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published in

Journal of Electromyography and Kinesiology
2018

DOI (link to publisher)

[10.1016/j.jelekin.2017.02.006](https://doi.org/10.1016/j.jelekin.2017.02.006)

document version

Publisher's PDF, also known as Version of record

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citation for published version (APA)

van Beek, N., Stegeman, D. F., van den Noort, J. C., H E J Veeger, D., & Maas, H. (2018). Activity patterns of extrinsic finger flexors and extensors during movements of instructed and non-instructed fingers. *Journal of Electromyography and Kinesiology*, 38, 187-196. <https://doi.org/10.1016/j.jelekin.2017.02.006>

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Activity patterns of extrinsic finger flexors and extensors during movements of instructed and non-instructed fingers

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ARTICLE INFO

Article history:

Received 11 November 2016

Received in revised form 10 February 2017

Accepted 17 February 2017

Keywords:

Kinematics

EMG

Coactivation

Muscle compartmentalization

Neuromuscular control

ABSTRACT

The fingers of the human hand cannot be controlled fully independently. This phenomenon may have a neurological as well as a mechanical basis. Despite previous studies, the neuromechanics of finger movements are not fully understood. The aims of this study were (1) to assess the activation and coactivation patterns of finger specific flexor and extensor muscle regions during instructed single finger flexion and (2) to determine the relationship between enslaved finger movements and respective finger muscle activation. In 9 healthy subjects (age 22–29), muscle activation was assessed during single finger flexion using a 90 surface electromyography electrode grid placed over the flexor digitorum superficialis (FDS) and the extensor digitorum (ED). We found (1) no significant differences in muscle activation timing between fingers, (2) considerable muscle activity in flexor and extensor regions associated with the non-instructed fingers and (3) no correlation between the muscle activations and corresponding movement of non-instructed fingers. A clear disparity was found between the movement pattern of the non-instructed fingers and the activity pattern of the corresponding muscle regions. This suggests that mechanical factors, such as intertendinous and myofascial connections, may also affect finger movement independency and need to be taken into consideration when studying finger movement.

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1. Introduction

The human hand has evolved to be able to perform complex actions, such as grasping and manipulating objects. Although the hand shows a tremendous capacity for dexterity, fully independent finger control is not possible (Kilbreath and Gandevia, 1994; Lang and Schieber, 2004). When moving one finger, the neighbouring fingers commonly move to some extent as well, a phenomenon named enslaving (van Duinen et al., 2009; Zatsiorsky et al., 1998). This can limit performance in tasks like piano playing or typing (Hager-Ross and Schieber, 2000; Leijnse et al., 1992; Leijnse et al., 1993). Finger enslaving has been attributed to mechanical factors, such as intermuscular and intertendinous connections, and neural factors, such as the spatial overlap of finger regions in the motor cortex (Schieber and Hibbard, 1993; Schieber and Santello, 2004; van Duinen and Gandevia, 2011).

The contribution of these different factors to enslaving has not yet been fully elucidated.

The muscles controlling finger movement can be divided into two groups: intrinsic muscles (located within the hand) and extrinsic muscles (located within the forearm). Large finger movements are produced predominantly by the extrinsic muscles (i.e. two flexors: flexor digitorum superficialis, FDS, and flexor digitorum profundus, FDP, and one extensor: extensor digitorum, ED) (Butler et al., 2005; McIsaac and Fuglevand, 2007; Reilly and Schieber, 2003), which have insertions on each of the four fingers. Studies on the FDP muscle both in rhesus monkeys and in humans have shown that extrinsic muscle regions can partly be activated independently during finger flexion (Reilly and Schieber, 2003; Schieber, 1993). The inability to completely activate each compartment separately has been explained by motor unit synchronization, whereby the degree of synchronization was higher for the adjacent muscle compartments (Reilly et al., 2004; Reilly and Schieber, 2003). One study using magnetic resonance imaging showed that the activated regions of the FDP and FDS muscles in humans depends on which finger is moved (Jeneson et al., 1990).

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However, these muscle regions also displayed a substantial overlap, especially for the ring finger (Fleckenstein et al., 1992). This is in agreement with the complex anatomy of the FDS muscle (Frohse, 1908).

Although finger movement and its corresponding muscle activation pattern have been studied in detail, only one study in rhesus monkeys (Schieber, 1995) has looked at the relationship between the enslaved finger movement and muscle activation. Based on a mathematical model of finger movement, it was concluded that the combined local muscle activations of the FDS, FDP and ED muscles were able to account for both the movement of the instructed, as well as the enslaved fingers. Investigating the relationship between muscle activation and enslaved finger movement can provide more insight into the neuromechanics of finger independency, but has so far not been studied for the human hand. Both flexor and extensor muscles need to be taken into account for a complete picture of how finger independence can be influenced by muscle coactivations.

The aims of the present study were (1) to assess the activation and coactivation patterns of finger specific flexor and extensor muscle regions during instructed single finger flexion and (2) to determine the relationship between the enslaved finger movements and the respective finger muscle activation. For this purpose, we assessed muscle activation patterns from regions within the FDS and ED muscles that could be associated with movements of individual fingers. The above described anatomical complexity of these muscles obscures the assessment of muscle activation patterns using only a few bipolar surface EMG (sEMG) channels. Hence, we used a multichannel sEMG grid approach.

2. Methods

2.1. Subjects

Nine young, right-handed subjects participated in the study (age 22–29y). All participants had no known neuromuscular disorder, no experience with playing musical instruments for more than two years over the course of the past five years and no disability or surgery in the upper limb in the last two years. To assess hand dominance, each subject filled out the Edinburgh Handedness Inventory (Oldfield, 1971). Right handedness was confirmed by a laterality index of [94–100]. The Research Ethics Committee of the Arnhem-Nijmegen Region approved the study protocol and each subject signed a written consent before participating in the study.

2.2. Data acquisition

2.2.1. Assessment of finger kinematics

Finger movements were recorded with the PowerGlove (Kortier et al., 2014) (University Twente, Enschede, Netherlands), a measurement system that consists of eighteen sensor units (magnetometers, accelerometers and gyroscopes) that are placed on each finger segment (proximal, middle and distal phalanges of the fingers) and the back of the left hand for which the PowerGlove was designed. The PowerGlove was calibrated for each subject using a standard set of hand and finger postures (Kortier et al., 2014).

2.2.2. Measurement of electromyographic signals

Muscle activation was assessed using a grid of sEMG electrodes placed over a large area estimated to cover the FDS and ED muscles (Fig. 1A). Before placing the electrode grid, the length of the left forearm (i.e., the distance between lateral epicondyle of the humerus and ulnar styloid) and its circumference (at $\frac{1}{4}$, $\frac{1}{2}$, and $\frac{3}{4}$

of the length of the arm), as well as width of the wrist were measured. For positioning the grid over the extensor muscles, a reference line from the lateral epicondyle to the ulnar styloid was drawn (Fig. 1A, top). For the grid over the flexor muscles, a line from the medial epicondyle to the middle of the wrist was drawn (Fig. 1A, bottom). Muscle position was checked by using palpation during voluntary movements of specific fingers. Cloth electrodes (KendallTM H69P Cloth Electrodes, Covidien, Zaltbommel, The Netherlands) were reduced in size to obtain an interelectrode distance of approximately 1.7 cm on the proximal-distal axis and 1.3 cm on the medial-lateral axis, with a central circular conductive area of 1 cm in diameter. A grid of 45 (5 rows by 9 columns) surface electrodes was placed over both the flexor and extensor muscles with the middle row aligned with above described reference lines (Fig. 1A). sEMG signals were collected in a monopolar montage with the ground electrode placed on the olecranon and the reference electrode placed on the ulnar styloid, amplified with a 128-channel amplifier and sampled at 2048 samples/s (Refa-136; TMSi, Oldenzaal, The Netherlands).

2.3. Experimental protocol

The left forearm rested on a custom-made armrest that supported elbow and wrist (Fig. 1B). The main task tested in the present experiment was full range single finger flexion for each of the four fingers (2–5). In addition, a single finger hyperextension was performed. This hyperextension was used to localize the finger specific extensor muscle regions and to normalise the sEMG amplitudes during the full range flexion task (see below). For the full range flexion task, the hand was held palmside up in a 45° pronation angle relative to the anatomical position with the fingers held straight and in line with the metacarpals (i.e., metacarpophalangeal, MCP, proximal interphalangeal, PIP, and distal interphalangeal, DIP, joints at 0°) to have a consistent initial finger posture across all subjects.

Subjects were asked to flex the instructed finger in one second until the tip of the finger touched the palm of the hand and to immediately extend the finger back towards its starting position in the following second. Movements were repeated five times for each finger. A metronome (60 bpm) was used to help the subjects with the timing of flexion and extension movements. For the finger hyperextension task, the hand was placed horizontally on a flat surface with the wrist and elbow supported by the armrest. Subjects were instructed to extend their finger maximally, hold this position for five seconds and then return to the starting position. In both tasks, subjects were instructed to move each finger separately (index, middle, ring and little finger) and to not actively resist involuntary movements of the non-instructed fingers. Movements of each finger were repeated five times.

2.4. Data analysis

The PowerGlove data were analyzed with a custom-made algorithm applying the anatomical segment calibration and information from the sensor units (Kortier et al., 2014). Because FDS only spans the MCP and PIP joints, the angles of these joints were summed to represent the movement of the finger that can be the result of FDS activity. All kinematic data were low-pass filtered using a second order, zero-lag Butterworth filter (5 Hz) before angular velocity was derived. Both the kinematic and the EMG signals were synchronised using a trigger signal. Zero-crossings of the angular velocity signal of the instructed finger were used to determine the end of the flexion and extension phase for both the kinematic and the EMG data (Fig. 2). EMG signals were band-pass filtered using a fifth order, zero-lag Butterworth filter (10–500 Hz). Subsequently, the signals were rectified on basis of the

Fig. 1. (A) Position of the electrode grid on extensor muscles (upper picture) and flexor muscles (lower picture) shown for one subject. A reference line from the lateral epicondyle of the humerus (LE) to the ulnar styloid (US) for placement of the extensor grid was drawn. A reference line from the medial epicondyle of the humerus (ME) to the middle of the wrist (MW) was drawn for placement of the flexor grid. The third row of each grid was aligned with the reference line. The grid consisted of 45 electrodes with an interelectrode distance of approximately 1.7 cm on the proximal-distal axis and 1.3 cm on the medial-lateral axis. (B) The experimental set-up with the left forearm resting on a custom-made armrest which supported the elbow and wrist.

Fig. 2. sEMG envelopes of the 45 extensor and 45 flexor muscle channels, finger angle (deg) and finger velocity (deg/s) of five whole finger movement repetitions. The repetitions were divided into flexion and extension phase using the zero-crossings of the angular velocity signal of the instructed finger. The first repetition was omitted from the calculations (shaded gray area).

Hilbert transformation and a low-pass Butterworth filter at 5 Hz. To focus on the changes in amplitude, the baseline level (i.e., the minimum in the 30 ms before the start of flexion) was subtracted to form an EMG envelope, which values were calculated for each channel of the grid.

All data were averaged over the last four repetitions. The first repetition was omitted because its start-up character made that the movement pattern frequently differed from the other four repetitions. For spatial localization of the muscle regions associated with each finger within the sEMG electrode grid, zero-lag cross-covariance between the sEMG envelopes from each channel and the finger angle were determined. The three, unique channels with the highest covariance were selected as a finger specific cluster for further analysis. This procedure (Fig. 3) was applied to the flexion phase of the full range flexion task and to the extension phase of the finger hyperextension task. Apart from the smaller signal to

noise ratio, identical extensor finger clusters were found when instead of the hyperextension task, the extension phase of the full range flexion task was used to identify the channels corresponding to the different fingers. For each subject, eight channel clusters (four flexor and four extensor clusters, one cluster for each finger) were identified.

The sEMG signals were normalized to the maximum sEMG amplitude found for each finger over all tasks. The maximum sEMG amplitude for the flexor clusters was found during flexion phase of the full range flexion task and for the extensor clusters during the extension phase of the hyperextension task.

To characterise the timing of sEMG activity during the flexion movement of each finger, the time point of onset and peak muscle region activation of the sEMG signals were determined (Fig. 4). The start- and end- point of both flexion and extension was determined from the instructed summed finger angle. The time point at the

Fig. 3. Distribution of peak amplitudes on the electrode grid placed over the FDS (Flexor digitorum superficialis - A) and ED muscles (Extensor digitorum - B) during movements of the instructed fingers. Data of a representative subject are shown. Black dots (●) indicate the ten channels with the highest correlation between finger movement and sEMG amplitude which corresponded with the instructed finger movement. Yellow dots (●) represent the three, unique channels with the highest cross-correlation (see main text) for each finger. LE = epicondyle of the humerus, US = ulnar styloid. (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)

end of the flexion phase at which the summed finger angle was maximal (= mid-point), was set to zero (Fig. 4).

Fig. 4. Mean waveforms of the cyclic sEMG signal with subtracted baseline (Δ EMG) of the flexor and extensor EMG clusters corresponding to the index finger as well as the average sum of MCP and PIP angles of the index finger. Data of one representative subject during index finger flexion. Timing (sec) of the following minima and maxima of flexor and extensor EMG signals were assessed: onset of muscle activation (●) and maximum activation at the end of finger flexion phase (▲).

2.5. Statistical analysis

All the statistical analyses were performed using R (version 3.1.0; R Foundation for Statistical Computing (Team, 2013)). One-way ANOVAs were performed to test if the amplitude and timing of sEMG clusters were different between instructed and non-instructed fingers, and between flexor and extensor clusters. To determine the differences between each finger a post hoc pairwise analysis was performed with Bonferroni correction. A Pearson correlation analysis was performed to determine the relationship between the values of the cluster muscle activation and the peak ROM of non-instructed fingers. To assess if the movement amplitude of the non-instructed finger decreases the further away a finger is located from the instructed finger, a Pearson correlation between the non-instructed finger movement and distance to the instructed finger (a distance of 1 for the neighbouring finger, a distance of 2 for 1 finger in between instructed and non-instructed finger, and so on) was used. Significance level was set at a p-value of 0.05.

3. Results

3.1. Spatial localization of FDS and ED muscle regions

An evaluation of the location of the flexor muscle clusters across subjects revealed a low spatial variation in the distribution of the

middle and little finger (Fig. 5). The middle finger clusters were mainly concentrated distally towards the radial styloid process, while the little finger clusters were found more towards the ulnar styloid process. For the index and ring finger, a higher spatial variation between subjects was found. The index finger clusters specifically showed two different locations where index finger muscle activation was found, both distally and proximally. This variation between subjects supports our choice to identify individual finger clusters for each subject separately.

An evaluation of the location of the extensor muscle clusters across subjects revealed a more uniform cluster pattern in comparison to the flexor muscle clusters (Fig. 6). The location of the index finger was located more distally towards the radial styloid process while the middle finger was located more proximally along the forearm. The ring finger location was located between index and middle finger.

3.2. Flexor-extensor timing during the full range flexion task

Before the start of finger flexion, a substantial extensor activation of all four finger muscles was observed, which quickly lowered as flexion progressed (dotted lines in Fig. 7). Although all finger muscle region activations occur simultaneously, the sEMG amplitudes differ significantly between the subsequent finger flexing manoeuvres (see also next paragraph). During the flexion phase, when the instructed finger has flexed approximately halfway, a

rise in the activation of the extensor muscles was detected. Maximal activity of flexors and extensors was found when the finger was almost fully flexed. At the start of the extension phase, both flexor and extensor muscle amplitude decreased. Subsequently the extensor cluster activation peaked at the end of the finger extension phase. Thus, for the first half of the finger extension movement the extensor clusters were not (highly) activated. Only during the last half of the finger extension movement, straightening the fingers to 0°, high extensor activation was observed. For some finger tasks a rise of the flexor muscles was detected at the end of the extension phase.

No significant differences in the timing of synergistic muscle region activation were found between finger movements, thus both instructed and non-instructed finger muscle regions activated simultaneously (Table 1). Between flexor and extensor muscle clusters significant differences in timing were found for the onset and peak flexion time points. During index, middle and ring finger flexion, the extensor clusters were activated later (≈ 0.1 – 0.25 s) than the flexor clusters. No significant differences in timing were found during little finger flexion.

3.3. Range of movement and sEMG amplitudes during finger flexion

As the task required, most joint movement was indeed found in the instructed finger (Fig. 8, right column). For the non-instructed fingers, the highest amount of movement was always found in the

Fig. 5. Cumulative frequency over all subjects of the flexor muscle cluster (three sEMG channels) localization of index, middle, ring and little finger shown for an electrode grid of 5 rows x 9 columns. (medial epicondyle of the humerus = ME, middle of the wrist = MW).

Fig. 6. Cumulative frequency over all subjects of the extensor muscle cluster (three sEMG channels) localization of index, middle, ring and little finger shown for an electrode grid of 5 rows $\times$ 9 columns. (lateral epicondyle of the humerus = LE, ulnar styloid = US).

Index finger EMG - Middle finger EMG - Ring finger EMG - Little finger EMG - Instructed finger angle

Fig. 7. Normalized muscle cluster activation (median of the clusters) and summed angle of instructed finger during index, middle, ring and little finger flexion of a representative subject.

Table 1

Timing (sec) and standard deviation of finger specific signal for both flexor and extensor clusters (= EMG) and instructed finger angle (= PG) during two time points: onset muscle activation and peak of flexion movement. Time point of events was expressed relative to $t = 0$, which was defined as the end of the flexion phase of the instructed finger (Fig. 5). The cluster corresponding to the instructed finger is shown in orange in each row. Means $\pm$ SD of 9 subjects are shown.

		Index finger flexion						Middle finger flexion			
		flexion phase						flexion phase			
		onset flexion		peak flexion				onset flexion		peak flexion	
		flexors	extensors	flexors	extensors			flexors	extensors	flexors	extensors
EMG (sec)	Index	-0.82 $\pm$ 0.23	-0.65 $\pm$ 0.16	-0.13 $\pm$ 0.14	-0.18 $\pm$ 0.21	EMG (sec)	Index	-0.80 $\pm$ 0.24	-0.86 $\pm$ 0.18	-0.40 $\pm$ 0.29	-0.24 $\pm$ 0.20
	Middle	-0.82 $\pm$ 0.20	-0.63 $\pm$ 0.27	-0.17 $\pm$ 0.12	-0.06 $\pm$ 0.25		Middle	-0.92 $\pm$ 0.23	-0.88 $\pm$ 0.13	-0.42 $\pm$ 0.29	-0.34 $\pm$ 0.24
	Ring	-0.79 $\pm$ 0.19	-0.64 $\pm$ 0.22	-0.19 $\pm$ 0.13	-0.06 $\pm$ 0.35		Ring	-0.83 $\pm$ 0.26	-0.71 $\pm$ 0.39	-0.42 $\pm$ 0.29	-0.18 $\pm$ 0.32
	Little	-0.77 $\pm$ 0.19	-0.61 $\pm$ 0.23	-0.17 $\pm$ 0.11	-0.09 $\pm$ 0.35		Little	-0.91 $\pm$ 0.22	-0.83 $\pm$ 0.33	-0.42 $\pm$ 0.29	-0.21 $\pm$ 0.33
PG (sec)		-1.02 $\pm$ 0.08		0.00		PG (sec)		-1.13 $\pm$ 0.11		0.00	

		Ring finger flexion						Little finger flexion			
		flexion phase						flexion phase			
		onset flexion		peak flexion				onset flexion		peak flexion	
		flexors	extensors	flexors	extensors			flexors	extensors	flexors	extensors
EMG (sec)	Index	-0.82 $\pm$ 0.20	-0.70 $\pm$ 0.16	-0.14 $\pm$ 0.20	-0.14 $\pm$ 0.18	EMG (sec)	Index	-0.79 $\pm$ 0.21	-0.77 $\pm$ 0.16	-0.15 $\pm$ 0.07	-0.11 $\pm$ 0.12
	Middle	-0.78 $\pm$ 0.25	-0.68 $\pm$ 0.16	-0.16 $\pm$ 0.18	-0.04 $\pm$ 0.33		Middle	-0.73 $\pm$ 0.20	-0.61 $\pm$ 0.26	-0.13 $\pm$ 0.09	-0.09 $\pm$ 0.13
	Ring	-0.81 $\pm$ 0.22	-0.67 $\pm$ 0.17	-0.18 $\pm$ 0.16	-0.14 $\pm$ 0.19		Ring	-0.77 $\pm$ 0.21	-0.74 $\pm$ 0.19	-0.09 $\pm$ 0.13	-0.07 $\pm$ 0.17
	Little	-0.81 $\pm$ 0.21	-0.69 $\pm$ 0.16	-0.17 $\pm$ 0.16	-0.14 $\pm$ 0.19		Little	-0.74 $\pm$ 0.22	-0.71 $\pm$ 0.15	-0.13 $\pm$ 0.09	-0.09 $\pm$ 0.21
PG (sec)		-0.99 $\pm$ 0.15		0.00		PG (sec)		-0.99 $\pm$ 0.13		0.00

Fig. 8. Mean normalized muscle activation (normalized to the maximum sEMG amplitude found for each finger over all tasks) and the range of movement of all fingers during index, middle, ring and little finger flexion. The horizontal lines above the vertical bars indicate significant differences with instructed finger ($p < 0.05$). Comparisons between the instructed finger angles and the non-instructed finger angles were found to be significant ($p < 0.05$) for all tasks and are not shown in the figure.

closest non-instructed neighbouring fingers. A significant negative correlation ($r = -0.57$, $p < 0.05$) was found between the movements of the non-instructed finger and the distance to the instructed finger. This indicates that the extent of movement in the non-instructed fingers decreased when a finger was located further away from the instructed finger. The post hoc pairwise analysis of the non-instructed fingers showed a significant difference between the two furthest non-instructed fingers during all

tasks. During the middle and little finger flexion task there was also a significant difference in the ROM between the ring and little finger and the middle and ring finger.

The ANOVA's indicated a main effect of instructed finger on the sEMG amplitude of the flexor clusters except during the little finger flexion task. Post hoc analysis showed that the sEMG amplitude of the instructed finger was higher than one or more of the neighbouring fingers during index, middle and ring finger flexion

(Fig. 8A–C). There were no differences in sEMG amplitude between regions associated with the non-instructed fingers during all finger movements. For the extensor clusters, post hoc analysis indicated that during middle finger flexion the sEMG amplitude of the middle finger was lower than the index, ring and little finger extensor clusters. No differences were found between the non-instructed finger extensor regions. A significant positive correlation was found between the sEMG amplitude of the flexor regions and the corresponding non-instructed finger movement during index finger flexion. No other significant correlations could be found for either flexor or extensor regions for all other finger flexion tasks.

4. Discussion

Although several aspects of human finger independence have been studied before, this is the first study in which movement of non-instructed fingers is related to local muscle activation during completely free, natural finger flexion movements. Our main results show (1) a variation in the cluster localisation of the muscle regions between subjects, (2) no differences in timing of muscle activation between fingers, (3) a high coactivation of the non-instructed finger flexor muscle regions during all finger movements, (4) a co-contraction of the respective finger extensor muscle regions during all finger movements and (5) except for index finger flexion, no correlations between the non-instructed finger movement and the corresponding muscle activations.

4.1. Muscle activation patterns during finger flexion

A pattern which is noticeable for all finger movements, yet only significant during index and middle finger flexion, is that the instructed finger flexor cluster had a higher muscle activation than the non-instructed finger clusters. However, overall a high coactivation of the non-instructed finger clusters is visible during single finger flexion, with no measurable difference in muscle activation between the non-instructed finger clusters. In contrast to our observations, studies which measured FDS activation using intramuscular electrodes, found limited coactivation in the adjacent non-instructed FDS muscle compartments (Birdwell et al., 2013; Butler et al., 2005). The different results may be explained by differences in experimental methods and/or protocols. In the study of Birdwell et al. (2013) and Butler et al. (2005), the subjects were trained to activate one specific compartment without activating any other muscle compartments. Our experiment involved natural, unrestricted movements of the instructed and non-instructed fingers without training. As such, it is therefore highly likely that a higher muscle activation was measured in our experiment for the non-instructed finger clusters of the FDS as more finger enslaving was possible.

Intramuscular EMG also assumes that a small muscle region can be a representation of the activation of the whole muscle belly. Yet the complex FDS anatomy, where finger specific muscle bellies are divided into multiple muscle regions connected to one another, may obscure the assessment of the muscle activation patterns (Frohse, 1908). Thus, when measuring sEMG in a specific pre-allocated position, such as with intramuscular EMG or when using bipolar sEMG, muscle activity may be misinterpreted or overlooked. As intramuscular EMG was used in the studies by Birdwell et al. (2013) and Butler et al. (2005), this may be another explanation for finding smaller levels of coactivation. By using an sEMG grid which covers the whole forearm and then later on specifying the specific finger muscle regions we can circumvent the complex anatomy so that the muscle activations measured are fully finger specific. The complex forearm anatomy is also confirmed by the high variability in some cluster locations between

subjects observed in our study. Thus, the differences measured between our sEMG muscle activations and other studies using intramuscular EMG can be explained by the measurement of different finger muscle regions.

As finger movement is not solely controlled by flexor muscles, it is necessary to take the extensor muscle activation into account as well. Not surprisingly, the pattern of the finger extensor clusters appeared to be the opposite of the finger flexor clusters, i.e. the instructed finger cluster apparently had a lower muscle activation than the neighbouring non-instructed finger cluster(s). Yet, this pattern was only significant during middle finger flexion and in general a high coactivation of the non-instructed finger clusters was found. Extensor muscle activation is necessary during finger movements for two reasons: restricting the movement of the non-instructed fingers and finger stabilization of both instructed and non-instructed fingers. In the setup used in this experiment all fingers were held straight at 0° before finger flexion was performed. The higher extensor muscle activation found in our results can be explained by this starting point of 0°, since in other studies the fingers were either partly or completely limited in their movements by use of a splint. To hold the non-instructed fingers in their respective angle of 0° a high amount of extensor muscle activation of the non-instructed muscles was necessary.

Our results show that during natural finger flexion a high coactivation of the non-instructed finger regions of both extensor and flexor muscles are used to perform a fluid finger movement. Thus, when studying the effects of disease or aging on natural finger movement, it is important to take this complexity of muscle activation into consideration and not solely focus on studies where non-instructed fingers are restricted, as this can give a limited view of finger muscle control.

4.2. Relationship between sEMG and kinematics of non-instructed fingers

Insight in what causes the movements of the non-instructed fingers may be provided by the relationship between the flexor and extensor sEMG and the corresponding non-instructed finger kinematics. We found that fingers cannot move independently and that enslaving was highest in the adjacent fingers and decreased when the distance to the instructed finger increased. Finger (in)dependency has been extensively studied and a similar kinematic enslaving pattern has often been reported (Hager-Ross and Schieber, 2000; Li et al., 2004). The question is, if this can be explained by the activation patterns of the FDS and ED muscle regions uniquely associated with the non-instructed fingers.

The simultaneous activation of muscle regions corresponding to non-instructed fingers has been reported in several studies for the FDS, FDP and ED muscles (Butler et al., 2005; Darling and Cole, 1990; Hu et al., 2015; Leijnse et al., 2008; Mclsaac and Fuglevand, 2007; Reilly and Schieber, 2003; van Duinen et al., 2009). One study looking at the FDP using intramuscular EMG showed that muscle activation of the non-instructed fingers was lower the larger the distance was to the instructed finger (Reilly and Schieber, 2003). Although a significant positive correlation was found between the non-instructed finger movements and the respective flexor muscle activation during index finger flexion, no other significant correlations were found for the other finger tasks in our study.

In general, the kinematic pattern of the non-instructed fingers, where the fingers located nearby have a higher amount of movement than the fingers located further away, cannot be traced back to our muscle activation data of the non-instructed finger regions. Thus, a clear difference between finger movement and sEMG amplitudes of the non-instructed fingers is found for all finger movements. This implies that the observed non-instructed finger

movements cannot be solely explained by their respective muscle region activations and that other factors, such as mechanical factors need to be taken into consideration.

4.3. Timing

Our sEMG timing results show that a delay between the instructed and non-instructed finger muscle activations could not be found for both flexor and extensor finger muscle regions. Although the kinematics of non-instructed finger movement is a well-studied phenomenon (e.g. (Hager-Ross and Schieber, 2000; van Duinen and Gandevia, 2011)), the range in which independent movement can occur is not well known. Movement delays between the instructed and non-instructed fingers were reported by us recently (Van den Noort et al., 2016) and a study of Li et al. (2004) has also shown a time lag between enslaved and instructed fingers with a larger delay for the fingers the furthest away from the instructed finger (Li et al., 2004).

Our results suggest that factors other than those of neural origin, thus a difference in the muscle activation timing between instructed and non-instructed finger muscle regions, should be taken into account. Two mechanical factors have been described in other studies: tendinous connections between the distal tendons (Leijnse et al., 1997) and connective tissue linkages between muscle bellies (Maas et al., 2003). The potential role of these connections has been illustrated in other studies (Lang and Schieber, 2004; van Duinen and Gandevia, 2011), but has not been systematically investigated.

4.4. Using multi-electrode surface sEMG to assess activity in FDS and ED compartments

One of the main challenges of the present study was to identify the regions that correspond to the FDS muscle regions of the different fingers using the electrode grid on the surface of the arm. For each individual finger a main cluster position was identified from the sEMG grid for both flexor and extensor muscles, which generally corresponded to the underlying anatomy as described in the literature (Frohse, 1908) and as found in our own dissections (not shown). Specifically, the cluster positions highlight the area where sEMG surface electrodes are best able to extract finger specific muscle activity. For the ED, the index and middle finger clusters showed the least amount of overlap. Yet even with this overlap the location of the extensor clusters was consistent with those reported in previous studies (Gallina and Botter, 2013; Gazzoni et al., 2014; Hu et al., 2015; Leijnse et al., 2008). The flexor muscle location has mainly been determined using ultrasound and cadaver studies. Our FDS cluster locations and those of previous studies were very similar for the index, middle and little finger (Bickerton et al., 1997; Gazzoni et al., 2014; Henzel et al., 2010).

A second limitation may be the use of sEMG itself. In this experiment sEMG was used for a multitude of reasons. Compared to intramuscular needle or wire EMG, sEMG is usually considered a cost and time efficient method of measuring muscle activation. sEMG is also regarded as a more comfortable measuring approach for the subjects. However, we have to take into consideration that sEMG may not be precise enough to measure some aspects, such as individual finger muscle timing, and thus small differences may be overlooked. Intramuscular EMG could be a better technique to look into this aspect more accurately.

A third important limitation is the potential effects of crosstalk on our results. The possibility for effects of crosstalk were minimized, because the distances between the unique finger channel clusters were quite large (an average distance on the proximal-distal axis of $[4 \pm 1]$ electrodes (equalling ≈ 5 cm) and on the

medial-lateral axis $[2 \pm 1]$ electrodes (equalling ≈ 3 cm). Modelling and experimental studies have shown that a substantial decay of motor unit action potential amplitudes occurs with an increasing distance between the electrodes (Lowery et al., 2004; Roeleveld et al., 1997). Both studies showed that with a distance of approximately 2 cm between the electrodes the contribution of the neighbouring electrodes to the RMS amplitude was reduced to only 10–20%. We also found co-activity of the most spatially separated muscle bellies, that of the index and ring finger (mean distance on the proximal-distal axis of 4 electrodes). Due to the fact that we found also similar activation patterns in those distant muscle regions, which are unlikely the result of cross-talk, we feel confident that also the signals from more proximate channels were not highly contaminated by cross-talk.

5. Summary

Our data show a disparity between the muscle activations of the non-instructed fingers and their kinematic movement patterns. These differences were also found in the non-instructed finger movement delay which could not be traced back to any differences in muscle activation timing between finger muscle regions. In addition, variation in the muscle region localisation was found between subjects which suggests that a sEMG grid approach is warranted as a superior alternative to bipolar sEMG using few channels. Our results imply that during natural single finger movement tasks other mechanisms, such as intertendinous and myofascial connections, may also affect finger independency and need to be taken into consideration.

Conflict of interest

The authors declare that they have no conflict of interest or financial ties to disclose.

Acknowledgements

The authors thank the subjects for participating in the study, Thomas van der Kraan and Barry Thonen for assisting in the measurements and Henk Kortier, Ed Droog and the technical division of the department of Human Movement Sciences for technical assistance. This research is funded by the European Commission through MOVE-AGE, an Erasmus Mundus Joint Doctorate program (2011-0015) and by the Division for Earth and Life Sciences of the Netherlands Organization for Scientific Research (grant 864-10-011).

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