL and BF electromyography (EMG) signals were reduced, rectified, and filtered using a Butterworth digital low-pass, zero-lag filter with a cutoff frequency of 50 Hz. The EMG signals recorded during the BFP protocol were used to calculate the EMG outcomes, which were normalized by the maximum EMG values from the IMFT protocol. Specifically, the peak VL EMG signal in the IMFT protocol was identified, and the root mean square (RMS) of the VL EMG signal within a ±100 ms window around the peak was calculated in the trial with the maximum knee extensor force. The same procedure was applied to the BF EMG signal in the trial with the maximum knee flexor force.

In the BFP protocol, two outcome variables related to EMG were computed: the rate of agonist muscle activation scaling factor (RAgMA-SF) and the index of agonist-antagonist coactivation (I_{COA}). To compute the RAgMA-SF, after filtering and normalizing the VL EMG time-series (see earlier description), we (1) calculated the rate of VL EMG activation, (2) filtered it with a Butterworth filter with a cutoff of 5 Hz, (3) detected the peaks of normalized VL EMG and the respective peaks of the rate of VL EMG, (4) scatter plotted these peaks, (5) found the best fit line by a regression analysis, and (6) determined RAgMA-SF as the slope of this line. This process was similar to the one used to estimate the RFD-SF. The R^2 values from the regression analyses reveals the relationship consistency in the force and EMG domains [3,11]. An R^2 value near 1 indicates that the participants effectively adjusted force/muscle activation rate with peak force/muscle activation magnitude.

To compute the I_{COA}, we first identified the force peaks and the corresponding peaks of the normalized VL EMG that preceded these force peaks. For each VL EMG pulse, we found the moment when it exceeded 10% of its peak value, establishing the onset of phasic VL activation. Next, we estimated the time interval between the onset and peak of VL activation. Within this interval, we calculated the areas under the curve of VL and BF activation to obtain the integral of the electromyographic activities of both muscles ($IEMG_{VL}$ and $IEMG_{BF}$). To obtain the I_{COA}, we divided $IEMG_{BF}$ by $IEMG_{VL}$ and multiplied by 100. Finally, we averaged the coactivation levels across all pulses, resulting in the I_{COA} for each subject.

Statistical analysis

As the dependent variables were normally distributed according to the results of the Shapiro-Wilk test, we performed Pearson's correlation between RFD-SF and RAgMA-SF and between RFD-SF and I_{COA} to test our hypotheses. Pearson's correlation coefficients and 95% confidence intervals (95% CI) are presented. Alpha value was set at 0.05

RESULTS

The R^2 values of the best fit lines obtained from the relationship between the force and RFD peaks ranged from 0.57 to 0.96 with a median of 0.87. The R^2 values of the relationship between the peak VL EMG and the corresponding rate of VL EMG ranged between 0.52 and 0.9 with a median of 0.76. Furthermore, the results revealed a positive strong correlation between RFD-SF and RAgMA-SF [r=0.74 (0.25:0.93), p=0.009, two-tailed], Figure 2, left-hand side). However, there was no significant correlation between RFD-SF and I_{COA} [r=-0.29 (-0.76:0.38), p=0.38, two-tailed, Figure 2, right-hand side].

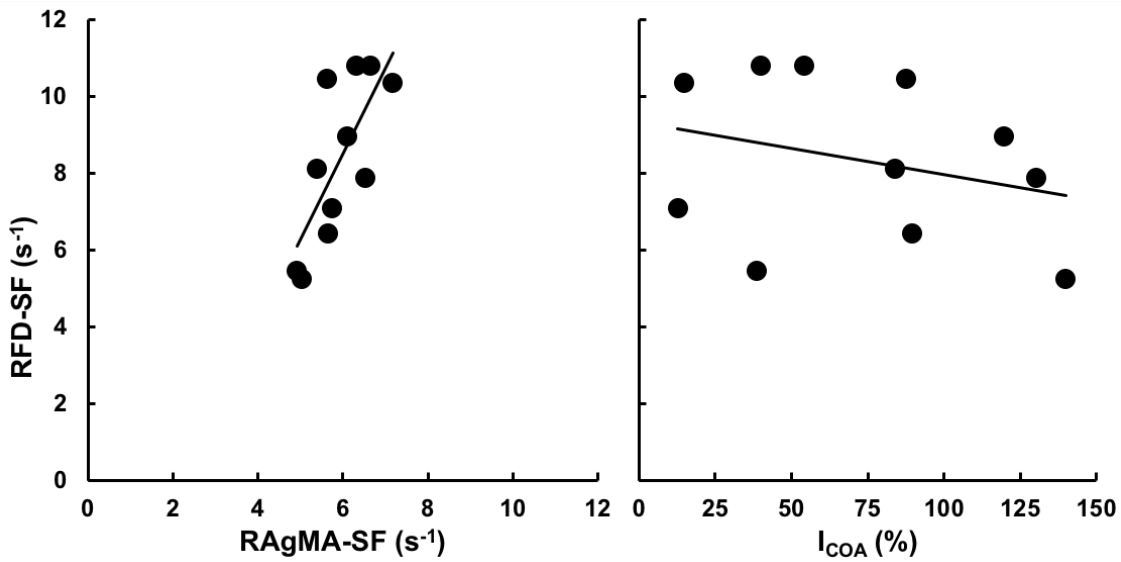

Figure 2. Scatter plots showing the relationships between rate of force development scaling factor (RFD-SF, y-axis) and rate of agonist muscle activation scaling factor (RAgMA-SF, x-axis) in the left-hand side and RFD-SF (y-axis) and index of coactivation (I_{COA}, x-axis) in the right-hand side.

DISCUSSION

In this study, two hypotheses were tested: there would be 1) a moderate to strong positive association between RFD-SF and RAgMA-SF and 2) a weak to moderate negative association between RFD-SF and I_{COA}. First hypothesis was confirmed and the second was falsified, since the results revealed a high correlation between RFD-SF and RAgMA-SF and no correlation between RFD-SF and I_{COA}.

The expected strong relationship between RFD-SF and RAgMA-SF is not surprising given the crucial role of the agonist muscle (VL) in knee extension, contributing to both variables. Although not found in existing literature, our study, as the first to investigate this, observed a correlation supporting our hypothesis of a strong association between RFD-SF and RAgMA-SF. Several studies show a nearly linear relationship between force and EMG activity, indicating an increase in EMG signal amplitude with greater force and/or rate of force development [14–16]. A quasi-linear relationship between force and EMG activity was observed in the left tibialis anterior [15], vastus lateralis, vastus medialis, and rectus femoris, with the strongest linear relationship found in the vastus lateralis [14,17]. This may be due to the fact that the vastus lateralis is solely involved in knee extension as a uniarticular muscle. Furthermore, individuals after a strength training program showed a parallel increase in both RFD and the rate of EMG activity development (REMGD), indicating that an increase in REMGD modifies RFD. This is attributed to the increase in efferent neural drive, specifically motor unit firing frequency [18,19]. Also, a previous study observed age-related declines in maximum RFD and reduced REMGD amplitude during the early phase of rapid muscle contractions, indicating a direct association between neuromuscular activation rate and rapid movement performance [20].

In addition to the agonist muscle's role in RFD-SF, we examined the relationship between antagonist activation and RFD-SF using the index of agonist-antagonist coactivation (I_{COA}). While no study directly examined the inverse relationship between antagonist muscle activation and RFD-SF, suggestions have been made that antagonist muscle coactivation could negatively affect RFD-SF [7,11]. Based on this, we hypothesized that increased antagonist muscle activation could negatively influence RFD-SF, as higher antagonist muscle activation during maximal efforts tends to result in lower maximal force and RFD production [2]. However, our results did not support this hypothesis, as we found no significant relationship between I_{COA} and RFD-SF. Consequently, we proposed several factors that might have contributed to the absence of correlation between RFD-SF and I_{COA}.

The absence of correlation between RFD-SF and I_{COA} may be attributed to the fact that the antagonist muscle is not directly involved in rapid force production during submaximal force levels. However, we remain unconvinced that this is the sole explanation. In our study, we focused solely on evaluating one antagonist muscle involved in the task (i.e., BF). Possibly, other muscles contributing to knee flexion may influence the decreased force and RFD observed in the agonist action, but they were not assessed in our study. Similarly, we solely examined the contribution of the VL to rapid force generation, while other quadriceps muscles may contribute similarly or even more meaningfully to pulse force generation. Therefore, to enhance the reliability of this index in future studies, it is recommended to assess additional muscles involved in knee extension and other antagonist muscles. Another approach could be to assess elbow flexion or extension.

Additionally, we acknowledge the small sample size in our study and recommend addressing it in future research. Also, it is crucial to evaluate a comparable sample size of both men and women to determine potential differences in coactivation levels between sexes and their impact on RFD-SF. Similarly, it would be important to examine the involvement of agonist-antagonist coactivation in the difference between young and older adults in the RFD-SF [5,7]. Future studies should also investigate the motor unit behavior during the generation of brief force pulses performed to submaximal magnitudes.

CONCLUSION

Based on our findings, we conclude that RFD-SF is strongly related to agonist muscle activation, suggesting that RFD-SF can be considered as a valid measure to assess neuromuscular capacity. However, we found no relationship between RFD-SF and agonist-antagonist coactivation, indicating that RFD-SF may not accurately estimate muscle coordination. Future research should include multiple agonist/antagonist muscles involved in knee extension/flexion to draw more reliable conclusions in this regard.

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