The effect of strength training on normalized mutual information of untrained subjects' sEMG signals during bench press

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ABSTRACT

Despite the proven effectiveness of strength training (ST) as a method to increase force output, the underlying neurophysiological mechanisms are not yet fully understood. Besides changes in muscular architecture, ST is thought to provoke adaptations in the nervous system as well, e.g. changes in intermuscular coordination. The latter can be investigated by computing the normalized mutual information (NMI) between the respective surface electromyography (sEMG) signals of two muscles. The present study used data from a previous randomized controlled trial by analyzing the sEMG recordings of a training group (TRA) of untrained males that underwent a five-week ST program focusing on the bench press (BP), and those of a control group (CON). The BP cycle was divided into 4 phases, and 15 pairs of the main agonist and antagonist muscles were formed, yielding a total of 60 muscle pair – movement phase combinations (MPMPC). TRA improved their three-repetition maximum (3RM) of the BP significantly (averages: 56kg in the pre- and 64.8kg in the posttest), while CON's 3RM remained stable (averages: 55kg in the pre- and 55.2kg in the posttest). Nevertheless, a statistically significant effect of the training program was found in only two MPMPCs (anterior deltoid – triceps brachii (medial head) in the first part of the eccentric phase ($p = 0.024$); anterior deltoid – biceps brachii in the first part of the concentric phase ($p = 0.019$)). The results indicate that changes in intermuscular coordination do not seem to contribute heavily to the initial strength gains experimented by beginners that take up ST.

Keywords: Strength training, normalized mutual information, bench press, surface electromyography

INTRODUCTION

Strength training (ST) is a widely used method to elicit physical adaptations for improved athletic performance, preventive or rehabilitative measures, or recreational purposes. These adaptations can be grouped into two categories, since they either relate to neural factors or to changes in the morphology of the muscle-tendon-complex.[123] Said neural factors may include modifications at the level of the single muscle, such as increased motor unit (MU) activation or firing rate,[4] and changes in the co-activation patterns between muscles.[5] There is a consensus that the initial strength gains experimented after the first 4-6 weeks of a resistance training program are due to neural factors, while morphological changes have a bigger impact if the ST program lasts 8-12 weeks or longer.[6] The relationship between the different types of neural adaptations and their precise anatomical locations are still not fully understood, since said changes may occur at the spinal or supraspinal level, and the possibilities to link experimental results to one of these levels remain limited.[2] Further, a measured increase in neural drive may be caused by a reduction of inhibitory or an increase in excitatory mechanisms.[7]

Neural mechanisms responsible for muscular contractions can be studied through surface electromyography (sEMG),[89] which records the electric potential field of the muscle's outer membrane.[10] However, the sEMG signal is not a direct depiction of muscular effort or neural drive,[1112] as its shape is codetermined by the properties of the muscle fiber membranes.[13] Consequently, an increase of force after a ST protocol is not necessarily detectable in an sEMG recording.[14] Further, maximal force as measurable outcome does not only depend on the capabilities of the individual muscles' motor units, but is produced by several muscles that act in concert around a given joint.[8] The insight that muscles work in constant interaction with each other has given rise to complex approaches that seek to establish and quantify muscle synergies by analyzing multichannel sEMG recordings that include a variety of different muscles.[5]

Recently, investigators have begun to analyze sEMG recordings with a set of novel mathematical tools called functional connectivity indices (FCIs),[151617] which originated in brain research. FCIs are used to compare oscillations measured at different parts of the brain or the neuromuscular system, in order to find patterns whose similarity to each other is too great to be caused by chance.[18] Normalized mutual information (NMI) is a FCI that computes the shared entropy between the sEMG signals of two muscles, or the reduction in uncertainty about Muscle A's sEMG values if those of Muscle B are known. It is able to detect both linear and non-linear statistical dependencies.[19] In the field of sEMG research, NMI has been used to quantify intermuscular coordination, i.e. in the presence of fatigue[20] and chronic pain,[17] or in comparisons of motor strategies between age groups[21] and between sexes.[22]

Despite the consensus on the importance of neural factors in early strength increases, the respective contributions of MU firing rate, MU recruitment and improved intermuscular coordination remain difficult to estimate.[232] The fact that the rapid strength gains of beginners are task-specific to a significant extent[2] invites the supposition that changes do not only affect MU properties like maximal firing rate or recruitment threshold, but also the subjects' ability to translate these properties into force production under task constraints via the coordinated interplay of several muscles. Research in this area has reported that deliberate practice can affect inhibitory reflex circuits that connect synergist muscles, e.g. brachioradialis and biceps brachii.[23] Further, a reduction of agonist-antagonist coactivation is often observed during movement skill acquisition.[24] Given that agonist and antagonist produce forces of opposite directions, this mechanism could potentially contribute to the increase in force output shortly after the onset of ST . To detect adaptations of these kinds, a computational tool that quantifies pair-wise interactions, such as NMI, appears to be well-suited.

The aim of the present study was to investigate possible changes in intermuscular coordination as an early adaptation to ST stimuli. It reanalyzes data recorded by Kristiansen et al.,[25] who conducted a randomized controlled trial (RCT) in which ST-naive subjects underwent a 5-week ST intervention focused on the bench press (BP). The pre- and post-intervention NMI values of different muscle pairs are compared.

Bench press (BP) is considered one of the fundamental upper-body exercises in ST,[26] and is widely used for training and testing purposes.[27] It is a barbell exercise whose starting position has the subject laying in a supine position on a bench, with the feet planted on the ground and the arms extended. The eccentric phase consists of lowering the barbell towards the chest, while the concentric phase corresponds to bringing it back up.[262728] In general, the relationship between sEMG amplitude and force is known to differ between concentric and eccentric contractions (Madeleine et al. 2001).[29] Further, in the BP different muscle synergies are at work in the two phases.[30] The main agonist muscles of this multi-joint exercise are *pectoralis major* (PM), *anterior deltoid* (AD) and *triceps brachii* (TB).[262731] To the author's knowledge no previous study on BP has used NMI as an outcome measure.

It was hypothesized that the five weeks of ST would have a significant impact on the intermuscular coordination of TRA. As more coordinated agonist muscle action should have a positive impact on the participants' strength levels, a significant increase in the NMI of agonist muscle pairs was expected in TRA. Conversely, simultaneous activity of antagonist and agonist muscles provides joint stability,[7] but does not act in the same direction as the applied force, which is why its inhibition is thought to constitute a hallmark of skilled motor performance.[24] For this reason, the NMI of muscle pairs consisting of one agonist and one antagonist was expected to decrease significantly in TRA after the ST intervention.

METHODS AND MATERIALS

This paper uses data recorded by Kristiansen et al.[25] and only differs from their publication in the analysis of it. For convenience, a brief summary of the subjects and the training and testing protocol will be provided nevertheless.

Experimental approach to the problem: The study was designed as a randomized controlled trial (RCT) and thus compared a training group (TRA) to a control group (CON). The initial familiarization session served to accustom the subjects to the test protocol and laboratory environment, along with the recording of anthropometric measurements. All subjects complete the pretest at least a week after the familiarization session. Matched pairs were formed according to the participants' anthropometric data and their initial BP performance, before being split up randomly into TRA and CON. The posttest was performed after TRA had completed their 5-week ST program.

Subjects: Participation in the study was restricted to males of 18 to 40 years of age who had no history of upper-extremity injuries requiring surgery and who did not participate in any ST / resistance training program for at least two years prior to the start of the study. The thirty participants were divided into 17 members of TRA (age: 26 ± 5 years (mean ± standard deviation (SD)), height: 180.0 ± 6.6cm, body mass: 77.2kg ± 11.1kg) and 13 members of CON (age: 23 ± 3 years, height: 180.4 ± 7.9cm, body mass: 77.2kg ± 16.2kg). Unequal group sizes accounted for anticipated possible TRA dropouts during the training program. The subjects were made aware of the experimental protocol and the associated risks of participation verbally and in writing. They gave their written informed consent, and the North Jutland Region's ethics committee approved the study (N-20120036), which received number ISRCTN10375612 in the international registry of RCTs.

Test protocol: While the original study by Kristiansen et al.[25] used BP sets of 8 repetitions with a load of 60% of the subjects' three repetition maximum (3RM), the present reanalysis employs the sEMG recordings of their 3RM tests, completed after the initial warm-up in both test sessions. The subjects performed 8-10 BP repetitions with an empty 20kg barbell as first warm-up set. Thereafter, the load was increased by 10-40kg for the second warm-up set of five repetitions. The third and last warm-up set consisted of three repetitions, the weight increment was 5-30kg. The subjects' BP performance in the previous familiarization session was used to individualize the load during warm-up and testing. A standard rest period of four minutes was observed between all sets of the test sessions. In the 3RM test, the participants performed sets of three repetitions until their 3RM was established, using load increments of 2.5-10kg. On average, four test sets were needed to determine the 3RM.

Training program: The ST intervention consisted of three weekly sessions (on monday, wednesday and friday) of approximately one hour each. Master students of sports science with at least two years of experience in the field of ST acted as personal trainers and supervisors, ensuring compliance with the training program and the correct execution of the exercises. Further, they adjusted the subjects' training loads whenever they deemed it necessary. The training program was comprised of the BP as main exercise, five assistive back exercises included to avoid the development of anterio-posterior muscular imbalances, and the push down as complimentary exercise that targeted *triceps brachii*. The number of sets and repetitions followed a progression in the BP while remaining constant in the assistive exercises, where a progressive overload was accomplished by adjusting the load only. Three submaximal BP sets constituted the standardized warm-up protocol, with 12 repetitions in the first, 10 in the second and 8 in the third warm-up set. During the workouts, rest intervals were 3 minutes for BP and 2 minutes for the assistive exercises. BP was performed in every session, while assistive exercises were alternated. Tables 1 and 2 below show the programming used in the 5-week ST intervention.

Table 1: BP progression

Week 1-2	Week 3-4	Week 5
3*6 at 7RM	3*5 at 6RM	4*3 at 4RM

*Notation: number of sets * number of repetitions at load intensity*

Table 2: alternation of assistive exercises

Mondays	Wednesdays	Fridays
Barbell bent over rows: 4*12 at 14RM	Push down: 3*12 at 14RM	Seated rows: 3*12 at 14RM
Face pulls: 3*12 at 25RM	Lat pull down (neutral grip): 3*12 at 25RM	Bent over reverse dumbbell flies: 3*12 at 25RM

*Notation: number of sets * number of repetitions at load intensity*

sEMG recording: In each testing session, surface EMG electrodes ((Ambu Neuroline 720 01-K/12, Ag/AgCl, inter-electrode distance 20mm, Ambu A/S, Ballerup, Denmark) were placed on the shaved, abraded and cleaned skin of the subjects before they started warming up. The muscles included in this study are PM, AD, *latissimus dorsi* (LD), *biceps brachii* (BB), and the long and medial head of *triceps brachii* (TBL and TBM, respectively). The right side of the body was used in all subjects, and the exact electrode locations were taken from the SENIAM guidelines,[32] if available. No SENIAM recommendations exist on PM and LD, whose positions were as follows:[33]

- PM: four fingerbreadths below the clavicle and medial to the anterior axillary border
- LD: three fingerbreadths distal to and along the posterior axillary fold, parallel to the scapula's lateral border

The right ankle's lateral malleolus was used as the location of a reference electrode. The same person was in charge of mounting all electrodes on all subjects. A skin-electrode impedance check was performed, and the recording procedure was allowed to start if there were no electrodes with an impedance of more than 10 kΩ.[34]

Barbell position: A potentiometer (Model KS60, NTT Nordic Transducer, Hadsund, Denmark) was attached to the center of the barbell, which allowed to track its vertical position.

Data acquisition and processing: A 128-channel surface EMG amplifier (EMGUSB, LISiN - OT Bioelectronica, Turin, Italy) was used to amplify the sEMG signal with a subject-specific gain factor between 500 and 2000. An initial band-pass filter of 10-750Hz was applied. The sampling frequency was set to 2048Hz.

Digital data processing was carried out using MATLAB (Version 2021a, MathWorks Inc., Natick, USA). The signal was filtered using a 4th-order digital Butterworth filter of 5-500Hz with an additional 1 Hz notch filter centered at the power line frequency of 50Hz. Due to the high load of a 3RM test and possible fatigue effects setting in during the second repetition, only the first repetition of each 3RM trial was selected for further analysis. The recordings of two subjects belonging to TRA had to be discounted due to data quality issues.

NMI: Computing the NMI of two data series requires calculating the signals' entropy values first, which in turn requires the selection of a bin number to construct histograms. Said bin number was determined using the Freedman-Diaconis rule, which is based on the number of data points and the interquartile range.[35] The corresponding equation is as follows:

$$n_{bins} = \lceil \frac{max(x) - min(x)}{2Q_x n^{-1/3}} \rceil$$

where Q_x represents the interquartile range (or the difference between the 75th and the 25th percentile) of data distribution X, n is the total number of data points and max(x) and *min(x)* correspond to the maximal and minimal value, respectively. This rule finds the optimal number of bins for a single variable.[35] To determine a single bin number across multiple muscles, subjects and test sessions, the above equation was applied to all data series, and the average value was rounded to the next higher integer.[19] This led to a bin number of 16.

The individual entropy of each time series can be calculated via the equation:

$$H(X) = -\sum_{i=1}^{n} p(x_i) log(p(x_i))$$

where H stands for the measure of entropy, n is the aforementioned bin number, and $p(x_i)$ represents the probability of a given value of time series X falling into bin number i.[19] A similar formula is employed to find the *joint* entropy of two time series:

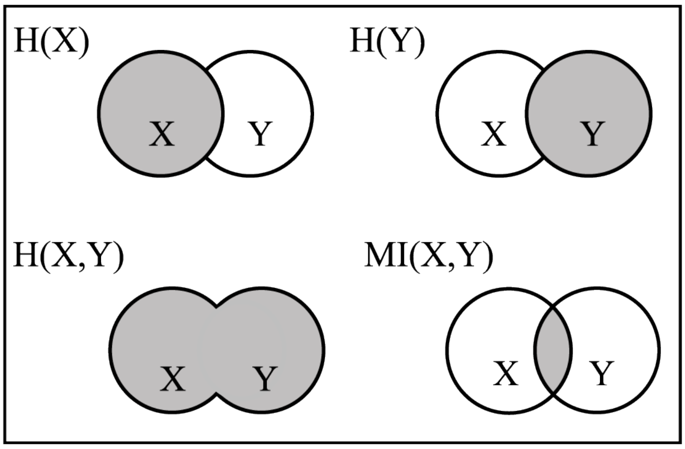
$$H(X,Y) = -\sum_{i=1}^{n}\sum_{j=1}^{m} p(x_i,y_j)log(p(x_i,y_j))$$

where the second time series Y has been added, along with its corresponding index j. Mutual information (MI) of two time series can now be obtained by summing up individual entropy and subtracting joint entropy:[19]

$$MI(X,Y) = H(X) + H(Y) - H(X,Y)$$

The relationship between the individual entropy, joint entropy and MI of two time series is represented graphically in Figure 1 below.

Figure 1: relationship between entropy and MI of two time series



The upper row shows entropy of time series X (left) and Y (right), while the lower row shows joint entropy (left) and MI (right). Adapted from Cohen (2014)

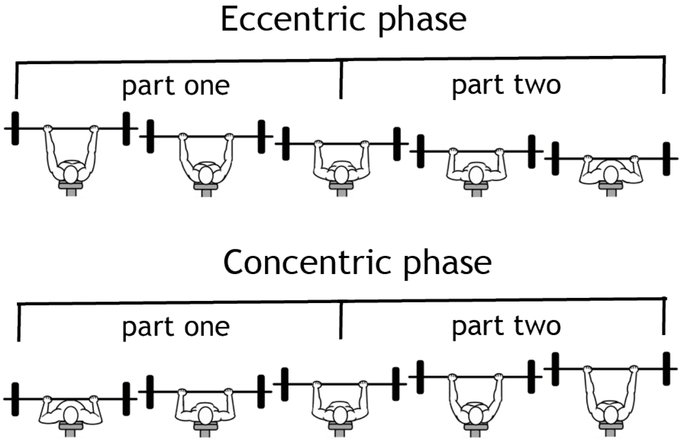
As MI's upper bound depends on the total entropy of the signals involved, comparisons between different samples are not feasible. For this reason, in the field of sEMG research MI is commonly normalized through the following equation:[15]

$$NMI(X,Y) = \frac{MI(X,Y)}{min(H(X),H(Y))}$$

The resulting value is a positive number between 0 (stochastic independence) and 1 (functional dependency).[15]

The length of the moving window used to calculate the NMI was set to 250 ms to preserve a high degree of stationarity during dynamic muscle contractions,[36] the overlap between two calculation steps was 80 %. The concentric and eccentric phase of the 3RM test's first repetition were analyzed separately to avoid obtaining an NMI value containing parts of both phases. Bar position data was used to track whether the instant represented by the center of the moving window corresponded to the first or the second half of each phase. All values of each half were then averaged, yielding a total of four NMI values per subject, test session and muscle pair. Figure 2 illustrates the division of the BP cycle.

Figure 2: BP cycle division



The instance where the barbell reached 50% of its vertical displacement served as delimiter of the two parts of each phase.

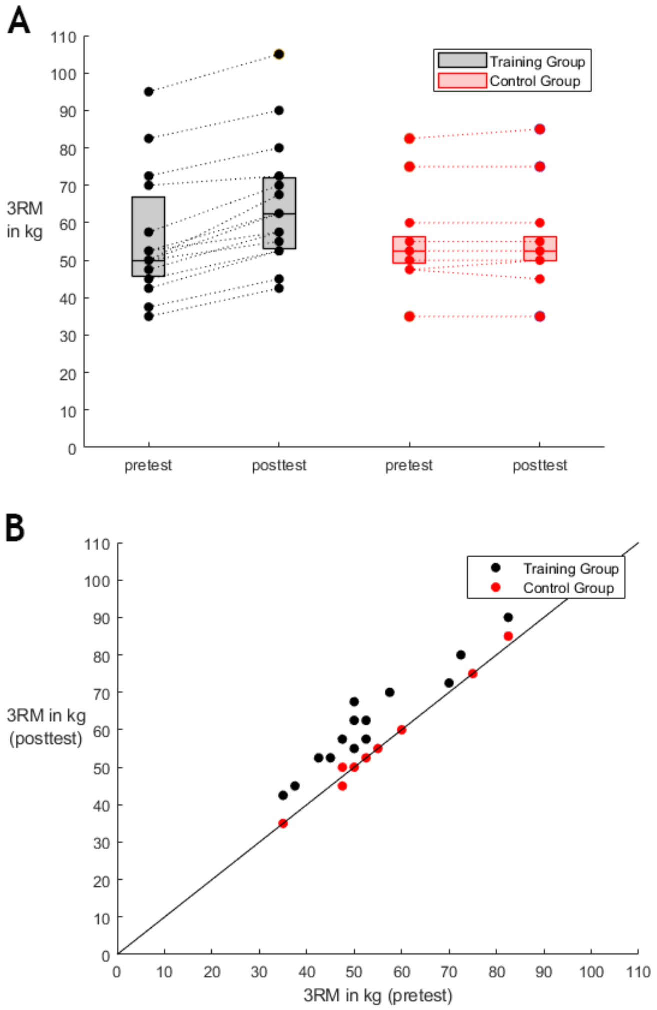
The further division of eccentric and concentric phases reflects previous results of studies on the BP, which show that sEMG amplitude curves of the muscles involved do not remain constant, but instead vary throughout the eccentric and concentric phase.[253137] Analyzing the complete length of each phase might lead to an underestimation of subphase-specific effects by averaging them out. For this reason, two parts of equal barbell displacement distance were created.

Statistics: All statistical tests were conducted using SPSS (Version 27, IBM, Armonk, USA). Visual inspection of Q-Q plots revealed that the NMI data followed a normal distribution. Statistical significance was tested via a two-factor ANOVA with repeated measures of the second factor (group × time). As the experimental setup included only two groups, estimated marginal means (EMM) were used in lieu of a post-hoc test. An level of $p = 0.05$ was selected as threshold of statistical significance. Partial eta squared (η_p^2) was calculated as measure of effect size. G*Power (Version 3.1.9.7, Universität Düsseldorf, Düsseldorf, Germany) was employed to perform post-hoc calculations of statistical power.

RESULTS

3RM: Figure 3 shows the evolution of the 3RM values for TRA and CON.

Figure 3: Evolution of the subjects' 3RM values

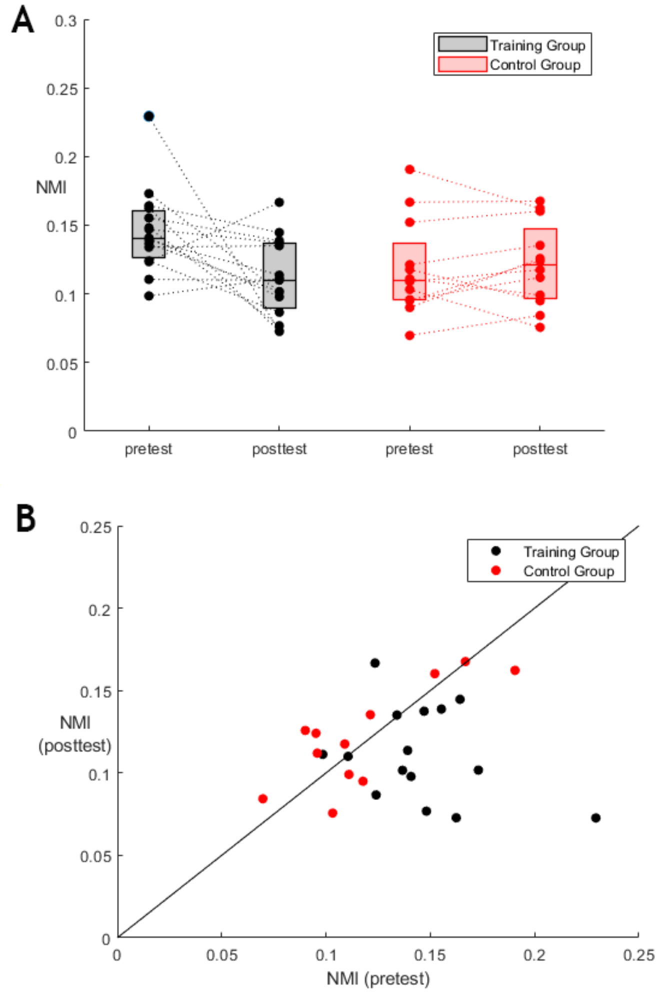


Boxes in Figure 2A represent the interquartile range (band is placed at the median), dots in 2A and 2B represent individual subjects

After the five-week ST program, TRA improved their BP 3RM from 56kg ± 16.87kg (pre test, mean and standard deviation) to 64.83kg ± 16.86kg (post test). No comparable increase was observed in CON (pre test: 55kg ± 12.12kg, post test: 55.19kg ± 12.64kg). A two-way repeated measures ANOVA showed that that the interaction *group* × *time* was statistically significant (F = 66.242,p <0.001). The p-values for the EMM comparisons were p=0.355 for *group* and p<0.001 for *time*. η_p^2 was 0.718, and the statistical power value reached $(1\text{-}\beta) = 1$.

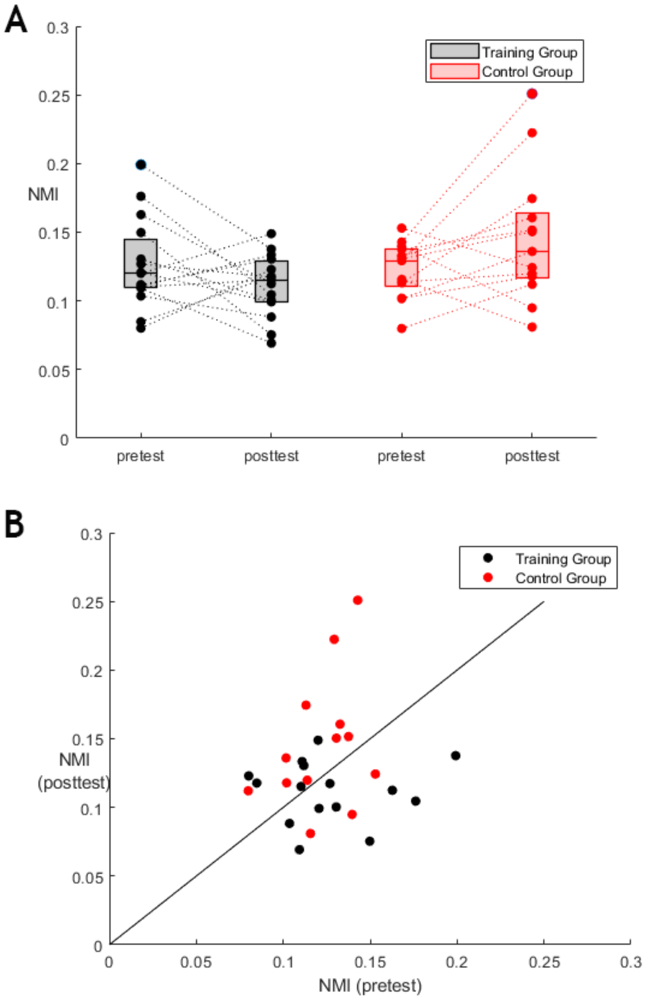
NMI: Data on the evolution of all NMI values of the two groups can be found in Table 3 (agonist-agonist muscle pairs) and Table 4 (agonist-antagonist muscle pairs and the lone antagonist-antagonist muscle pair) on pages 8-9. In total, sixty muscle pair - movement phase combinations (MPMPCs) were analyzed. A significant *group* × *time* interaction (p<0.05) was found in two of them, i.e. *anterior deltoid - biceps brachii* in the first part of the concentric phase (AD-BB), and *anterior deltoid - triceps brachii (medial head)* (AD-TBM) in the first part of the eccentric phase. For AD-BB, the α value was $p = 0.019$. EMM significance values were $p = 0.046$ for *time* and $p = 0.377$ for *group*. The effect size reached $\eta_p^2 = 0.2$, and the result of the post-hoc calculation of statistical power was $(1\text{-}\beta)$=0.965. For AD-TBM, the α value was $p = 0.024$, and EMM significance values reached $p = 0.599$ for *time* and $p = 0.119$ for *group*. The effect size was $\eta_p^2 = 0.181$, and the result of the post-hoc calculation of statistical power was $(1\text{-}\beta)$=0.942. Figure 4 (*anterior deltoid - biceps brachii*, first part of the concentric phase) and Figure 5 (*anterior deltoid - triceps brachii (medial head)*, first part of the eccentric phase) on the next page provide more detailed information on the evolution of the subjects' NMI values for the two MPMPCs where the interaction *group* × *time* was statistically significant.

Figure 4: Evolution of the NMI values of *anterior deltoid - biceps brachii*, first part of the concentric phase



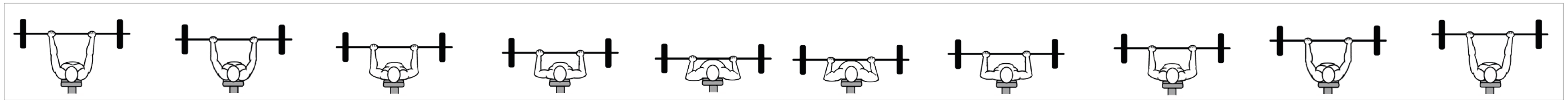
Boxes in Figure 4A represent the interquartile range (band is placed at the median), dots in 4A and 4B represent individual subjects. One outlier in CON with a posttest NMI value of 0.426 was removed from the data set.

Figure 5: Evolution of the NMI values of *anterior deltoid - triceps brachii (medial head)*, first part of the eccentric phase



Boxes in Figure 5A represent the interquartile range (band is placed at the median), dots in 5A and 5B represent individual subjects.

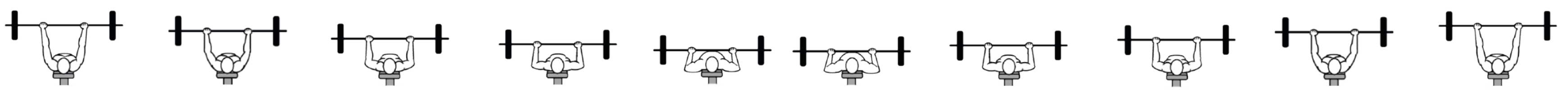
Table 3: NMI values of agonist-agonist muscle pairs



	Eccentric phase - part 1				Eccentric phase - part 2				Concentric phase - part 1				Concentric phase - part 2			
		Pre test	Post test			Pre test	Post test			Pre test	Post test			Pre test	Post test	
Pectoralis major - Anterior deltoid	Training group	0.126 ± 0.032	0.148 ± 0.057	F = 0.589; p = 0.45	Training group	0.164 ± 0.066	0.155 ± 0.056	F = 0.155; p = 0.697	Training group	0.118 ± 0.017	0.13 ± 0.026	F = 2.136; p = 0.156	Training group	0.125 ± 0.024	0.107 ± 0.023	F = 0.069; p = 0.795
	Control group	0.136 ± 0.053	0.138 ± 0.061		Control group	0.147 ± 0.023	0.146 ± 0.034		Control group	0.129 ± 0.039	0.121 ± 0.032		Control group	0.139 ± 0.034	0.118 ± 0.024	
Pectoralis major - Triceps (long head)	Training group	0.125 ± 0.022	0.122 ± 0.025	F = 0.294; p = 0.592	Training group	0.121 ± 0.036	0.132 ± 0.024	F = 2.05; p = 0.164	Training group	0.128 ± 0.02	0.114 ± 0.028	F = 0.29; p = 0.595	Training group	0.118 ± 0.022	0.111 ± 0.02	F = 0.42; p = 0.523
	Control group	0.131 ± 0.03	0.12 ± 0.03		Control group	0.131 ± 0.027	0.123 ± 0.028		Control group	0.121 ± 0.029	0.115 ± 0.029		Control group	0.126 ± 0.039	0.128 ± 0.027	
Pectoralis major - Triceps (medial head)	Training group	0.123 ± 0.036	0.12 ± 0.022	F = 0.135; p = 0.717	Training group	0.129 ± 0.035	0.137 ± 0.018	F = 2.191; p = 0.151	Training group	0.112 ± 0.017	0.111 ± 0.022	F = 1.098; p = 0.304	Training group	0.119 ± 0.028	0.111 ± 0.028	F = 0.076; p = 0.785
	Control group	0.13 ± 0.04	0.133 ± 0.041		Control group	0.124 ± 0.033	0.11 ± 0.026		Control group	0.124 ± 0.02	0.111 ± 0.022		Control group	0.116 ± 0.028	0.111 ± 0.049	
Anterior deltoid - Triceps (long head)	Training group	0.138 ± 0.051	0.119 ± 0.049	F = 3.708; p = 0.065	Training group	0.127 ± 0.031	0.133 ± 0.024	F = 0.003; p = 0.953	Training group	0.117 ± 0.015	0.112 ± 0.017	F = 1.04; p = 0.317	Training group	0.127 ± 0.031	0.107 ± 0.023	F = 0.368; p = 0.549
	Control group	0.121 ± 0.02	0.133 ± 0.033		Control group	0.14 ± 0.029	0.145 ± 0.035		Control group	0.12 ± 0.02	0.123 ± 0.027		Control group	0.124 ± 0.026	0.122 ± 0.02	
Anterior deltoid - Triceps (medial head)	Training group	0.126 ± 0.033	0.112 ± 0.023	F = 5.731; **p = 0.024***	Training group	0.137 ± 0.018	0.144 ± 0.022	F = 0.084; p = 0.774	Training group	0.111 ± 0.022	0.119 ± 0.018	F = 1.504; p = 0.231	Training group	0.125 ± 0.024	0.113 ± 0.027	F = 3.05; p = 0.093
	Control group	0.122 ± 0.02	0.146 ± 0.048		Control group	0.129 ± 0.019	0.14 ± 0.032		Control group	0.129 ± 0.034	0.122 ± 0.031		Control group	0.139 ± 0.034	0.118 ± 0.025	
Triceps (long head) - Triceps (medial head)	Training group	0.177 ± 0.077	0.147 ± 0.074	F = 2.315; p = 0.14	Training group	0.159 ± 0.058	0.187 ± 0.104	F = 0.233; p = 0.633	Training group	0.161 ± 0.068	0.147 ± 0.073	F = 1.215; p = 0.28	Training group	0.143 ± 0.051	0.153 ± 0.085	F = 0.583; p = 0.452
	Control group	0.194 ± 0.115	0.226 ± 0.139		Control group	0.181 ± 0.116	0.236 ± 0.157		Control group	0.173 ± 0.094	0.197 ± 0.109		Control group	0.166 ± 0.097	0.207 ± 0.129	

Data presented as mean and standard deviation. Subphases are based on barbell displacement data. '' denotes statistical significance at $p<0.05$. EMM values for* anterior deltoid - triceps (medial head), eccentric phase, part 1 *were $p=0.599$ for* time *and $p=0.119$ for* group. Effect size was $\eta_p^2 = 0.181$, statistical power was $(1-\beta)=0.942$.

Table 4: NMI values of agonist-antagonist muscle pairs and the lone antagonist-antagonist muscle pair



	Eccentric phase - part 1				Eccentric phase - part 2				Concentric phase - part 1				Concentric phase - part 2			
		Pre test	Post test			Pre test	Post test			Pre test	Post test			Pre test	Post test	
Pectoralis major - Latissimus dorsi	Training group	0.112 ± 0.022	0.134 ± 0.036	F = 3.938; p = 0.058	Training group	0.127 ± 0.038	0.139 ± 0.032	F = 1.176; p = 0.288	Training group	0.113 ± 0.032	0.118 ± 0.02	F = 0.713; p = 0.406	Training group	0.109 ± 0.033	0.127 ± 0.027	F = 4.097; p = 0.053
	Control group	0.117 ± 0.023	0.11 ± 0.033		Control group	0.121 ± 0.019	0.117 ± 0.022		Control group	0.112 ± 0.02	0.126 ± 0.015		Control group	0.13 ± 0.018	0.121 ± 0.025	
Pectoralis major - Biceps	Training group	0.13 ± 0.04	0.128 ± 0.061	F = 0.329; p = 0.571	Training group	0.133 ± 0.023	0.125 ± 0.027	F = 1.746; p = 0.198	Training group	0.118 ± 0.024	0.122 ± 0.031	F = 0.194; p = 0.663	Training group	0.115 ± 0.035	0.11 ± 0.028	F = 0.007; p = 0.932
	Control group	0.123 ± 0.057	0.135 ± 0.029		Control group	0.123 ± 0.021	0.133 ± 0.017		Control group	0.123 ± 0.027	0.121 ± 0.039		Control group	0.124 ± 0.043	0.12 ± 0.029	
Anterior deltoid - Latissimus dorsi	Training group	0.133 ± 0.031	0.112 ± 0.024	F = 2.825; p = 0.105	Training group	0.143 ± 0.039	0.118 ± 0.018	F = 1.468; p = 0.237	Training group	0.127 ± 0.024	0.121 ± 0.027	F = 1.349; p = 0.256	Training group	0.135 ± 0.041	0.134 ± 0.028	F = 0.009; p = 0.924
	Control group	0.112 ± 0.022	0.118 ± 0.042		Control group	0.132 ± 0.026	0.126 ± 0.029		Control group	0.123 ± 0.041	0.134 ± 0.034		Control group	0.128 ± 0.032	0.125 ± 0.025	
Anterior deltoid - Biceps	Training group	0.116 ± 0.024	0.102 ± 0.029	F = 0.053; p = 0.819	Training group	0.141 ± 0.035	0.136 ± 0.037	F = 0.004; p = 0.952	Training group	0.146 ± 0.031	0.111 ± 0.028	F = 6.131; **p = 0.019***	Training group	0.136 ± 0.041	0.113 ± 0.03	F = 3.415; p = 0.076
	Control group	0.131 ± 0.043	0.121 ± 0.029		Control group	0.155 ± 0.028	0.149 ± 0.037		Control group	0.119 ± 0.035	0.122 ± 0.031		Control group	0.121 ± 0.046	0.138 ± 0.044	
Triceps (long head) - Latissimus dorsi	Training group	0.118 ± 0.022	0.14 ± 0.037	F = 2.405; p = 0.133	Training group	0.13 ± 0.026	0.142 ± 0.032	F = 3.776; p = 0.063	Training group	0.128 ± 0.028	0.112 ± 0.026	F = 0.704; p = 0.409	Training group	0.122 ± 0.034	0.117 ± 0.029	F = 0.69; p = 0.414
	Control group	0.131 ± 0.028	0.126 ± 0.036		Control group	0.142 ± 0.04	0.117 ± 0.045		Control group	0.148 ± 0.052	0.12 ± 0.027		Control group	0.123 ± 0.023	0.132 ± 0.03	
Triceps (long head) - Biceps	Training group	0.126 ± 0.03	0.112 ± 0.051	F = 0.106; p = 0.747	Training group	0.139 ± 0.024	0.13 ± 0.039	F = 0.119; p = 0.733	Training group	0.169 ± 0.057	0.165 ± 0.08	F = 0.035; p = 0.853	Training group	0.201 ± 0.081	0.188 ± 0.102	F = 0.282; p = 0.6
	Control group	0.135 ± 0.053	0.128 ± 0.032		Control group	0.149 ± 0.032	0.134 ± 0.028		Control group	0.157 ± 0.064	0.149 ± 0.049		Control group	0.165 ± 0.051	0.17 ± 0.075	
Triceps (medial head) - Latissimus dorsi	Training group	0.118 ± 0.035	0.126 ± 0.031	F = 0.103; p = 0.751	Training group	0.121 ± 0.047	0.126 ± 0.041	F = 0.025; p = 0.876	Training group	0.125 ± 0.04	0.12 ± 0.026	F = 0.172; p = 0.682	Training group	0.111 ± 0.022	0.137 ± 0.053	F = 0.663; p = 0.423
	Control group	0.115 ± 0.031	0.129 ± 0.059		Control group	0.123 ± 0.022	0.124 ± 0.041		Control group	0.125 ± 0.052	0.129 ± 0.024		Control group	0.127 ± 0.035	0.137 ± 0.028	
Triceps (medial head) - Biceps	Training group	0.122 ± 0.039	0.13 ± 0.049	F = 0.037; p = 0.848	Training group	0.134 ± 0.044	0.138 ± 0.054	F = 0.17; p = 0.683	Training group	0.157 ± 0.058	0.163 ± 0.089	F = 0.246; p = 0.642	Training group	0.132 ± 0.058	0.141 ± 0.073	F = 1.188; p = 0.286
	Control group	0.13 ± 0.031	0.134 ± 0.034		Control group	0.129 ± 0.033	0.123 ± 0.017		Control group	0.136 ± 0.05	0.129 ± 0.041		Control group	0.118 ± 0.044	0.167 ± 0.109	
Latissimus dorsi - Biceps	Training group	0.112 ± 0.023	0.117 ± 0.053	F = 0.208; p = 0.652	Training group	0.127 ± 0.033	0.137 ± 0.033	F = 2.746; p = 0.109	Training group	0.125 ± 0.034	0.114 ± 0.029	F = 0.332; p = 0.569	Training group	0.101 ± 0.029	0.136 ± 0.04	F = 0.837; p = 0.369
	Control group	0.122 ± 0.031	0.119 ± 0.022		Control group	0.146 ± 0.029	0.13 ± 0.024		Control group	0.166 ± 0.68	0.145 ± 0.065		Control group	0.124 ± 0.028	0.146 ± 0.042	

Data presented as mean and standard deviation. Subphases are based on barbell displacement data. ’’ denotes statistical significance at* $p < 0.05$. *EMM values for* anterior deltoid - biceps brachii, concentric phase, part 1 *were p=0.046 for* time *and p=0.377 for* group. Effect size was $\eta_p^2 = 0.2$, statistical power was $(1-\beta)=0.965$.

Out of 60 total outcome measurements (15 muscle pairs, two movement phases, two subphases), 30 belonged to the eccentric and 30 to the concentric phase of the lift. For TRA's pretest and posttest, Tables 5 and 6 show the ten MPMPCs that recorded the highest NMI values.

Table 5: Highest recorded NMI values during the pre- and posttest of TRA (eccentric phase)

Training group - Pretest			Training group - Posttest		
Muscle pair	**Half**	**NMI**	**Muscle pair**	**Half**	**NMI**
Triceps (long head) - Triceps (medial head)	1st	0.177 ± 0.077	Triceps (long head) - Triceps (medial head)	2nd	0.187 ± 0.104
Pectoralis major - Anterior deltoid	2nd	0.164 ± 0.066	Pectoralis major - Anterior deltoid	2nd	0.155 ± 0.056
Triceps (long head) - Triceps (medial head)	2nd	0.159 ± 0.058	Pectoralis major - Anterior deltoid	1st	0.148 ± 0.057
Anterior deltoid - Latissimus dorsi	2nd	0.143 ± 0.039	Triceps (long head) - Triceps (medial head)	1st	0.147 ± 0.074
Anterior deltoid - Biceps	2nd	0.141 ± 0.035	Anterior deltoid - Triceps (medial head)	2nd	0.144 ± 0.022
Triceps (long head) - Biceps	2nd	0.139 ± 0.024	Triceps (long head) - Latissimus dorsi	2nd	0.142 ± 0.032
Anterior deltoid - Triceps (long head)	1st	0.138 ± 0.051	Triceps (long head) - Latissimus dorsi	1st	0.140 ± 0.037
Anterior deltoid - Triceps (medial head)	2nd	0.137 ± 0.018	Pectoralis major - Latissimus dorsi	2nd	0.139 ± 0.033
Triceps (medial head) - Biceps	2nd	0.134 ± 0.044	Triceps (medial head) - Biceps	2nd	0.138 ± 0.054
Pectoralis major - Biceps	2nd	0.133 ± 0.023	Pectoralis major - Triceps (medial head)	2nd	0.137 ± 0.018

Data presented as mean and standard deviation. Half *refers to the subphase, established according to barbell displacement data.*

Table 6: Highest recorded NMI values during the pre- and posttest of TRA (concentric phase)

Training group - Pretest			Training group - Posttest		
Muscle pair	**Half**	**NMI**	**Muscle pair**	**Half**	**NMI**
Triceps (long head) - Biceps	2nd	0.201 ± 0.081	Triceps (long head) - Biceps	2nd	0.188 ± 0.102
Triceps (long head) - Biceps	1st	0.169 ± 0.057	Triceps (long head) - Biceps	1st	0.165 ± 0.08
Triceps (long head) - Triceps (medial head)	1st	0.161 ± 0.068	Triceps (medial head) - Biceps	1st	0.163 ± 0.089
Triceps (medial head) - Biceps	1st	0.157 ± 0.058	Triceps (long head) - Triceps (medial head)	2nd	0.153 ± 0.085
Anterior deltoid - Biceps	1st	0.146 ± 0.031	Triceps (long head) - Triceps (medial head)	1st	0.147 ± 0.073
Triceps (long head) - Triceps (medial head)	2nd	0.143 ± 0.051	Triceps (medial head) - Biceps	2nd	0.141 ± 0.073
Anterior deltoid - Biceps	2nd	0.136 ± 0.041	Triceps (medial head) - Latissimus dorsi	2nd	0.137 ± 0.053
Anterior deltoid - Latissimus dorsi	2nd	0.135 ± 0.041	Biceps - Latissimus dorsi	2nd	0.136 ± 0.04
Triceps (medial head) - Biceps	2nd	0.132 ± 0.058	Anterior deltoid - Latissimus dorsi	2nd	0.134 ± 0.028
Triceps (long head) - Latissimus dorsi	1st	0.128 ± 0.028	Pectoralis major - Anterior deltoid	1st	0.13 ± 0.026

Data presented as mean and standard deviation. Half *refers to the subphase, established according to barbell displacement data.*

DISCUSSION

The main finding of this study is that, contrary to the initial hypotheses, no significant pre- to posttest changes took place in 58 of 60 MPMPCs. This complements the work by Kristiansen et al.,[?] who found that the same training intervention did not affect muscle synergies in a significant manner. Muscle synergy analysis is a very holistic approach to quantify intermuscular coordination, which looks at the overall movement pattern and not at interactions between muscle pairs. The present results indicate that the 5 weeks of BP training did not alter intermuscular coordination patterns at the muscle pair level, either. The subjects' significant increase in strength, measured by their 3RM values, must thus be due to other training-induced adaptations. Recently, Del Vecchio et al.[38] used MU population data to demonstrate that increases in MU firing rate and changes in the MU recruitment threshold occurred after only 4 weeks of ST. As changes in intermuscular coordination patterns are largely absent, these factors provide a more likely explanation for the strength increase experimented by this study's subjects.

As the 3RM test measures concentric strength, it is important to point out that changes in a MPMPC that includes the first part of the eccentric phase should not be assumed to have influenced performance levels, as there is no plausible mechanism of how this could have happened. The opposite is true for *anterior deltoid - biceps brachii (first part of the concentric phase)*. As stated earlier in the explanation of the hypotheses, it is argued that strength-trained individuals display less agonist-antagonist coactivation during maximal voluntary contractions,[397] although results showing this effect do not replicate reliably.[7] As BB acts as antagonist during the concentric phase of the BP, TRA's reduction in NMI values between AD and BB support this theory. Interestingly, the pre- to posttest evolution of the same muscle pair's NMI value during the second part of the concentric phase showed the same tendency, but did not reach statistical significance.

Among the previous studies on this effect, only few have examined dynamic contractions during free weight barbell exercises, which are considered to place a particularly high demand on coordination and movement stabilization.[4] Stock et al.[40] found no change in the agonist-antagonist coactivation patterns of previously untrained women that had undergone 4 weeks of squat and deadlift training. Stock and Thompson[41] reported insignificant changes of antagonist activity after 10 weeks of deadlift training, but also stated that there was a low level of similarity between training and testing tasks. The same is true for Arabatzi and Kellis' work,[42] who documented that 8 weeks of olympic weightlifting or traditional weightlifting training differ in their effects on the level of antagonist coactivation measured during a vertical leap test. There is a variety of possible explanations for the partial disagreement of the present study's findings with the aforementioned publications. As subjects trained with sets of 3 BP repetitions in the last week of training, the agreement between training and testing task is near total, in contrast to the works of Stock and Thompson[41] and Arabatzi and Kellis,[42] who furthermore used recreationally trained and not ST-naive subjects. The disagreement with Stock et al.'s[40] findings could be due to sex differences is motor strategies, or due to differences between upper-body and lower-body adaptations. More, it must be noted that all studies mentioned here have used correlation-based indices to quantify the degree of agonist-antagonist coactivation, which are measures that are insensitive to any kind of nonlinear relationships.

In all phases, the muscle pairs located in the upper arm are among the pairs that record the highest NMI values. This is unsurprising, given that these muscles routinely act in conjunction during the fine tuning of elbow flexion and extension movements.[43] These preexisting neural connections and motor patterns could also be the reason why the AD-BB muscle pair is more affected by training than TBL-BB or TBM-BB, in which BB is also the antagonist. Since the untrained subjects do not have any experience with BP movement patterns, they tackle the novel task using the motor repertoire they dispose of. Depending on the muscle pair in question, the corresponding neural patterns may be more or less stable, and may vary in their response to training stimuli.

Apart from TBL, TBM and BB, AD is also part of several high-ranking MPMPCs, in combination with trunk muscles like PM and LD, but also with TB and BB. This mirrors AD's anatomical location, linking trunk and upper extremities. Regarding the division into subphases, it is striking that the second half of the BP's eccentric movement phase is overrepresented in the list of highest recorded NMI values if compared to the first half. A possible biomechanical explanation for this pattern is that during the eccentric phase's first part, the subjects limit themselves to control the speed with which the bar moves downward, while in the second part, the need to bring it to a complete halt requires more muscular work and more concerted contractions.

TRA's significant increase of BP 3RM (15.8% from pre- to posttest) is in line with the results of similar intervention-based studies on the effect of ST in novice subjects. Both Paulsen et al.[44] and Candow and Burke[45] included the BP in a 6-week full-body ST program for untrained participants and the corresponding pre- and posttests. Comparing two training groups, Paulsen et al. found BP 1RM increases of 14% and 16%,[44] while Candow and Burke reported improvements of 22% and 30%.[45] These findings underline the proven efficiency of ST as a method to improve strength, and confirm that previously untrained individuals are able to increase their strength rapidly after the onset of a ST program.[1]

The design as RCT is one of the strengths of the present study. Further, the relatively short duration of the ST protocol limits the development of muscle hypertrophy in TRA,[6] which is another important contributor to increased muscular strength[46] and, if neural adaptations to ST are to be investigated, a potential confounding factor. Considering that the subjects were untrained, fairly high loads and low numbers of repetitions were used in the BP sets, again putting emphasis on provoking neural training effects.[47] During the last week of the ST intervention, subjects trained with three repetitions per set, while the pre- and posttest was a 3RM effort. The high degree of similarity between training and test tasks is an important feature of the study design, since a significant amount of the initial neural adaptations to ST are task-specific.[2]

The short duration of this study's training program can also be viewed as a limitation if a different perspective is taken. After a total of fifteen sessions, only the earliest effects of ST can be observed. Further, any examination of weightlifting movements with very high loads deals with the limitation that only a small number of repetitions are available for analysis. The ensuing increase in variability between samples was already discussed earlier. Lastly, since the present study's population was comprised of male subjects only, it is unclear whether the findings would also apply to untrained females, as research on sex differences in strength development has so far yielded heterogeneous results.[48]

CONCLUSION

The present study examined whether an increase of intermuscular coordination, measured via NMI of muscle pairs formed by the BP's main agonists and antagonists, contributed to the early gains in muscular strength of untrained male subjects. While the evolution of one MPMPC hinted at a reduction of BB co-contraction during the lifts concentric phase, the fact that no significant changes were found in the overwhelming majority of MPMPCs makes a strong case against this assumption. This means that the marked increase in 3RM strengths is due to other factors.

After a few weeks of ST, the development of hypertrophy becomes a larger contributor to beginners' strength gains if compared to neural factors. However, neural adaptations to ST continue to play a role in the performance trajectory of individuals that seek to excel in muscular strength, e.g. elite athletes. The present findings indicate that changes in intermuscular coordination patterns may change slower and more gradual than MU firing rate or MU recruitment threshold, where changes can be detected in novice subjects after a few weeks of training. To track the evolution of intermuscular coordination, future works that opt for an intervention-based approach could increase the length of the ST program, even if this comes at the price of concurrent muscular hypertrophy. Cross-sectional studies could include populations of recreationally trained, but non-elite athletes whose strength levels are at an intermediate stage between the untrained and the experts, and examine whether their intermuscular coordination patterns mirror the *middle ground* status of their strength levels.

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DEDICATION

Dedicated to the loving memory of Michael Skoruppa.
⋆27.09.1942 †19.06.2021

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Accompanying Worksheet

to the thesis submitted by

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This worksheet provides additional material to the article it accompanies. It contains two chapters that deal with the current state of research on neural adaptations to strength training (Chapter 1) and the bench press exercise (Chapter 2), containing texts that were not included entirely in the article for reasons of scope.

1. Neural adaptations to strength training

1.1. Introduction

Adaptation to an external stimulus is the mechanism that enables humans to train their bodies purposefully (Zatsiorsky et al., 2021: 3), whether it be to pursue athletic goals, to improve health outcomes, or for recreation. As strength is defined as the capacity to exert muscular force (Harman, 1993: 18), the term *strength training* in its broadest sense may refer to any planned activity that aims to increase strength through physical exercise and the ensuing adaptations. To laypersons, visible increases in muscle volume are the most prominent illustration of the adaptive mechanisms provoked by ST. However, training stimuli also leave their marks on the nervous system, which are invisible from the outside and more difficult to detect. Nevertheless, said adaptations have been known to physiologists and sports scientists for several decades. Several observations have led to the conclusion that neural adaptations contribute significantly to the strength increase attainable through training. One of them is the phenomenon of *cross-transfer*, an improvement in strength of untrained limbs that is provoked by exposing their contra-lateral counterpart to training (Gabriel et al., 2006: 139). Secondly, the fact that strength gains manifest themselves earlier in a training program than muscle hypertrophy has been interpreted as an indirect proof of neural contributions (ibid.: 133). A less frequently stated variant of this argument is that prepubescent children also become stronger through ST, despite showing far less muscle hypertrophy than adults (Faigenbaum et al., 2009: S64). Further, significant parts of the improvements in strength that occur after the onset of ST are specific to the exercise used while training and do not transfer to other exercises that involve the same muscle groups (Škarabot et al., 2020: 675-676). Consequently, this has evoked the assumption that neural factors play a role in the development of muscular strength, similar to their role in the acquisition of motor skills.

Several decades of scientific investigation have refined our understanding of the nervous system’s reaction to ST stimuli. The present chapter will attempt to outline the current state of research on the matter, following a five-step approach: Firstly, the parts of the nervous system that are involved in motor control will be recalled in brevity. Next, subchapter 1.3 will explain the different techniques and methods employed in ST research and how variation in measurement techniques, training programs, study population and other aspects may influence study outcomes. This does not only aim to provide a foundation for the following subchapters, but is also necessary to put the study at the heart of this thesis into context. Thereafter, it will be laid out how ST changes the properties of the signal that arrives at the neuromuscular junction, thus categorizing adaptive effects according to their result. In contrast, subchapter 1.5 focuses on the origins by addressing possible anatomical locations of the previously described modifications. Lastly, some brief concluding remarks form the last part of chapter 1. Throughout this chapter, the key concepts of a paragraph or section will appear in bold font.

1.2. Anatomical structures involved in voluntary muscle contractions

1.2.1. Structures belonging to the nervous system

Both the central and the peripheral nervous system are active during the execution of a motor pattern. The next paragraphs will comment on the specific anatomical structures involved by following the motor signal along its path from the brain towards the muscles. The signal itself consists of a cell membrane's electric potential that travels along the length of the neuron. This electric potential is due to the difference in ion concentration on the two sides of the cell membrane (thus, inside and outside of the cell), which can be regulated by the motor neuron through four different ion channels and receptors (Enoka & Duchateau, 2016: 5-6). Researchers studying the neuromuscular system seek to understand the signal's pathways and characteristics by recording and analyzing it using the methods and techniques described in Chapter 1.3.1.

The **primary motor cortex (M1)** is the most important brain region in the execution of motor tasks (Burdet et al., 2013: 31). It is connected to several other brain areas, such as the pre-motor area, the secondary motor area, the cerebellum and the basal ganglia (ibid: 30-31 & Bhattacharjee et al., 2021: 259), which are involved in the planning and preparation of movement and provide information on the movement's context through sensory feedback (Burdet et al., 2013: 32). The different areas of M1 contain neurons that are connected to a specific body part, giving rise to a so-called *brain map* of motor control (ibid: 31). Although newer research suggests that M1 may contribute to more brain processes than motor execution, its main role consists in providing motor signal output (Bhattacharjee et al., 2021: 258-259). Said signal travels along the bundles of **upper motor neurons**, through the brain stem and the medulla oblongata's pyramids, before *decussating* (crossing the sagittal plane) and moving further down the spine, mainly in the corticospinal tract (Waxmann, 2017: 520).

The signal's transmission to the **lower motor neurons**, which directly connect to the muscle, happens in the spinal cord. While there is evidence for the existence of direct connections between upper and lower motor neurons (thus enabling the signal to travel from M1 to the nervous system's periphery crossing only one synapse), upper motor neurons connect to spinal interneurons as well (Nielsen, 2016: 83-85). Neuron networks in the spinal grey matter are responsible for the integration of sensory information provided by afferent neurons (connected to e.g. the muscle spindles and the Golgi tendon organs) and the descending signal from M1, modifying the resulting overall output signal to the muscles (ibid.: 87-88). These integration processes are very complex and still not well understood. Spinal networks are hypothesized to play a role in the generation of rhythmic movement patterns via a network structure called central pattern generator (CPG), and in coordinating the interplay of agonist, synergist and antagonist muscle contractions (ibid.: 88-91).

The lower motor neurons originate in the ventral horn of the spinal cord's grey matter and terminate at the skeletal muscles (Cramer & Darby, 2013: 401). Their somas and dendrites are located within the spine, whereas their axons exit it in bundles (Burdet et al., 2013: 19). The cervical and lumbar enlargement are the two respective zones where the upper and lower extremities' motor nerves debranch from the spinal cord (Cramer & Darby, 2013: 7). The exit points of the lower motor neurons are considered the border between the central and the peripheral nervous system (ibid.: 65). As the motor neurons approach the muscle they innervate, more and more branches form (Burdet et al., 2013: 20). The basic functional unit of the neuromuscular system is called **motor unit (MU)** and consists of a single motor neuron and the set of all muscle cells innervated by that neuron (Enoka & Duchateau, 2016: 1-3). The group of a single MU's muscle cells is called **muscle unit** (ibid.). The **innervation number**, which indicates the number of muscle fibers in a single MU, varies considerably within the same muscle and is the main determinant of the force a MU can produce (Enoka & Duchateau, 2019: 130). The **motor neuron pool** is the sum of all MUs that correspond to a single muscle and ranges from below 100 for smaller muscles to thousands for bigger ones (Enoka & Duchateau, 2016: 2). Within the motor neuron pool, the distribution of the MUs' force generation capacities is distributed exponentially (Enoka & Duchateau, 2019: 130). According to the so-called size principle (or Henneman principle), the lower recruitment threshold of smaller motor neurons causes them to be recruited first, while the biggest motor neurons of a given muscle are only recruited if a high amount of force or a fast contraction is needed (Enoka & Duchateau, 2016: 6-8). For most muscles this recruitment process peaks at about 85% of its maximal voluntary contraction (MVC) intensity, more intense contractions are accomplished by increasing the motor neuron discharge (or *firing*) rate (Duchateau et al., 2006: 1767). Figure 1.1. on the next page illustrates the lower motor neuron's role as final common pathway of the motor signal.

Figure 1.1: The lower motor neuron as transmitter of the motor signal

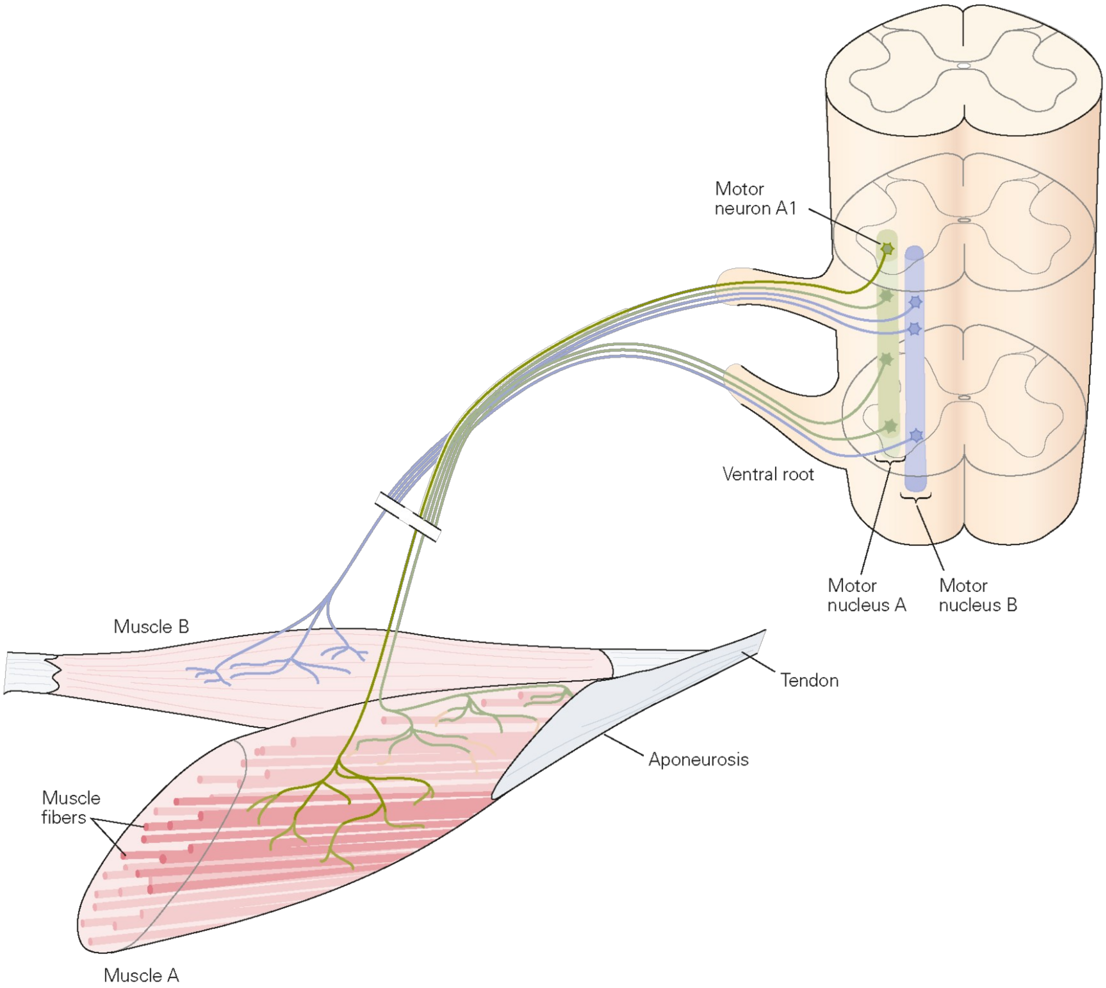

Figure 1.1 depicts two motor units on the bottom part of the image. A number of muscle fibers is innervated by a single lower motor neuron. As we follow along the motor neuron's length towards the spine, we observe that it is joined by many more neurons, forming a nerve. It can be observed that motor nerves insert in the spinal cord, in groups called motor nuclei*. From Enoka (2021: 738).*

1.2.2. Muscle fibers

The motor neuron's endings attach to the muscle fibers at a site called **motor end plate** (Waxmann, 2017: 23-24). Training-induced changes of the muscle fibers' properties do not fall in the category of neural adaptations, but may lead to similar effects in terms of strength gains, which is why attributing training effects to one or the other category poses a considerable methodological challenge (Buckner et al., 2016: 1012-1013). Further, morphological muscle fiber properties influence the surface electromyography (sEMG) signal (Farina et al., 2016: 41). For these reasons, a very brief description of the muscle fibers' characteristics and working mechanisms will be provided in the following.

The fundamental mechanism of muscular force generation is the displacement of microscopic proteic cross-bridges, which is impulsed by the arriving motor signal and transmitted along the muscle cell, acting on the bone via the muscle-tendon-complex (Frontera & Ochala, 2015: 188-189). The maximal amount of force each muscle fiber is able generate is defined by the size of its cross-sectional area and a specific force parameter, which can be expressed in mN/µm^2 (Enoka & Duchateau, 2016: 9). During a contraction, the fiber's current state of contraction or elongation and the contraction or elongation velocity determine the force output as well (ibid.: 12). Scientists divide muscle fibers into several categories, using either the enzyme ATPase or the amino acid sequence of the myosin molecules' heaviest protein as biological markers. The resulting categories depict the fibers' contraction speeds, which correlate with their propensity to fatigue and the maximal force output of the single fibre. However, it is necessary to keep in mind that the contractile properties of different muscle fibers form a spectrum, and that there is considerable overlap between the categories (Enoka & Duchateau, 2018: 130-132). More, hybrid fibers exist (ibid.: 132), and exposure to training may lead to muscle fibers switching their type, albeit to a limited degree (Wilson et al., 2012: 1725-1726).

In pennate muscles, the direction and magnitude of the net force generated by a contracting muscle bundle (or *fascicle*) not only depends on the factors mentioned above, but also on the pennation angle, which is defined as the angle between the fascicle's fibers and the tendon's line of action (Lee et al., 2015: 1474). In the same way, the whole muscle's contribution to joint movement is not a simple sum of the individual bundles' force magnitudes, but the vector sum of all resulting force vectors. The same principle applies for overall joint motion: during voluntary movement muscles rarely act in isolation, so that the observable movement of limbs is usually the product of the coordinated interplay of several muscles (Carroll et al., 2001: 829). One basic principle of said interplay is that the human musculoskeletal system is functionally redundant: a given joint movement could be caused by an infinite number of different muscle activation patterns, and the principles governing the pattern selections observed in practice are still a subject of debate (Hirashima & Oya, 2016: 80-81). This presents a methodological challenge to motor control researchers, since muscle activation cannot be inferred from movement, but must be measured with specialized equipment.

1.3. ST studies and their methodological variation

1.3.1. Parameters or signals being studied and/or employed

Possible neural adaptations to ST are investigated by comparing the change of one or more parameters before and after the supposed adaptation process has taken place. This involves the recording of the nervous system's electrical signals, but may also be accompanied by the tracking performance parameters as a measurement of the neuromuscular system's output. The present subchapter will briefly describe these different parameters and their value in studying the neural response to ST, moving from indirect to more direct measurements.

ST studies often document an increase in the subjects' **motor performance** (McGuigan et al., 2012: 3; Lawton et al., 2011: 415). While the measurable outcome of any athletic discipline could be categorized as such, strength and conditioning researchers employ a narrower set of movements in their pre- and post-tests, such as the one-repetition maximum (1RM) of strength training exercises or measures of power like the countermovement jump (Androulakis-Korakakis et al., 2019: 753; Carlson et al., 2009: 85-87). The interest in the results of this kind of tests is shared by athletes and coaches, since they have been linked to overall performance in various sports (McGuigan et al., 2013: 209-210). However, without additional measurements, it is of course not possible to link improvements in motor performance to neural or non-neural factors. Further, while motor performance and tests of isometric force generation capacity tend to be highly correlated (Lum et al., 2020: 7 & 18-19), these two parameters cannot be used interchangeably, as motor performance tests require force output at a range of joint angles throughout the movement (ibid.: 2 & 27). According to the principle of muscle redundancy mentioned in Chapter 1.2.2, increased motor performance may also be due to switching to a completely different motor pattern altogether. While these kinds of improvements may be very valuable to athletes and coaches, they do not contribute to answering the question of how to improve strength if the subject already uses a near-optimal motor pattern.

Force output can also be measured directly by using setups involving strain gauges, force platforms or dynamometers, which give out the result in N. Usually, this is done as a test of isometric force, by recording the subject's maximal-effort try to move an immovable object. **Maximal voluntary contraction (MVC)** testing is not only used as reference in clinical diagnostics (Bohannon, 2019: 1-2), but is also an often-reported outcome in ST intervention studies (Hortobágyi et al., 2021: 79-81). While one disadvantage of the MVC test is its lack of similarity to the dynamic movements of competitive sports, it entails less safety concerns than the 1RM (Niewiadomski et al., 2008: 115). More importantly, in the scientific context the MVC test is not only conducted to obtain the resultant output force, but is also a prominent auxiliary method in sEMG studies in general, where it is used to normalize measurements, and in nerve conduction studies using reflex waves (see below).

In contrast to both 1RM and MVC, the **sEMG signal** does not measure a visible output of motor action, but the electric potential fields of the muscle fibers' outer membranes, using electrodes (Farina et al., 2016: 30). These potential fields sum up across all active MUs, generating the sEMG signal. While sEMG has proven to be a valuable tool in research on the human motor system, it comes with a set of important limitations and caveats. Firstly, the voltage of said electric potential fields can be positive or negative, so that the parallel presence of a positive and negative field may lead to the two fields partially canceling each other out, yielding a net voltage that misrepresents the corresponding MU's true level of activity (Enoka & Duchateau, 2016: 8). Secondly, to a significant extent, the EMG signal's amplitude is shaped by the properties of the volume conductor, in this case the tissue and skin between the muscle fibers and the sEMG electrode (Farina et al., 2016: 30). More, sEMG recording is sensitive to further influences, e.g. the electrodes' size, angle, inclination and distance relative to the muscle fibers, cross-talk from other muscles' signals, and impedance at the electrode contact site, with the latter in turn depending on factors like skin temperature, the size of the subcutaneous fat layer and humidity (ibid.: 41-43). This means that the sEMG signal's absolute values in mV are not a useful criterion for pre- to post-intervention or subject-to-subject comparisons. Instead, sEMG signal amplitude is usually normalized to its peak value during an isometric MVC test or the execution of a dynamic reference task (Rainoldi et al., 2016: 502). Thus, sEMG amplitude is expressed as a relative value of the subject's personal maximum, usually tested on the same day. Consequently, if the subject improves uniformly across the recorded movement and the reference test, the corresponding sEMG amplitude is unable to reflect these strength gains. Despite these caveats, sEMG is perhaps the most widely-used tool in neuromuscular research, and can provide valuable insights into the muscular activation patterns that underlie the execution of voluntary movement (Vigotsky et al., 2018: 7-10). Recent advances in the technology of Electroencephalography (EEG) have permitted motor control researchers to employ this instrument, traditionally used in cognitive neuroscience and psychology, in a few studies involving dynamic tasks. Despite these advances, movement artifacts and the ensuing signal quality issues remain a considerable challenge (Enders & Nigg, 2015: 417-419). Future improvements in this area could fuel investigations on the coherence between EEG and sEMG signals in highly dynamic athletic movements, which could produce valuable insights into neural adaptation processes caused by ST (ibid.: 421-424).

Typically, sEMG is used to record muscle contractions during voluntary movements. **Peripheral nerve stimulation (PNS)** studies that analyze externally induced muscle activity are a notable exception. In contrast to the generation of a volitional motor signal, the instant the motor system is triggered can be established precisely. If the stimulation signal is applied at a limb, examining the muscles' response provides information on the state of peripheral neurons and spinal circuits (Zehr, 2002: 455-456). In this case, the afferent and efferent neurons are excited simultaneously. The latter relays the stimulation signal directly towards the muscle, while the former transfers it towards the spinal cord, where local interneurons process the afferent input and pass it on to the motor neuron, which causes the connected MUs to become active a second time. The visible sEMG oscillations caused by this experimental setup are called M-wave (caused by the direct stimulation of the efferent neuron) and H-reflex (signal reaching the muscle via afferent neuron, spinal circuitry and efferent neuron). Common measurements reported in reflex studies include the minimal stimulus intensity necessary to evoke H-reflex and M-wave, and the intensity at which they reach their maximal amplitude (ibid.: 455-457). Since the processing of the stimulation signal is done by lower sensory and motor neurons and spinal circuitry, this experimental setups allows researchers to investigate a limited portion of the neuromuscular system in isolation, and to assign a range of anatomical locations to the neural adaptations that underlie changes in the muscular response to the stimuli. A variation of it features the application of the same stimulus during a voluntary muscular effort, typically an isometric MVC trial (Aagaard et al., 2002: 2311). Under these circumstances, the lower motor neuron transmits the signal generated by the subject's effort and the external stimulus, which runs in the opposite direction as the MVC signal. This leads to a *collision* of the two signals that is called the *antidromic effect*, triggering a momentary contraction pause in the corresponding muscle unit. Shortly thereafter, a part of the provoked H-reflex impulse arrives at the muscle fibers, giving rise to an sEMG oscillation called V-wave (for *volitional*) (ibid.). A higher number of active motorneurons during the MVC effort leads to a higher number of *collision sites*, and consequently to a more pronounced reflex impulse arriving at the muscle unit, leading to an increase in V-wave amplitude. As a result, researchers are able to draw conclusions on the efferent output of lower motorneurons and the supraspinal parts of the CNS involved in voluntary muscular contractions, which is why V-wave studies amount to a helpful instrument in motor control and ST investigations (ibid.: 2312).

An external stimulus to the nervous system can also be applied at its central parts, for example through **transcranial magnetic stimulation (TMS)**. Here, an electromagnetic coil placed over the subject's motor cortex triggers upper motor neurons, and the signal follows the full pathway described in Chapter 1.2.1, allowing sEMG electrodes to record the muscular stimulus response. This is known as motor evoked potential (MEP). Depending whether the upper motor neurons are stimulated directly or via interneurons, the resulting signal wavelets are called D-wave or I-wave, respectively (Carroll et al., 2011: 129). Just as PNS, TMS can be applied at rest or during voluntary contractions, whose intensity is typically expressed as percentage of a previously measured MVC. While the TMS method allows for a greater part of the motor signal's overall pathway to be included in studies using external stimuli, this property can be considered a double-edged sword, since the stimulus has to transverse two very complex and highly connected neural structures in M1 and the spinal circuitry. This makes it difficult to assign any observed changes in MEP to a precise anatomical location (ibid.: 130-132). The possibility of invoking the MEP by placing the electromagnetic coil over the medulla oblongata (thereby surpassing M1) is limited because of its propensity to cause pain in the subjects (ibid.). More sophisticated repeated TMS (rTMS) protocols are able to disrupt cortical activity for up to an hour after stimulation and might therefore allow scientists to examine M1 adaptations in a more isolated manner, but have only found limited use in sports and exercise science (ibid.: 133-134).

Since M1 and the areas it connects to are thought to adapt to ST, corresponding longitudinal changes could potentially be detected by **brain imaging**. Functional magnetic resonance imaging (fMRI) is able to depict blood oxygenation states that last for a few seconds or more, permitting experimental setups involving tasks that match or exceed that time span (ibid.: 134). A small number of studies have taken this approach to look for cortical adaptations to ST (ibid.) or for differences between the brain activity of highly motor skilled athletes and a nonathletic control group (Di et al., 2012). However, the fact that initial changes in cortical activity may reverse after further exposure to training and the inability to assign the captured blood oxygenation patterns to excitatory or inhibitory neural circuits are two important caveats to keep in mind when trying to draw conclusions from this kind of investigations (Carroll et al., 2011: 134-135).

1.3.2. Intervention characteristics

The term *neural adaptations* implicitly makes clear that there must be a stimulus the nervous system adapts to. ST practitioners seek to optimize the continuous stimulation to the neuromuscular system in order to help athletes reach their goals, and training programs are as diverse as those goals are. The wide variety of different protocols used in sports science experiments mirrors this diversity, which is why the individual characteristics of each training intervention must be taken into account when its results are interpreted. Consequently, this subchapter will try to shed light on some important intervention parameters and their expected effects on the neural adaptations elicited in the studies' subjects.

The first category of studies are those that do not include any intervention at all: **cross-sectional population studies** carry out group-wise comparisons at a single given moment in time. In sport science, this is often done by recruiting a group of elite athletes and a control group that is completely untrained or only recreationally active. Several physical characteristics of strength athletes have been investigated in this manner, e.g. muscle fiber type distribution (see Tesch and Alkner, 2003: 268 for an overview), respiratory performance (Brown et al., 2013), and neurophysiological properties such as sEMG frequency power spectrum (Moritani and Muro, 1987) or reaction to an externally applied stimulus (Kotzamanidis et al., 1997). Since athletes train for years or even decades to reach elite status, the accumulated training effects should give rise to larger between-group differences than in short-term intervention studies, which is an advantage of this investigative approach. On the other hand, the researchers have no control over the stimuli the athletes have adapted to over the years, and the differences to the control group may also be due to genetic factors and self selection (meaning that athletes that lack a certain set of traits are fail to become world class). Cross-sectional comparison studies alone are unable to provide an answer to this question (Folland and Williams, 2007: 153).

In longitudinal, intervention-based studies, the **duration of the training program** is a crucial characteristic. While longer interventions can be expected to yield higher strength gains and more pronounced neural adaptations, they also allow for a greater increase in muscle hypertrophy, a possible confounding factor. Earlier studies on ST derived changes in neural mechanisms from improved muscular output in absence of detectable hypertrophy (Gabriel et al., 2006: 133). Since then, the supposed onset of muscle hypertrophy during a ST program has been reevaluated, with some authors arguing that modest hypertrophy begins to manifest after 8-12 training sessions (Damas et al., 2018: 495). Despite the coexistence of hypertrophy and neural mechanisms in the early stages of exercise regimes, the assumption that the increase in strength that accompanies the first few weeks of ST is primarily due to neural factors is still considered valid (Škarabot et al., 2020: 675-676). Later on, muscle hypertrophy effects grow larger and may become the more important factor in force output improvements (Rainoldi et al., 2016: 515). However, the magnitude of the relative contribution of hypertrophy to strength increases remains a debated topic (Taber et al., 2019: 1993).

When designing a training program, researchers and strength coaches alike tend to pay close attention to **exercise selection**, mainly because the *principle of training specificity* dictates that training-induced adaptations only translate to better competition performance if the exercises used in training are similar to the movements used in the actual sport (Zatsiorsky et al., 2021: 6-8). Criteria for said similarity are exercise characteristics like the muscles used in the movement, joint angles, or velocity of execution (ibid.). ST research has demonstrated that an improved 1RM in one exercise does not necessarily translate to an increased 1RM in another one, even if the two exercises target the same muscles (Rainoldi et al., 2016: 512). Thus, the degree to which the task used for assessment was practiced in the training intervention itself becomes a crucial factor in the study design. Since most sports involve complex whole-body movements, multi-joint (or *compound*) free weight exercises are often used in ST and strength testing oriented on sports performance (McGuigan et al., 2013: 9-10). On the other hand, scientists want to employ easily controllable and repeatable testing devices and record data that is free from artifacts caused by undesired movement. This has led to a large number of investigations that include single-joint machine-based exercises, and a focus on the distal muscles of the upper limb, e.g. finger flexors and extensors (Carroll et al., 2011: 833). It is so for not clear how well the findings of these studies can be transferred to more complex movements that involve large muscle groups (ibid.), so that this can be considered a gap between ST research and practice (Škarabot et al., 2020: 677).

Two training regimes that employ the same set of exercises can trigger radically different outcomes if they differ in other factors. The combination of **contraction type** and **execution speed** is used to accentuate maximal strength or improvements in rate of force development (RFD), the ability to generate a high amount of force in a short amount of time. Isometric contractions, which do not involve limb movement, are very common in output force testing (see above) and research studies (ibid.). The most common form of completing a strength exercise includes both eccentric and concentric contractions: eccentric contractions elongate the muscle fibers (e.g. when lowering the weight during a squat or bench press), while concentric contractions shorten them (in the complementary phase of the exercise, e.g. moving the weight upwards in the two examples mentioned). However, some more advanced ST methods call for the isolation of the concentric or eccentric phase, and the latter is hypothesized to allow superior strength increases (Douglas et al., 2017: 917-918; 937-938). Regarding the speed of execution, exercises can be classified as static (no movement) or dynamic (includes movement), with the subcategory of ballistic (fastest possible movement). Ballistic versions of common ST exercises are the squat jump and the bench throw, where the subject releases the bar into the air at the end of the concentric phase of a bench press, performed at maximal velocity. This kind of movement execution is employed to elicit an increase in RFD (Enoka & Duchateau, 2016: 18).

Another crucial aspect in designing a ST program is the interplay of **training volume, load / intensity and rest intervals between sets.** There is a negative relationship between the weight that the subject has to move and the number of repetitions they can complete without having to pause (LeSuer er al., 1997: 211). If the goal is to maximize the adaption to the training stimulus (which might not be always desirable due to load management concerns), the number of repetitions should be high enough to at least approach exhaustion at the end of the set. If the exercises are performed non-balistically, a high number of repetitions (≥12) with a low amount of weight (<67% 1RM) is used to train strength endurance, an intermediate amount of repetitions (6-12) with an intermediate amount of weight (67%-85% 1RM) primarily promotes muscle hypertrophy, and a low number of repetitions (1-6) with a high amount of weight (85%-100% 1RM) elicits the most intense neural adaptations and maximal strength gains (Sheppard & Triplett, 2015: 458). It is important to note that strength endurance, hypertrophy and maximal strength do not grow in isolation from one another, especially in novices. More, a recent review by Schoenfeld et al. (2021: 2-3; 7-10) suggested that the repetition and weight ranges that allow to develop hypertrophy and muscular endurance (but not maximal strength) might be a lot broader than previously thought. On the topic of rest intervals between sets, both Grgic et al. (2018b: 149) and Suchomel et al. (2018: 777) reported that resting for over 2 minutes between sets with an intermediate to high number of repetitions may lead to superior results, although the former made an exception to this rule of thumb for untrained individuals. However, efforts with a low number of repetitions and a very heavy load do induce CNS fatigue, training at this intensity calls for longer rest intervals between sets (2-5 minutes) (Sheppard & Triplett, 2015: 465).

A substantial degree of variation also exists on the next higher level of planning and programming. Training periodization uses macrocycles (one season), mesocycles (one month) and microcycles (one week) as basic units for time spans (Zatsiorsky et al., 2021: 80), and the **training frequency and volume in a microcycle** is another variable to consider. The challenge of choosing the adequate number of sessions and sets consists in providing a strong stimulus to trigger desired adaptation effects while still allowing the body to restore and prepare for the overload of the next session (ibid.: 85-86). In general, highly trained athletes can handle higher workloads than beginners, but recommended minimal resting periods also depend on the characteristics of the workout, with high-load, low-repetition ST being considered especially taxing (ibid.: 86). Most scientific ST studies do not feature elite athletes as subjects, and in this context, most publications state that for beginners, 2-3 sets per exercise are preferable to single-set workouts, while 4-6 sets are recommended for more trained individuals (Suchomel et al., 2018: 776). Regarding the number of training sessions per week, a recent meta-analysis by Grgic et al. (2018a: 1218) found that the correlation between higher weekly training frequency and superior strength gains could be mainly due to a higher overall weekly training volume.

1.3.3. Subject characteristics

On several occasions, the last subchapter briefly mentioned that some properties of ST programs should be adapted to the trainee and their training history. Here, a brief overview of subject attributes that are assumed to influence their capacity to develop muscular strength will be provided.

The level of **previous exposure to ST** is one of the most prominent and influential attributes of ST participants. Intuitively, it is easy to understand that compared to expert performers, starting from a low baseline level of strength leaves a lot more room for further improvement. Further, Chapter 2.3.2 outlined that some of the early neural adaptations to ST are exercise-specific, thus a steeper learning curve can be expected if subjects are unfamiliar with the movement in question. The concept of *training age*, which is the number of years a subject has been training regularly (Myer et al., 2013: 16), is used in sports science to facilitate comparisons between study participants. In order to permit conclusions at group level, categories like *novice*, *intermediate* or *expert* are also used frequently, in some cases without clear-cut definitions (Baker et al., 2015: 147-148-152). Evidently, a training intervention of a few weeks comprises only a small fraction of an expert athlete's training age, but may actually be all the training a person has ever done if the researchers succeed in recruiting training-naive subjects. Thus, the probability to provoke significant changes in any of the measurable parameters discussed in Chapter 2.3.2 is lower in highly trained participants, although not zero (e.g. Judge et al., 2003: 424-425).

The **biological age** of the study participants is another factor that codetermines the body's adaptability to exercise. In children and adolescents, the nervous system is undergoing a natural maturation process, which is believed to be one reason behind the larger relative strength gains (compared to adults) that this population experiences after taking up ST (Myers et al., 2017: 138-139). Further, neural factors are thought to play a larger role than in adults, since children lack the hormone levels to trigger the muscle growth rates that are attainable after puberty (Faigenbaum et al., 2009: S64). At the other end of the age spectrum, elderly people suffer from age-related changes in their neuromuscular system that affect both muscle architecture (loss of muscle fibers and decreased elasticity in the remaining ones) and neural structures (loss of spinal motor neurons and maximal firing frequency) (Mayer et al., 2011: 359). Still, ST provokes the same adaptive mechanisms (hypertrophy and neural changes) in the elderly as in young adults, obviously parting from a lower baseline (ibid.: 360; Walker, 2021: 2). A review by Walker (ibid.: 5) focused on neural adaptations in older subjects (>60 years) and concluded that intervention studies showing said adaptations clearly outnumber null findings. Thus, while the absolute strength numbers of young adults are unreachable, the nervous system's ability to adapt to exercises provides previously inactive older people with a possibility to increase their muscular strength.

According to a recent meta-analysis, there are **sex differences** in the rate of upper-body, but not lower-body strength increase after taking up ST, with females gaining strength more rapidly males (Roberts et al., 2020: 1454). MU number and activation are thought to be similar in both sexes, while relative lean body mass is not (ibid.: 1454-1456). However, women lose strength quicker during muscle unloading, which could hint at sex differences in the adaptability of neuromuscular control structures (Deschenes et al., 2009: 894-895). In general, there is a lack of thoroughly executed studies on sex differences, and the existing ones show considerably heterogeneity in their findings (Roberts et al., 2020: 1458-1459). One study on sustained moderate (10% and 25% MVC) isometric contractions of the vastus lateralis muscle found that rate coding may play a larger role in women than in men in this kind of contractions (Guo et al., 2021: 22-23). This indicates that sex differences in neuromuscular strategies could be a fruitful topic for future investigations.

1.3.4. Conclusion

Research studies on neural adaptations to ST were shown to possess several key characteristics that each allow for considerable variation. They add up to countless possible combinations, so that there are hardly two studies alike. This makes it difficult to compare their results, as methodological differences have to be kept in mind. Further, interpreting sEMG, the most prominent recording technology for the output signal of the neuromuscular system, is a complex task that comes with a set of pitfalls (Vigotsky et al., 2018: 2). In conclusion, assumptions on how the nervous system reacts to exercise-induced stress should be made on the basis of synthesizing several studies. The next two subchapters will deal with the current state of scientific evidence on the topic, turning to reviews and meta-analyses, if possible. Chapter 1.4 will deal with how ST modifies the motor signal's properties, while Chapter 1.5 will try to sum up what is known about the anatomical locations that are responsible for these changes.

1.4. Motor signal determinants and their adaptations to training

1.4.1. MU recruitment

As described in Chapter 1.2.1, the MU is the basic functional unit of the neuromuscular system. Just as the single muscle fiber, an MU has only two states of activation (fully recruited or not recruited) (Enoka & Duchateau, 2016: 2), which means that any process of gradual force regulation has to take place on a higher organizational level. Within a given muscle, the two mechanisms that determine the total output force curve are MU recruitment and MU discharge rate (Wiegel et al., 2019: 2331). At the joint level, the coordination between the different muscles that act around the joint axis plays an important role as well (Ivanenko et al., 2016: 159).

The size principle states that the smaller MUs of a muscle are recruited first during a contraction, if more force is needed, more and larger MUs become active progressively (Enoka & Duchateau, 2016: 15). Whether a MU becomes active or not depends on two factors: its intrinsic electrophysical traits, which relate to fiber diameter and membrane properties, and the share of the motor signal the motor neuron receives as input, which in itself depends on the interplay of ionotropic signals hailing from supraspinal structures or sensory receptors, and slower neuromodulatory signal pathways that do not employ ions for transmission (Heckman et al., 2009: 2041-2042). Due to the large (about 10-fold) range of intrinsic excitability values among MUs, this factor is hypothesized to be the more important one (ibid.: 2042). As smaller motor neurons tend to innervate MUs with a higher percentage of slow-twitch muscle fibers little prone to fatigue, this order of recruitment is generally thought to facilitates smooth dosification of force and ensures that common continuous muscular tasks such as walking or posture control can be executed without eliciting too much fatigue (Hudson et al., 2019: 158). However, a recent simulation study demonstrated that it is the number of MUs and the distribution of innervation numbers that assures these outcomes, not the recruitment order itself (Dideriksen & Farina, 2019: 6154-6155).

While the size principle still provides a valid approximation of MU recruitment mechanisms, several decades of research succeeded in adding caveats to it. While the importance of the neurons' intrinsic properties limits the degree to which the recruitment order can be altered, deviations from the size principle have been observed in contractions that were influenced by sensory and visual feedback or glycogen depletion (Hodson-Tole & Wakeling, 2008: 58). Further, more recent studies have begun to move beyond the whole muscle as analytical unit, conceptualizing a division into groups of MUs called *task groups* (Hudson et al., 2019: 158-159). As the name indicates, these MUs are assumed to engage in synchronized efforts to complete a certain motor task. Within a task group, the size principle still governs the MU recruitment order, but the possibility of selectively prioritizing one task group over another in the same muscle could mean that the MU recruitment order deviates from the size principle when looking at the whole muscle. Task group models that include MUs from various muscles have also been proposed (ibid.).

The fact that the order in which MUs are recruited is relatively stable does not mean that the minimal force necessary to recruit a given MU is fixed. Instead, rapid contractions are characterized by a recruitment process that, compared to slow ramp-up contractions, activates three times as many MUs at the same force level (Duchateau et al., 2006: 1767). Each muscle has an upper limit for generating additional force by recruiting more MUs, for most muscles this limit corresponds to 85% of a MVC, meaning that further force can only be produced by rate coding (ibid.). Whether ST is able to change this limit is currently unknown (ibid.: 1771). Taking advantage of novel sEMG technologies that permit the tracking of individual MUs, Del Vecchio et al. (2019: 1884-1885) found that a 4-week intervention based on rapid contractions was able to lower the participants' MU relative recruitment threshold, indicating that some aspects of MU recruitment could be, to some extent, trainable.

1.4.2. Rate coding

The term *rate coding* refers to the number of times per second a motor unit delivers an impulse to its muscle fibers. Rate coding varies by muscle group and by task (Vigotsky et al., 2018: 3), and contributes to both force output and sEMG amplitude (Farina et al., 2016: 46-47). Just as MU recruitment, rate coding is determined by intrinsic physical properties of the MU and by the descending motor signal.

At high discharge rates, the muscle fiber does not reach a state of full relaxation after contracting before the next impulse causes it to twitch again. The residual activity and the newly arrived signal combine to form a contraction whose intensity exceeds that of a contraction carried out when the muscle fiber is fully rested (Kraemer & Looney, 2012: 15). The highest firing rates observed during voluntary activity can be observed at the onset of ballistic contractions, where they can exceed 100 Hz before decreasing rapidly (Enoka & Duchateau, 2017: 2-3). In comparison, slow, gradually increasing contractions show maximal firing rates below 50 Hz (ibid.: 2). In ramp-up MVCs, the firing rate curve shows steep inclinations at the onset and the end of the contraction, with a more gradual ascent (or even a plateau) in the middle part (Duchateau & Enoka, 2022: 8). The force-frequency-curves vary between MUs, but the reason for this has not been found yet (ibid.). Possible determinants of the firing rate saturation levels include factors related to the incoming neural input as well as the corresponding motor neuron’s properties, such as the number and sensitivity of transmitter channels along it (Fuglevand, 2015: 1311, 1319). Regarding the relationship between MU recruitment threshold and their firing rate, conflicting findings have impeded a definite conclusion so far – some studies have found higher discharge rates in MUs recruited earlier and some have established the opposite (Enoka & Duchateau, 2017: 2).

As stated in Chapter 1.4.1, most muscles display an upper limit of MU recruitment of about 85% MVC. This means that the increase of force from 85% MVC to 100% MVC is achieved entirely through higher firing rates. Thus, the ability to generate large forces and high RFD depends on rate coding to a significant extent (Enoka & Duchateau, 2016: 15). Depending on the MU in question, the difference between minimal and maximal discharge rate corresponds to a force difference of 300%-1500% (Suchomel et al., 2018: 768). This has implications for athletic skill, since explosive movements that demand a high RFD or elevated maximal strength levels can be found in many different sports.

While the exact causes of rate coding differences between MU remain elusive, it is known that the maximal MU discharge rate can be changed through ST. A cross-sectional population-based study found significantly higher vastus lateralis firing rates at maximal (but not intermediate) force in elder weightlifters, compared to an age-matched sedentary control group (Leong et al., 1999: 1641). Further, several longitudinal investigations have documented significant differences in rate coding between trained subjects and the corresponding control groups (Škarabot et al., 2020: 676). Notably, even short interventions may cause said adaptations, since the studies by Christie and Kamen (2010: 655) and Del Vecchio et al. (2019: 1883) found significant differences in firing rate patterns after only two and four weeks, respectively. A possible contributing factor is the more frequent appearance of *doublets*, which are two motor signal spikes that are separated by less than 5ms, a phenomenon occasionally observed directly after the depolarization of the motor neuron (Enoka & Duchateau, 2016: 22). In conclusion, it is highly likely that increased MU discharge rates contribute significantly to the initial strength gains after the first weeks of training.

1.4.3. Intermuscular coordination

MU recruitment and rate coding determine the output forces of single muscles. The next higher organizational level of human movement is the interaction of several muscles that produces net joint torque. As indicated by the principle of muscle redundancy, a given limb movement can be caused by an incalculable number of muscular activity patterns (see Chapter 1.2.2). Since only a small percentage of them can be considered optimal or near-optimal for the aim of the given movement, this variation also entails immense possibilities for improvement and learning through practice. Altering the interplay of muscles around a joint can modify the resulting joint movement or net force, even if the capabilities of the individual muscles stay the same.

A single muscle can take three basic roles in a given joint action: *agonists* are the main contributors to a movement, *antagonists* produce forces that act in the opposite direction, and *synergists* facilitate the agonists' work through auxiliary force contributions or the stabilization of the agonists at their origins (McManus et al., 2021: 3-5). In more complex, multi-joint movements, this clear division into three categories may become an oversimplification, as the roles of individual muscles are not always clear and easily distinguishable (ibid.). Despite creating opposing forces to the desired direction of movement, the simultaneous contraction of antagonist muscles is by no means a hinderance; rather, it increases joint stability and prevents injuries (Walker, 2019: 77). More, even though each muscle is colloquially associated with one main rotational movement it provokes (e.g. biceps brachii causes a rotation of the forearm around the elbow joint), the position of most muscles' attachment sites causes them to generate motion around a second axis as a by-product (Enoka, 2021: 755-756). Depending on the movement's aim, this may require the activation of another muscle that opposes this undesired consequence of the first muscle's contraction. Apart from joint stability and the correction of, coordination between muscles is also used to increase the resulting joint force or RFD, the same joint movement may elicit the participation of more muscles if it is executed at higher speed or with more power (ibid.: 756-757).

The vast amount of different possibilities for motor patterns offered by the redundant musculoskeletal system might lead to the impression of an equally large degree of variation during actual movement execution. However, scientists currently assume that the inventory of all possible movements is organized in a modular fashion, which means that certain combinations of muscle activity bursts act as *building blocks* that can be combined to form more complex movements (Singh et al., 2018: 2). They can be referred to as *motor modules,* or *motor programs*, and the specific interplay of several muscles in time and space that constitute a motor module is called a *muscle synergy* (Cheung & Seki, 2021: 1580). Several important aspects of this proposed sub-unit of human movement remain unclear or controversial, e.g. to what extent muscle synergies are reflected in neuro-anatomical structures and the interplay between these synergies and task characteristics (Singh et al., 2018: 1-2). Among other techniques, the ongoing research on this topic employs sEMG recordings and advanced signal decomposition algorithms to extract muscle synergies from motor signal data acquired during movement tasks (Cheung & Seki, 2021: 1581).

Regardless of the exact rules of motor pattern organization, it is certain that intermuscular coordination is improved by repetitive processes like learning or training. Using sEMG signal decomposition, researchers documented differences in the muscle synergies of newborns and preschoolers, and training-induced synergy changes in ballet dancers and runners (ibid.: 1588). In general, it is thought that the co-contraction of the antagonist muscles may become less pronounced as the individual becomes more skilled at the movement in question, since a more precisely executed movement means that less stabilizing additional forces are needed (Enoka, 2021: 758). This has also been confirmed by some sEMG-based studies on ST, but others have reported null findings on the same question (Walker, 2019: 77), casting doubt on whether intermuscular coordination patterns change quickly enough to manifest themselves in the typical time span of intervention-based experiments. One example of this is the study by Kristiansen et al. (2015), whose data set the present thesis reanalyzes. After a training intervention of 5 weeks that focused on the bench press, previously untrained subjects did not alter their muscle synergy components significantly (Kristiansen et al., 2015: 1956-1957). In summary, while it is well documented that coordination between muscles improves through training and learning, it is not yet known whether these improvements play a significant role in the early strength gains typically observed after the onset of ST.

1.5. Possible anatomical locations of neural adaptations to training

1.5.1. Introduction: Neuroplasticity and its manifestations at cellular level

The term *neuroplasticity* denotes the phenomenon that the nervous system is in a state of constant change and adaptation, responding to all actions and experiences of the individual. This does not only include modifications in neuron sensitivity or output frequencies, but also changes of the nervous system's physical structures itself through the creation of new connections (Costandi, 2016: 2, 13). Neuroplasticity is use-dependent (Taube, 2011: 347), meaning that said new connections are not formed on their own or at random, but dependent on the frequency the neurons are employed for certain tasks. This is also expressed in phrases that reflect convention, such as *"practice makes perfect"*, or in a phrase ascribed to neuroscientist Donald Hebb – *"cells that fire together, wire together"* (Cooper et al., 2013: 29). At the cellular level, several different plastic modifications can be elicited by neural activity. The dendrites of a cell can develop further branches in a process named *arborization*. New synapses can also be created, and the cortical motor map that represents different body parts can be restructured (Rothwell et al., 2021: 3074). Other adaptations that do not directly manifest in alterations of the nervous system's physical structure have also been observed, e.g. the aforementioned increase in firing rate, or a more precise synchronization of different areas like brain regions (ibid.). Furthermore, a current supposition ist that ST may provoke long-term-potentiation of synapses, which means that existing synapses are brought into a state of increased transmittive capacity that lasts for hours or even days after the training session (Tallent et al., 2021: 708). A general property of the nervous system that is important to recall in this context is that neurons are under the influence of inhibitory and excitatory influences, and that a stronger overall output motor signal could be the consequence of both decreased inhibition or increased excitation (Walker, 2019: 77).

Assigning an anatomical locus to the changes of the motor signal's output has proven to be a difficult endeavor, since non-invasive (or limitedly invasive) methods require the interpretation of more indirect evidence, as described in Chapter 1.3.1. The following two subchapters will deal with current efforts to locate the neural adaptations to ST, following the division into supraspinal and spinal adaptations used by other authors (e.g. Duchateau et al., 2021: 6).

1.5.2. Supraspinal adaptations to ST

Supraspinal adaptations include the modifications of the upper motor neurons and any anatomical structures upstream from them. TMS studies are the most widely used scientific instruments to investigate this topic. Spinal influences codetermine the amplitude of the MEP (Kidgell et al., 2017: 2649) and the so-called *silent period* (Škarabot et al., 2019: 636), an interruption of the sEMG signal that occurs when the transcranial stimulus is applied during a voluntary contraction. As a consequence, these measurements on their own are not sufficient to assign changes in post-intervention tests to the supraspinal level (ibid.). Nevertheless, other parameters used in TMS studies do allow this: the threshold for eliciting the MEP (as opposed to its amplitude) depends exclusively on cortical factors (Kidgell & Pearce, 2011: 3209-3210). In addition, a more sophisticated TMS protocol uses two pulses separated by 1-5ms, the first one being sub- and the second one supra-threshold. The reaction to this stimulus pair can be used to infer the presence or absence of intracortical inhibition mechanisms (Di Lazzaro & Rothwell, 2014: 4118-4119).

According to a recent meta-analysis, ST does not have a significant effect on the motor threshold (Kidgell et al., 2017: 2657-2658). On the contrary, changes in intracortical inhibition have been documented (ibid.: 2658). The latter result has been supported by another meta-analysis, with partial overlap in the included studies (Siddique et al., 2020: 17).

Fewer publications have examined possible supraspinal adaptations away from M1 by placing the TMS coil at the cervicomedullar junction, but so far have not found significant changes after a training intervention (ibid.: 18-19). This leads to the conclusion that so far, decreased intracortical inhibition is the best documented supraspinal reaction of the CNS to ST stimuli.

1.5.3. Spinal adaptations to ST

Research into the modifications of spinal anatomical structures makes extensive use of PNS, measuring possible training-induced changes in the H-reflex and the V-wave. As the PNS signal travels from the afferent nerve ending to the spinal cord and back to the efferent periphery, the H-reflex response arises exclusively at the spinal level of the motor system. The V-wave, on the other hand, is measured during a voluntary contraction, which involves a descending neural drive from M1.

Overall, H-reflex studies have shown no significant changes after ST interventions (ibid.: 19-20), but about half of the publications included in the meta-analysis by Siddique et al. (2020) did show a small effect. This assessment of conflicting findings is in line with Aagaard et al. (2020: 154), who remark that these mixed results might be owed to the heterogeneity of study designs (see Chapter 1.3). On the other hand, studies measuring the V-wave response did find significant increases after ST (Siddique et al., 2020: 19-20). The V-wave is an indication for the overall efferent neural drive and is influenced by the same spinal mechanisms as the H-reflex (Aagaard et al., 2020: 154). Thus, while the cortical participation in the V-wave impedes narrowing down its changes to spinal factors, the overall findings suggest that they are involved in early strength gains during ST programs (ibid., Siddique et al., 2020: 19-20, Vangsgaard et al., 2014: 1629). Due to methodological difficulties, the question of the precise anatomical locations of neural adaptions to ST may remain unsolved for years to come. The current state of research suggests that both the supraspinal and the spinal level contribute, with the evidence hinting at a more significant role of supraspinal adaptations (ibid.: 21).

1.6. Conclusion

Increasingly sophisticated measurement techniques and experimental protocols have succeeded in providing conclusive evidence for the existence of neural adaptations to ST. Training-naive subjects can be expected to obtain strength gains unexplained by hypertrophy through ST, no matter their age or sex. However, depending on the characteristics of the training regime, the associated pre- and post-intervention test protocols and other factors, not every research study may be able to point to a clear-cut measurement value that explains why its subjects have increased in muscular strength. The considerable variety of these parameters also makes it difficult to compare the findings of multiple studies.

Regarding the anatomical loci of adaptations to ST, it is probable that both spinal and supraspinal structures are modified through neuroplastic processes if the subject trains regularly. Cortical facilitation is one of the most frequently mentioned mechanism in this context, partly because other possible adaptive mechanisms require the interpretation of more indirect evidence for them. Changes in the spinal neuron circuitry are also thought to occur. Increases in motor unit firing rate are the best documented result at the level of the single muscle, while optimized motor unit recruitment patterns play an important role in RFD, but not necessarily in slower MVCs. At joint level, the interplay between several muscles is also subject to training-induced changes, but so far the evidence for this has been inconclusive for short-term intervention studies. Comparisons that track long-term developments by comparing skilled performers to non-experts or children at different developmental stages have been more successful at highlighting the malleability of intermuscular coordination patterns.

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2. The bench press exercise

2.1. Introduction: the bench press in the context of strength and conditioning

The bench press (BP) is one of the most frequently used upper-body strength exercise (Algra, 1982: 6), and one of three lifts that constitute the sport of Powerlifting. Competitors from many other sports employ the BP in their respective strength and conditioning programs. Prominent examples include throwing specialists in track and field (Waller et al., 2014: 74), Olympic weightlifters (Miller, 2011: 50), and athletes from contact-intensive team sports like rugby (Appleby et al., 2012: 2545) or American football (Pincivero & Bompa, 1997: 251). Apart from being an exercise used in training, it is also considered a vital tool for the monitoring of strength development in the aforementioned sports (McGuigan et al., 2013: 214). Additionally, the BP is popular among recreational weightlifters in gym settings (Duffey & Challis, 2007: 556). The experiment reexamined in this thesis used the most common BP equipment, that is, a barbell, a flat bench and a rack. If otherwise specified, the term BP always references this BP variety here. In this configuration, the BP is a free weights exercise, which means that in contrast to devices specifically designed for one exercise or a Smith machine, the barbell path is not fixed in any of the three dimensions. Therefore, free weights represent a more demanding task in terms of coordination and the use of stabilizing muscles, and training effects are thought to transfer better to sports settings, where movement constraints like the ones imposed by exercise machines are typically absent (Suchomel et al., 2018: 771). On the other hand, free weights require a certain degree of instruction and familiarity (and sometimes spotting) to minimize injury risks (Haff, 2020: 18-19). It is noteworthy that a recent meta-analysis concluded that strength gain comparisons between subjects that trained with machines and subjects that used free weights depended on the modality of assessment – each group performed better than the other group if *their* training equipment was used as strength testing device (Heidel et al., 2021: Abstract). For the BP in particular, both Schick (2009) and McCaw and Friday (1994) compared the muscle activation patterns of free weight BP and Smith machine BP using surface electromyography (sEMG). They found either no difference or a higher sEMG amplitude during the free weight version of the lift, which was significant only for the medial deltoid (Schick, 2009: 28) or both medial and anterior deltoid at 60% (but not at 80%) 1RM load (McCaw & Friday, 1994: 262-263). The flexion and extension of the elbow and the horizontal abduction and adduction of the shoulder are the primary joint actions during the BP, which can be categorized as multi-joint or *compound* exercise. Compared to single-joint or *isolation* exercises, these movements involve more muscle groups at once, are more similar to tasks occurring in daily life (Iversen et al., 2021: 2082) and sports (Suchomel et al., 2021: 771), and seem to elicit superior strength gains (Iversen et al., 2021: 2082). The ideal mixture of compound and isolation exercises in a strength and conditioning program remains a subject of debate in training theory circles (ibid.).

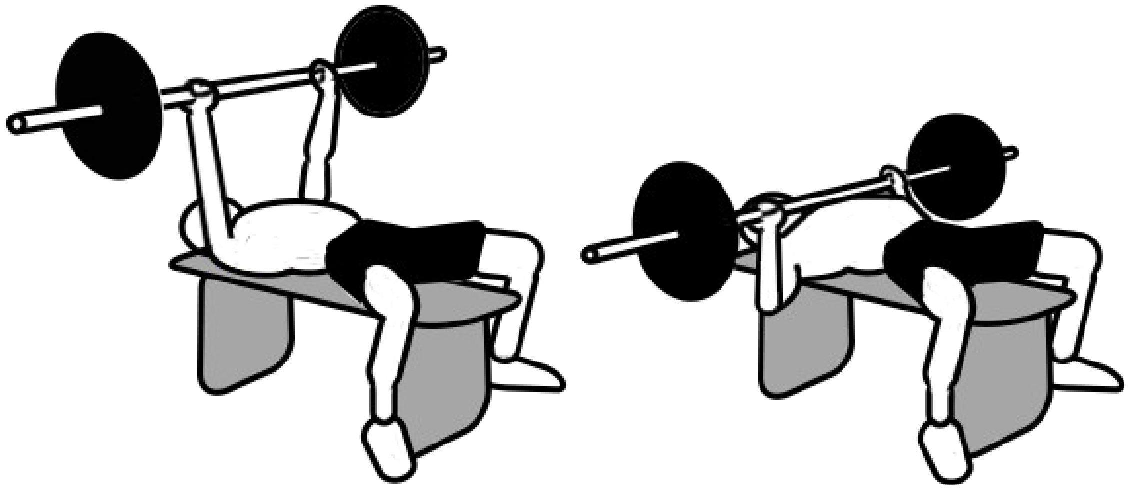
This chapter seeks to familiarize the reader with the most important characteristics of the BP, starting with a brief description of the overall movement. Next, the properties of the most important muscle groups are discussed, before another subchapter addresses the previous sEMG-based research conducted on this lift. Finally, a short conclusion sums up the most important point.

2.2. Description of the overall movement and its phases

A barbell (including weight disks), a barbell rack and an exercise bench make up the equipment required to execute a BP. The athlete lays down on the exercise bench in a supine position, with two feet planted on the floor beside the bench and the glutes and the upper back in contact with it. It must be noted that competitive powerlifters tend to arch their backs markedly in order to reduce the bar path and allow for more involvement of the leg muscles during the lift (García-Ramos et al., 2018: 262). In any case, the correct position on the bench is reached when the eyes are underneath the bar (Algra, 1982: 6). Subjects that are allowed to chose their own preferred grip width tend to place their hands far more than shoulder-width apart, at about 170% of their biacromial distance on average (Lee et al., 2020: 8). In the starting position, the barbell rests on the athlete’s outstretched arms. The barbell is then lowered towards the chest in a controlled manner by flexing the elbow and abducting the shoulder horizontally, until it touches the lower chest area near the xiphoid process (Algra, 1982: 7). This marks the end of the lift’s eccentric phase. Letting the bar bounce off the chest should be avoided, instead it should be pushed upward without the assistance of momentum (ibid.: 11). In the concentric phase of the BP, the athlete moves the barbell back into its starting position by extending their arms. In opposition to the eccentric phase, the shoulder is adducted and the elbow is extended. Since the bar’s position at the end of the eccentric phase is more caudal than at the beginning, it does not travel up and down in a straight line, but follows a slightly arched pathway in the sagittal plane (ibid.). A graphic representation of the BP is provided by Figure 2.1 on the next page.

Altering the BP setup or other characteristics of the lift gives rise to several BP varieties. A non-horizontal bench is used in the incline BP (head is at a higher position than the pelvis) or the decline BP (head is at a lower position than the pelvis). Prescribed grip widths are used to put emphasis on particular muscles (*triceps brachii*, or specific regions of *pectoralis major*). The bench throw is a ballistic BP variant in which the subject releases the bar into the air at the end of the concentric phase, which is executed at maximal speed. The impact of these variants on muscle activation patterns will be discussed in Chapter 2.4.

Figure 2.1: The BP exercise



The figure shows the starting position of the BP (left) and the end position of the eccentric phase (right). The concentric phase consists in bringing the barbell back into the starting position.

2.3. Most important muscles involved and their characteristics

According to a recent review, there is a consensus that the main agonists in the BP are the pectoralis major (PM), anterior deltoideus (AD) and triceps brachii (TB), while latissimus dorsi (LD) and biceps brachii (BB) act as antagonists (Stastny et al., 2017: 6, 11-13). Other muscles are used to provide additional trunk stability, especially among expert lifters (Kristiansen et al., 2015a: 95). Each of the listed muscles can be divided further into single segments, according to their line of action or anatomical criteria like insertion sites or the presence of intramuscular fascial thickenings (Wickham et al., 2004: 64). A study by Wickham et al. (2004) proposed a segmentation model for the three large trunk muscles surrounding the shoulder joint, i.e. PM, LD and the deltoideus. The authors distinguished six segments of PM and LD, respectively, and seven segments of the deltoideus (ibid.: 66). The medial insertions of PM's and LD's segments are markedly further away from each other than the lateral ones, and the segments are arranged at an angle to each other, giving rise to an overall muscle architecture that resembles a handheld fan (ibid.: 63-64). The segmentation of the deltoideus muscle is in line with previous findings, which indicate that the part of the deltoideus commonly referred to as AD consists of three different segments, each with a corresponding intramuscular tendon (Sakoma et al., 2011: 187). For BB and TB, their segments are eponymous: BB possesses a long and a short head, and TB can be split into a medial, a lateral and a long head (Peterson & Rayan, 2011: 883). The subdivision according to functional criteria is often, but not always reflected in anatomical structures: the superior segments of LD and the intermediate (if divided from superior to inferior) segments of PM can only be distinguished by their line of action, not by insertion, fascia characteristics or intramuscular tendons (Wickham et al., 2004: 64). The single segments of a muscle can have pronouncedly divergent lines of action. As an example, at 20° arm abduction in the frontal plane, the superior and inferior of the six PM segments exert their force along vectors that are nearly perpendicular in the frontal plane (ibid.: 67). As the vector sum of the segments' lines of action adds up to the net force contribution of the muscle, differences in the activation patterns of single segments may codetermine success or failure in a BP attempt, but are easily overlooked if the movement is only analyzed at the level of the whole muscle. In the context of sEMG research, it must be recalled that electrode placement determines which segments of the muscle are recorded, and that consequently, the other segments' contributions (and their influence on the overall movement) go unnoticed or show up in the signal as cross-talk. These potential confounders have to be acknowledged when interpreting the results and during the discussion of study limitations.

All of the muscles mentioned here are innervated by motor nerves that branch off the brachial plexus, a network of nerve trunks that branches off the spinal cord between C5 and T1 (Waxman, 2017: 927), although occasional contributions from C4 and T2 are also reported (Leinberry & Wehbé, 2004: 1). Following the nerves from their spinal origin towards the periphery, one observes five spinal roots that unite in three trunks, before splitting into six divisions that later recombine into three cords, from which the brachial plexus' five terminal nerves part (ibid.: 2). The large trunk muscles involved in the BP are not supplied by these terminal nerves, but by smaller nerves that branch off at a higher level. PM is innervated by the lateral pectoral nerve, which originates in the lateral cord, and the medial pectoral nerve, parting from the medial cord (ibid.: 1-3). LD, on the other hand, is controlled by the thoracodorsal nerve, which splits from the posterior cord (ibid.: 3). The other main contributors to the BP movement are innervated by three of the five terminal nerves: the musculocutaneos nerve (which innerves BB), the axillary nerve (AD), and the radial nerve (TB) (ibid.: 3-5). However, deviating innervation patterns are very common in practice (Erhardt and Futterman, 2017: 249-250 and Sassoli Fazan et al., 2003: 17).

The dimensions of a muscle, expressed through parameters like the total number of fibers, their length, the muscle's overall cross-sectional area (CSA) or its volume, are an important determinant of its maximal output force. While it is possible to estimate the overall number of fibers in a muscle from biopsy probes, the procedure to do so is laborious, and muscle fiber numbers are affected by the subjects' age to a great extent (Aagaard et al., 2010: 55). Further, physical training is hypothesized to offset these age-related effects to some degree (ibid.: 59), adding another variable that complicates the comparison between studies. In combination, these factors restrict the availability of data on muscle fiber numbers to a limited number of muscles, and two-fold variations across studies are common, even if the subjects were of similar age and of the same sex (Duchateau & Enoka, 2022: 5). Regarding CSA and volume, Holzbaur et al. (2007: 746) have determined these measures for the major upper-limb muscles of healthy young adult subjects via magnetic resonance imaging (MRI). The largest muscles of the upper body (*Deltoideus*, *Triceps*, *Pectoralis major*, *Latissimus dorsi*, in this order) (Holzbaur et al., 2007: 746) are active during a BP.

More, it has been established that upper-limb muscles tend to contain more fast-fatiguing fibers than than the muscles of the lower extremities (Tirrell, 2014: 59-60). Fiber type distribution generally reflects the muscle's capacity to fulfill different motor needs, with posture control and repetitive low-intensity efforts like walking being prime examples for tasks tailored to type I fibers, while short, intense efforts like jumping, punching and kicking demand the participation of fast-fatiguing type IIA or type IIX cells (Schiaffino & Reggiano, 2011: 1452). Table 2.1 shows fiber type distribution for the muscles engaged in the BP exercise, with some lower-body muscles added for reference.

Table 2.1 – Muscle fiber type distribution for selected upper- and lower-body muscles

Muscle	% MHC-1	% MHC-2A	% MHC-2X
Deltoideus	64.3 ± 7.2	20.8 ± 4.6	14.9 ± 3.8
Triceps brachii – lateral head	45.4 ± 10.5	32.3 ± 6.2	22.3 ± 5.5
Triceps brachii – long head	48.1 ± 7.1	30.0 ± 4.5	22.0 ± 5.1
Triceps brachii – medial head	42.4 ±3.6	39.6 ± 2.5	18.0 ± 2.7
Pectoralis major	54.4 ± 9.3	26.2 ± 5.5	19.3 ± 4.1
Latissimus dorsi	48.6 ± 9.4	29.6 ± 5.2	21.8 ± 4.8
Biceps brachii – long head	55.0 ± 8.1	25.3 ± 6.1	19.7 ± 5.4
Biceps brachii – short head	56.1 ± 9.5	25.7 ± 5.5	18.2 ± 5.0
Vastus lateralis	48.3 ± 9.5	37.5 ± 7.1	14.2 ± 2.9
Biceps femoris – long head	83.0 ± 8.3	6.0 ± 3.4	11.0 ± 5.6
Biceps femoris – short head	68.7 ± 10.1	17.2 ± 5.6	14.1 ± 4.7
Tibialis anterior	79.4 ± 4.8	15.4 ± 3.4	5.1 ± 2.5

Data presented as mean ± standard deviation. Cadaver study by Tirrell (2014: 91-95). n = 6, donor age: 83 ± 7 years, *equal number of male and female specimens.*

Due to these characteristics, upper-body muscles are more prone to hypertrophy than the muscles of the lower body (Folland & Williams, 2007: 148). Since hypertrophy is a confounding variable in studies on neural adaptations to ST, this must be taken into account when discussing results of interventions that primarily targeted the upper body. In addition, the size and fiber type distribution of the muscles involved makes the BP an exercise that allows subjects to move a considerable amount of weight. An illustration of this is Table 2.2, which displays the 1RM percentile values for young adult males and females, respectively, compiled by the American College of Sports Medicine (ACSM, 2010: 91-92). Even averagely strong males can be expected to lift slightly more than their own body weight one time.

Table 2.2 – BP 1RM percentiles for subjects aged 20-29

Sex	Percentile	BP 1RM as fraction of body weight
Males ♂	90	1.48
	75	1.26
	50	1.06
	25	0.9
	10	0.8
Females ♀	90	0.9
	75	0.77
	50	0.65
	25	0.53
	10	0.48

Source: ACSM, 2019: 91-92. n = 6106 for males and n = 1154 for females.

2.4. Previous sEMG studies on the BP

Due to its popularity, the BP is a common research topic in sports science. Several studies have used sEMG to analyze the lift, with varying focal points. The present subchapter will give a brief overview on previous research, emphasizing the findings that are relevant to this thesis' topic.

One line of research is to compare the muscular activation patterns of the BP with those of other upper-body exercises, particularly ones that are similar to the BP in terms of the overall movement or the muscle groups targeted. De Araújo Rocha Júnior et al. (2007: 45) reported less TB participation in the *peck deck* or *butterfly* exercise in comparison to the BP. The *barbell pullover* shows greater TB, but less PM activity than the BP (De Almeida Costa Campos and Fernandes da Silva, 2014: 202-203). For the *standing cable press*, Santana et al. (2007: 1275) found significant differences in agonist, antagonist and stabilizing muscles: the internal and external obliques and the LD showed higher relative sEMG amplitude in the standing cable press, the opposite was true for PM and erector spinae. Two studies have compared the BP to the push-up. Calatayud et al. (2015: 247) performed a longitudinal study which contrasted 5-week training programs based on either the Smith machine BP or elastic band push-ups. Both groups' load was matched to 6RM of the respective exercise (ibid.: 250). No significant differences between the groups were found in sEMG amplitude or strength gains (ibid.: 251). Alizadeh et al. (2020: 290-291) had their subjects perform push-ups on a load cell in order to match the BP load for their cross-sectional study. Their result was that at matched load conditions, subjects were able to perform significantly less repetitions of the BP than push-ups, and that the BP elicited significantly more TB and BB activity (ibid.: 293).

Another approach is to compare different variations of the BP exercise. Bench inclination angles have been investigated in terms of resulting sEMG patterns. Compared to the horizontal BP, the incline BP was reported to lead to a higher sEMG amplitude for the AD (Barnett et al., 1995: 224; Rodríguez-Ridao et al. 2020: 7). TB activity was shown to be less, but this decrease only reached significance in the study by Barnett et al., (1995: 224-225), not in the publications of Arseneault et al. (2021: 10) and Rodríguez-Ridao et al. (2020: 7). Regarding PM activity, the Rodríguez-Ridao et al. (2020: 7) found that both the sternocostal head and the middle portion of PM were less active in the incline BP, while the activity of the clavicular head was greatest at a 30° angle. In contrast, Barnett et al. (1995: 224-225) and Arseneault et al. (2021: 10) reported no significant differences. The decline BP does not engage the sternocostal head of PM more than a horizontal bench position, the activity of the clavicular head is reported as unchanged or decreased (Barnett et al., 1995: 224; Arseneault et al., 2021: 5-6). There were no significant differences in the sEMG of TB (ibid.) or AD (Barnett et al., 1995: 225).

Altering grip width gives rise to further BP variants. A narrative review by Lockie and Moreno (2017: 31-32) states that the close-grip BP elicits significantly higher TB activity, with the effect on the different parts of PM being less clear. Two more recent studies have confirmed a higher degree of TB activity for the close-grip BP (Calatayud et al., 2018a: 272; Mausehund et al., 2021: 5). For AD, both decreases in sEMG (Saeterbakken et al., 2021: 7) and the absence of a significant difference (Mausehund et al., 2021: 7) have been reported. If the athlete chooses a wider-than-normal grip, they lower the involvement of TB (ibid.; Saeterbakken et al., 2021: 7) and possibly increase the level of activation in PM’s sternocostal head (Arseneault et al., 2021: 9; Mausehund et al., 2021: 7), although the latter finding did not replicate in the publication by Saeterbakken et al. (2021: 7).

Further variables that have been studied include load, exhaustion and attentional focus. One would intuitively assume that moving higher loads causes larger average sEMG values. Indeed, this has been confirmed in various experiments (Schoenfeld et al., 2016: 221; Lagally et al., 2004: 362; Silveira Pinto et al., 2013: 4). Still, some details of the relationship between load and sEMG remain elusive. For example, Silveira Pinto et al. (2013: 4) found that sEMG increases with the load at 60%, 70% and 80% 1RM, but sEMG amplitude showed no significant differences between 80% and 90% 1RM. On the other hand, Gołas et al. (2018: 176 & 178) reported significant differences between 85% and 100% 1RM in some muscles, and sex-based differences in the subjects' neural recruitment strategies. Since the employed loads do not match perfectly, the results of these two studies are not entirely at odds with each other, but still show the need for further research into the properties of sEMG signals in tasks with close to maximal loads. The degree of exhaustion is another influential factor on sEMG amplitude. Van den Tillaar and Saeterbakken (2013: 1827; 2014: 63) observed higher sEMG amplitude values during the final repetitions of a given BP set, as did Tsoukos et al. (2021: 2077). Brennecke et al. (2009: 1934) used a single-joint exercise to induce pre-exhaustion before they had their subjects perform the BP – this experimental setup elicited greater sEMG activity in TB only (ibid.: 1937). This indicates that the concept of movement specificity, at least to some extent, also seems to apply to fatigue. Since coaches and athletes routinely use verbal instructions to correct the execution of a movement, the effect of attentional focus on the sEMG signal has also been investigated. The instruction to focus on engaging either the PM or the TB muscle provoked changes in the resulting sEMG curves: Snyder and Fry (2012: 2396) reported a higher degree of activation in the respective muscle in both cases, a finding that was replicated by Catalayud et al. (2018b: 1164), albeit with smaller differences in sEMG amplitude. In the study by Daniels and Cook (2017: 187), instructional emphasis on PM did not lead to a significant increase of PM activity, but to a decrease of TB activity. Kristiansen et al. (2018: 7) found that both an external (concentrating on the barbell's movement) and an internal focus (concentrating on the contraction of PM) elicited higher sEMG amplitudes in various upper-body muscles if compared to an uninstructed baseline condition. However, some of the studies cited here have also established conditions that limit the influence of attentional focus, as differences between foci tend to disappear with increasing load (Snyder and Fry, 2012: 2398) or with increasing speed of execution (Catalayud et al., 2018b: 1164-1165). This indicates that the influence of attentional focus on BP execution might be limited to submaximal efforts.

Conclusions on the interplay between muscles can be drawn from sEMG data in different ways. The simplest and maybe least robust one would be to look at the sEMG curves of different muscles throughout the BP cycle. However, since sEMG values are often reported as average or peak values, relatively few publications on the BP report complete sEMG amplitude graphs. A paper that contains this kind of information for different loads typically used in strength training (70% 1RM – 100% 1RM) is the work by Król and Gołaś (2017). The corresponding sEMG graphs are shown in Figure 2.2 (next page):

Figure 2.2: sEMG amplitude of selected muscles throughout a BP cycle

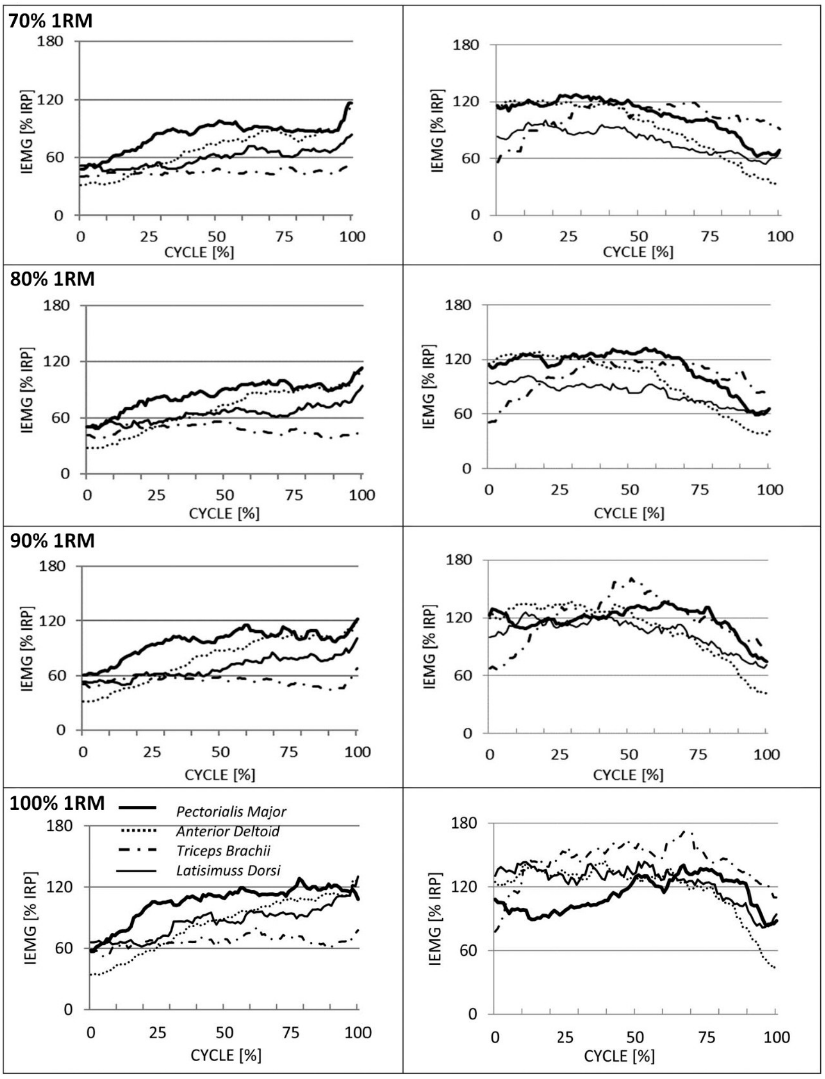


Figure 2.2: sEMG data of four muscles throughout a BP cycle. The four graphs on the left represent the eccentric phase of the movement, while the concentric phase is shown on the right. Data from n=20 participants with at least 1 year of experience in strength training. sEMG amplitude is normalized to peak amplitude of an MVIC reference test. Electrodes of Tricebs Brachii were placed on the long head. Taken from Król and Gołaś (2017: 1325), modified.

The visual inspection of the sEMG graphs indicates that PM, DA and LD all increase their level of activity continuously throughout the eccentric phase (shown on the left column of Figure 2.2), while TB remains fairly constant. This can be observed at all four load levels. The concentric part of the BP cycle shows a greater amount of variety between the different muscles' activity patterns. At all load levels, the sEMG curve of DA is relatively flat during the first half of the phase, before declining constantly in the second half. LD presents similar graphs, but the amplitude decrease towards the end of the concentric phase is less pronounced. PM's sEMG curve is similar to DA at 70% - 90% 1RM load, but differs from it at 100% 1RM, where it presents a rising graph throughout the first three quarters of the phase, before reaching its peak and decreasing steeply. TB shows a rise – peak – fall pattern at all load levels, but its peak is more prominent and occurs later in the phase at very high loads (90% and 100% 1RM). The strength-trained subjects of Kristiansen et al. (2018: 4) produced relatively similar sEMG curves with a weight of 60% 3RM, which corresponds to 52.6% 1RM if extrapolated according to the formula by Mayhew et al. (1992: 204). However, the data by Kristiansen et al. also shows that the overall sEMG graphs are the result of averaging the individual curves of participants that show a considerable degree of inter-subject variation (Kristiansen et al., 2018: 4). In contrast these two findings, Brennecke et al. (2009: 1937) reported that peak sEMG amplitude of PM, TB and DA all occurred after the 80% mark of the concentric movement phase at a load level of 100% 10RM.

The reading of sEMG curves gives away two important characteristics of muscular activity during a BP cycle. Firstly, the sEMG amplitude fluctuations over the BP cycle support the traditional division into concentric and eccentric phase, and maybe even further subdivisions of the two phases. Secondly, the relationships and interactions between the different muscles are complex enough to require more sophisticated investigation methods than simple visual inspection, as the graphs do not show easily recognizable relationship patterns (e.g. strictly parallel or reciprocal curves). The data set this thesis is based on was collected in order to inquire intermuscular coordination patterns during the BP by the means of non-negative matrix factorization (NMF). Kristiansen et al. (2015b: 1949-1951) extracted two muscle synergies that accounted for over 90% of sEMG variance during a BP cycle. The two synergies mirrored the cycle’s two phases (eccentric and concentric). In the muscle synergy most involved in the concentric phase, PM, DA, and the long and medial head of TB were the muscles with the highest synergy vector values (ibid.: 1951). Lower-body muscles (rectus fermoris, vastus lateralis and vastus medialis) and BB had the highest values in the second synergy, corresponding to the eccentric phase (ibid.: 1950). The researchers documented several other characteristics of these muscle synergies: they did not differ between a group of novice subjects that underwent 5 weeks of BP training and a control group, despite a significant increase in BP 3RM of the training group (ibid.: 1956). More, in recreationally trained subjects, the muscle synergies showed a high degree of reliability if re-tested a week after the original test (Kristiansen et al., 2016: 84). Interestingly, a cross-sectional study revealed that compared to beginners, expert powerlifters show less inter-subject variation in their muscle synergy vectors, but more variation in their synergy activation coefficients (Kristiansen et al., 2015c: 95). The synergy vectors represent the relative contribution of the muscles to the synergy, while the activation coefficient shows how much of the overall muscular activity pattern can be explained by the synergy. The variability of the activation coefficients may hint at the powerlifters’ ability to deploy individualized motor control strategies that fit their specific anthropometric and anatomical characteristics (ibid.). On the other hand, the lower variability in the synergy vectors could be interpreted as evidence for the existence of an ideal synergy composition for the BP movement, which the expert group could have reached through practice or through a process of self-selection.

In conclusion, while it is evident that intermuscular coordination is a requirement for BP performance, the nature of its contribution to strength increases remains poorly understood. The majority of previous studies on the BP has not delved into possible interactions between muscles and has instead opted to report sEMG values separately for each muscle. Attempts to establish activity patterns that operate at a higher level than the individual muscle have grown more popular in recent years, but as the number of movements sports scientists study is insurmountable, a lot of exercises have yet to be examined through some of the more recent research techniques. As the extraction of muscle synergies is a very holistic approach, more subtle changes that occur on the level of the muscle pair might not be detected by it. Research in this area has reported that deliberate practice can affect inhibitory reflex circuits that connect synergist muscles, e.g. brachioradialis and biceps brachii. (Aagaard et al., 2020: 159) Further, a reduction of agonist-antagonist coactivation is often observed during movement skill acquisition (Enoka, 2021: 758). Given that agonist and antagonist produce forces of opposite directions, this mechanism could potentially contribute to the increase in force output shortly after the onset of ST. To detect adaptations of these kinds, a computational tool that quantifies pair-wise interactions, such as normalized mutual information, appears to be well-suited.

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