Assessment of Individual Finger Muscle Activity in the Extensor Digitorum Communis by Surface EMG

J.N.A.L. Leijnse,1,2 N. H. Campbell-Kyureghyan,3 D. Spektor,3 and P. M. Quesada1

Departments of 1Mechanical Engineering and 3Industrial Engineering, Speed School of Engineering and 2Hand and Upper Extremity Research Laboratory, Price Institute for Surgical Research, Department of Surgery, University of Louisville, Louisville, Kentucky

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INTRODUCTION

The extensor digitorum communis (ED) is a slender muscle complex in the dorsal forearm from which tendons arise to the index (D2), medius (D3), ring (D4), and little (D5) fingers. The index and little fingers each have an additional extensor, the extensor indicis (EI) and extensor digiti minimi (EDM), respectively. Anatomically, the ED parts to the individual fingers are generally regarded as having limited independence. This view seems consistent with the often extensive tendinous interconnections, known as juncturae intertendinei, that exist between the ED tendons at the dorsum of the hand. These interconnections limit relative ED tendon displacements and therefore finger independence (el-Badawi et al. 1995; Hirai et al. 2001; Kitano et al. 1996; Leijnse et al. 1992, 1993; Mogk and Keir 2003; von Schroeder and Botte 1993, 1995; von Schroeder et al. 1990; Yoshida 1995; Zilber and Oberlin 2004). The common assumption that surface electromyography (EMG) cannot distinguish individuated ED activity with acceptably low levels of crosstalk is consistent with the common use of indwelling electrodes in ED EMG (Dennerlein et al. 1998; Harmon et al. 1993; Huesler et al. 2000; Keen and Fuglevand 2004; Schieber 1995; Seradge et al. 1999; Simoone et al. 2003; Steichen and Petersen 1984). However, detailed anatomic dissections have demonstrated that the ED parts to the different fingers have constant and widely spaced anatomical locations that promote independent function (Leijnse et al. 2008). These findings prompted the hypothesis that individuated ED activity assessment should be possible by appropriately placed small (4 mm) bipolar surface electrodes (Leijnse et al. 2008).

The study objective was to evaluate this hypothesis by measuring surface EMG from the ED parts, EDM, EI, extensor pollicis longus (EPL), abductor pollicis longus (APL), and wrist extensors, during individual finger tapping tasks. To avoid undue extrapolation to the feasibility of ED surface EMG in general hand/finger tasks, the experimental limitations must be emphasized. In the finger tapping tasks, the forearm, hand and nontask fingers were resting on a table to reduce as much as possible activity in elbow flexors, wrist extensors and supinator, and resulting crosstalk to the finger extensors. Electrodes were placed with the forearm resting in the tapping position at the table. Sufficiently large pronation-supination movement would likely shift the dorsal forearm skin relative to the narrow underlying muscles, putting (some) electrodes in less optimal anatomical locations.

We deemed these limitations consistent with the primary aim of demonstrating the feasibility of individuated ED surface EMG. While the experimental setup was optimally constrained, the EMG data themselves were recorded by simple bipolar electrodes and are presented unmitigated by crosstalk filtering. Consequently, such data might be further enhanced with crosstalk reduction techniques. However, due to close interlacing of the narrow extensor muscles and their tendons of origin and insertion (Leijnse et al. 2008), (spatial) crosstalk filtering (Farina et al. 2004; van Vugt and van Dijk 2001) may not be straightforward.

In combination with the anatomic study of Leijnse et al. (2008), the present results should help refine motor-control, ergonomic, and clinical finger function studies (Berguer et al. 1999; Boostani and Moradi 2003; Burgar et al. 1997; Claudon 2003; Gerard et al. 1999; Laursen et al. 2001; Lin et al. 2004; Petersen 1984). However, detailed anatomic dissections have demonstrated that the ED parts to the different fingers have constant and widely spaced anatomical locations that promote independent function (Leijnse et al. 2008). These findings prompted the hypothesis that individuated ED activity assessment should be possible by appropriately placed small (4 mm) bipolar surface electrodes (Leijnse et al. 2008).
correlations and anatomic proximity. These interpretations should be necessary for task execution, PEMG can be assumed to be mostly crosstalk (e.g., from synergists), but as task finger extensor activity is measured individuated ED EMG. To this end, AEMG quantification. The study’s purpose was to demonstrate feasibility of measuring ED EMG. In the following, muscles are indicated by their abbreviation and their electrodes by suffix e (e.g.: ED2e). EMG recorded by the task finger extensor electrodes will be called primary EMG (PEMG). EMG collected in other muscles' electrodes is called accessory EMG (AEMG). AEMG consists of coactivation of the targeted muscle and/or crosstalk. Recorded PEMG may in principle also contain crosstalk (e.g., from synergists), but as task finger extensor activity is necessary for task execution, PEMG can be assumed to be mostly primary motor activity. PEMG amplitude is therefore used for relative AEMG quantification. The study’s purpose was to demonstrate feasibility of measuring individuated ED EMG. To this end, AEMG analysis using advanced filtering was beyond the study scope. However, AEMG interpretations are proposed based on cross-subject correlations and anatomic proximity. These interpretations should be considered hypotheses to be validated or rejected by further research.

Measured ED muscle parts

ED2, ED3, and ED4 will indicate ED parts to index, middle, and ring fingers, respectively. A fourth ED part (ED5) is also consistently present. Its tendon—sometimes multiple tendon slips—inserts into both ring and little fingers. As the ED5 belly is not consistently anatomically separable from the ED4 (Leijnse et al. 2008), it was not targeted separately from ED4. The ED4, as placed in this study will likely record also activity from the proximal part of ED5. In surface EMG, ED5 is also of relevance in that its muscle belly may anatomically variably reach far distal and cause crosstalk in EIe and EPLc (Leijnse et al. 2008).

Subjects

Ten right arms were measured in 10 subjects, who were reasonably diverse in age, gender, and skin-thickness (Table 1). One potential subject was excluded from the study after detecting anatomic absence of EI (see RESULTS). Subjects signed an informed consent form approved by the University of Louisville Human Subjects Protection Program Office.

Tapping tasks

The forearm, including elbow, hand, fingers, and thumb, was rested on a surface. Six individual finger tasks were performed (Table 2). Four tasks (T2–T5) were individual finger tapping on the surface. The fifth task (T1V) was thumb tapping, vertical to the surface, using the carpo-metacarpal (CMC1) joint. The sixth task (T1H) was horizontally abducting and adducting the thumb, parallel to the table surface, using the CMC1 joint. Subjects were instructed to rest non-task fingers on the surface and relax them as much as possible. Each task served to elicit isolated activity in the task finger extensor(s) (Table 2). The index finger has two extenders (ED2 and EI). Consequently, T2 will elicit activity in at least one of them. The T1H task elicits synergistic activity in EPL and APL extensor pollicis brevis (EPB), as the thumb, while ab-adducting horizontally, must remain lifted in neutral extension above the table surface.

Measuring devices

EMG was recorded by sixteen bipolar 4-mm surface electrode pairs (E263N-LU; In Vivo Metric, Healdsburg, CA). Electrode placement sites were shaved and wiped with alcohol. Electrode cups, filled with conductive gel, were attached to skin by double-sided adhesive washers (E401M, in Vivo Metric). Electrodes were connected by screw terminals to preamplifiers (gain factor 20, Motion Lab Systems; Baton Rouge, LA), which connected via shielded wires to a 16-channel MA300 EMG unit (Motion Lab Systems), with high- and low-pass hardware filters set at 20 and 500 Hz, respectively. The filtered EMG was acquired by a DAQ card [PCI-6071E, National Instruments (NI), Austin, TX]. The EMG DAQ card was synchronized with the finger motion sensors’ DAQ card using Labview (version 7.1, NI).

Finger movements were recorded either with flexible goniometers (SG65, SG75, or F35, Biometrics, Ladoysmith, VA) at the finger’s metacarpophalangeal (MCP) joints and thumb CMC1 joint in subjects who subsequently participated in other tests or in the other subjects by miniature accelerometers in ultralight housings on the proximal phalanges (ADXL320, Analog Devices, www.analog.com). These sensors served primarily as switches for finger task identification during data processing. All motion sensor data were collected by shielded wires though a terminal block, signal conditioners, chassis and DAQ card (SCXI-1314, SCXI-1520, SCXI 1000, and PCI-M6221, respectively, NI) using Labview 7.1 as described in the preceding text.

Data collection procedures

ELECTRODE PLACEMENT. Subjects were seated in a height adjustable chair, with upper arm abducted/extended at 30–45° and with elbow, forearm, hand, fingers, and thumb rested on a padded surface. Electrogoniometers or accelerometers were placed as described in the preceding text. Superficial muscle outlines were palpated and marked for ED2, ED3, ED4, EDM, APL, extensor carpi radialis brevis (ECRB), extensor carpi radialis longus (ECRL), and extensor carpi ulnaris (ECU). Palpation forces were applied perpendicular to the skin to prevent lateral skin deformation that might cause markings to not corresponding to underlying structures. The deep EI and EPL were marked after palpation of end tendons and distal muscle bellies. Optimal electrode locations as determined in (Leijnse et al. 2008) were marked by small crosses. Ten electrode pairs were placed with 20 mm center-to-center spacing at ED2, ED3, ED4, EI, EDM, EPL, APL, ECRB, ECRL, and ECU, with a common ground electrode at the olecranon of the ulna (Fig. 1 and Table 3). At placement, EMG signal was verified by real-time display. Subjects were provided time to familiarize themselves with the tapping tasks, during which PEMG levels and AEMG levels in nontask extensor electrodes were qualitatively assessed. Based on these assessments, the remaining six electrode pairs were placed over other portions of ED2, ED3, EDM, EI, and EPL to investigate if PEMG recording could be improved and AEMG recording minimized. Electrode positions were then photographically documented (Fig. 1). For duplicated electrode pairs, only the results of the pair recording for all six tapping tasks the least overall AEMG levels as a proportion of the respective PEMG levels were selected for final processing.

<table>
<thead>
<tr>
<th>Subject</th>
<th>Hand</th>
<th>Gender</th>
<th>Age</th>
<th>Skin Thickness, mm</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>R</td>
<td>M</td>
<td>30</td>
<td>16.0</td>
</tr>
<tr>
<td>2</td>
<td>R</td>
<td>M</td>
<td>25</td>
<td>4.5</td>
</tr>
<tr>
<td>3</td>
<td>R</td>
<td>F</td>
<td>31</td>
<td>15.5</td>
</tr>
<tr>
<td>4</td>
<td>R</td>
<td>M</td>
<td>43</td>
<td>5.8</td>
</tr>
<tr>
<td>5</td>
<td>R</td>
<td>M</td>
<td>50</td>
<td>2.5</td>
</tr>
<tr>
<td>6</td>
<td>R</td>
<td>M</td>
<td>26</td>
<td>3.8</td>
</tr>
<tr>
<td>7</td>
<td>R</td>
<td>F</td>
<td>33</td>
<td>6.0</td>
</tr>
<tr>
<td>8</td>
<td>R</td>
<td>M</td>
<td>29</td>
<td>6.2</td>
</tr>
<tr>
<td>9</td>
<td>R</td>
<td>F</td>
<td>26</td>
<td>5.9</td>
</tr>
<tr>
<td>10</td>
<td>R</td>
<td>M</td>
<td>42</td>
<td>4.5</td>
</tr>
</tbody>
</table>

Mean St dev 6M, 4F 33.5 ± 8.6 7.1 ± 4.7

Subjects gender, age, and skin thickness measured halfway the forearm at the extensor digitorum communis (ED).
generally also performed somewhat slower. Than in less constrained fingers. Thumb tasks, especially T1H, were more anatomically constrained fingers, tapping was usually slower tapped at their comfortable speeds, which differed individually. In extension to a comfortable range, however small it might be. Subjects were instructed to avoid excessive effort by limiting finger such as the juncturae intertendinei. To limit muscle coactivations, often severely limited, by hypothesis due to anatomical constraints dent extension of ring and occasionally little finger in tapping was sequence. In total, 50 – 80 s of EMG was collected per task. Independent per second. Sequences were spaced by small periods of relaxation. Recorded during sequences of 40 –50 tappings at one to three cycles per minute. MVC recording. Prior to data collection, amplifier gains were adjusted such that maximum voluntary contraction (MVC) signals for each muscle were within ±5V. Maximum isometric contraction EMG was then recorded for each instrumented muscle, as specified in Table 4.

FINGER TAPPING EMG. EMG and finger motion sensor outputs were recorded during sequences of 40–50 tappings at one to three cycles per second. Sequences were spaced by small periods of relaxation. Approximately 3 s of resting EMG was collected prior to each sequence. In total, 50–80 s of EMG was collected per task. Independent extension of ring and occasionally little finger in tapping was often severely limited, by hypothesis due to anatomical constraints such as the juncturae intertendinei. To limit muscle coactivations, subjects were instructed to avoid excessive effort by limiting finger extension to a comfortable range, however small it might be. Subjects tapped at their comfortable speeds, which differed individually. In more anatomically constrained fingers, tapping was usually slower than in less constrained fingers. Thumb tasks, especially T1H, were generally also performed somewhat slower.

Data processing procedures

EMG selection and filtering. Let \( V_{s,d,e} \) be the EMG in the electrodes \( e \) of a single tapping \( i \) in tapping task \( d \) by subject \( s \). In each tapping test, EMG data of 10 consecutive tappings \( (V_{s,d,e,1:10}) \) were selected based on amplitude consistency, minimum overall AEMG and absence of artifacts. EMG bursts and resting phases were generally of about equal duration (Fig. 2). Signals were cut in the middle of resting phases using Diadem 9.1 software (NI). EMG was offset to zero

\[
V_{s,d,e,1:10} = V_{s,d,e,1:10} - \text{mean}(V_{s,d,e,1:10}) \quad (I)
\]

The resting noise spectra showed mainly the 60-Hz power supply component, which was filtered in Diadem from both tapping EMG and MVCs with a band-stop Butterworth filter, order 3, 58–62 Hz.

## Table 2. Finger tasks, anatomical extensors

<table>
<thead>
<tr>
<th>Finger Task</th>
<th>D1</th>
<th>D2</th>
<th>D3</th>
<th>D4</th>
<th>D5</th>
</tr>
</thead>
<tbody>
<tr>
<td>T1V</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>T1H</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>EPL</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>EPL</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>APL</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>APL</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>EPB*</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ED2 EI</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ED3 ED4 EDM</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Finger tasks with targeted anatomical extensors. In T2, ED2 and extensor indicis (EI) are both index extensors. In T1H, extensor pollicis longus (EPL) is synergistic with abductor pollicis longus (APL); extensor pollicis brevis (EPB) in thumb abduction in the horizontal plane. *EPB was not individually targeted, electrodes were placed on APL.

EMG normalization and quantification. Root mean squares (RMSs) of the filtered samples \( V_{s,d,e,1:10} \) were calculated in Diadem for a 1% window of sample duration. This window length was approximately a fifth of the duration of a single tapping EMG burst. For normalization, RMS of 2 s filtered MVC EMG \( (V_{s,e,MVC}) \) was calculated for a 100% window. Further processing was performed in MATLAB. After normalization

\[
NRMS_{s,d,e,1:10} = \frac{\text{RMS}_{10\%}(V_{s,d,e,1:10})}{\text{RMS}_{100\%}(V_{s,e,MVC})}
\]

peak detection was performed over ten equidistant time intervals, producing one peak \( P_{s,d,e,i} \) per tapping EMG burst \( i \) per electrode \( e \), of which the means and SD were calculated

\[
MP_{s,d,e,i} = \text{mean}(P_{s,d,e,i}) \quad (a)
\]

\[
StdP_{s,d,e,i} = \text{std}(P_{s,d,e,i}) \quad (b)
\]

Multiple electrode data selection. For each muscle with two electrode pairs, data were selected of the pair that recorded in the six tasks the least overall normalized mean peak AEMG \( (MP_{s,d,e}) \). Statistical significance-two-tail paired t-test. For the selected data, for each task \( d \) the cross-subject statistical significance of the

### Table 3. Electrode locations

<table>
<thead>
<tr>
<th>Electrode</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>ED2</td>
<td>Mid-forearm at radial border of ED</td>
</tr>
<tr>
<td>ED3</td>
<td>Proximal electrode just proximal to humerus-radius joint, electrodes at ED midline</td>
</tr>
<tr>
<td>ED4</td>
<td>Near ulnar border of ED, opposite ED2 electrode</td>
</tr>
<tr>
<td>ED5</td>
<td>Not targeted</td>
</tr>
<tr>
<td>ED6</td>
<td>Halfway forearm, or somewhat more distal, according to palpation muscle belly</td>
</tr>
<tr>
<td>EI</td>
<td>Just radial to distal head of ulna</td>
</tr>
<tr>
<td>EPL</td>
<td>Distal electrode: about 1 cm proximal to Listers’ tubercle, other electrode 20 mm proximal, on line from Listers’ tubercle intersecting with ulna at proximal third</td>
</tr>
<tr>
<td>APL</td>
<td>Proximal border of APL, lateral-dorsal at radius (EPB is not targeted)</td>
</tr>
<tr>
<td>ECRB</td>
<td>Radial to ED, at about 30% of forearm length</td>
</tr>
<tr>
<td>ECRL</td>
<td>Proximal electrode just distal to lateral rim humerus, about .40 mm proximal to the humerus-radius joint space, after palpation. Distal electrode 20 mm distal to first electrode</td>
</tr>
<tr>
<td>ECU</td>
<td>At 50% of forearm length, about 5 mm dorsal to palpatd dorsal rim of ulna</td>
</tr>
</tbody>
</table>

Electrode locations (see Fig. 1).

EMG normalization and quantification. Root mean squares (RMSs) of the filtered samples \( V_{s,d,e,1:10} \) were calculated in Diadem for a 1% window of sample duration. This window length was approximately a fifth of the duration of a single tapping EMG burst. For normalization, RMS of 2 s filtered MVC EMG \( (V_{s,e,MVC}) \) was calculated for a 100% window. Further processing was performed in MATLAB. After normalization

\[
NRMS_{s,d,e,1:10} = \frac{\text{RMS}_{10\%}(V_{s,d,e,1:10})}{\text{RMS}_{100\%}(V_{s,e,MVC})}
\]

peak detection was performed over ten equidistant time intervals, producing one peak \( P_{s,d,e,i} \) per tapping EMG burst \( i \) per electrode \( e \), of which the means and SD were calculated

\[
MP_{s,d,e,i} = \text{mean}(P_{s,d,e,i}) \quad (a)
\]

\[
StdP_{s,d,e,i} = \text{std}(P_{s,d,e,i}) \quad (b)
\]

Multiple electrode data selection. For each muscle with two electrode pairs, data were selected of the pair that recorded in the six tasks the least overall normalized mean peak AEMG \( (MP_{s,d,e}) \). Statistical significance-two-tail paired t-test. For the selected data, for each task \( d \) the cross-subject statistical significance of the

![Fig. 1. Bipolar electrode positions on extensor digitorum communis to index, medius, and ring fingers (ED2, ED3, ED4), extensor digiti minimi (EDM), extensor indicis (EI), extensor pollicis longus (EPL), abductor pollicis longus (APL), extensor carpi radialis brevis (ECRB), extensor carpi radialis longus (ECR), and extensor carpi ulnaris (ECU). Two electrode pairs at ED2, ED3, ED4, EDM, EI, and EPL are interfaced because of the narrow anatomical optimal ranges. In some subjects, wider ranges were explored. The most proximal ED3, pairs gave good electromyograms (EMG), the distal pair somewhat slower than the proximal. EI, electrodes might be placed even more ulnarily. On basis of anatomic proximity, large crosstalk is expected in ED4, and EDM, (mutually) and EPL, (from EI, not so much inversely with very ulnar placed EI), confirmed by results. With two electrode pairs, the pair recording least overall AEMG in all tapping tasks was selected for final data processing (see METHODS).](image)
mean peak EMG $MP_{d,e}$ was determined by a two-tail paired $t$-test. The null-hypothesis was that no differentiated EMG activity would exist in the recorded muscles in the finger tasks. The mean peak EMG recorded at nine muscles was thus individually paired across the subjects against the mean peak EMG recorded at the anatomical extensor ($ed$).

$$t_{score_{d,e}} = t_{test} \{MP_{d,e}, MP_{ed} \}$$

For each task, this constituted nine independent hypotheses on the EMG data for ten recorded muscles. Therefore the significance level was adjusted by the Bonferroni correction to $\alpha = 0.0056 = 0.05/n, n = 9$.

**TABLE 4. Tests for maximal voluntary contraction EMG**

<table>
<thead>
<tr>
<th>Muscle</th>
<th>ED2</th>
<th>ED3</th>
<th>ED4</th>
<th>EDM</th>
<th>EI</th>
<th>EPL</th>
<th>APL</th>
<th>ECRB</th>
<th>ECRL</th>
<th>ECU</th>
</tr>
</thead>
<tbody>
<tr>
<td>Isometric force</td>
<td>D2</td>
<td>D3</td>
<td>D4</td>
<td>D5</td>
<td>D2</td>
<td>MC1</td>
<td>MC1</td>
<td>MC2</td>
<td>MC2</td>
<td>MC4/5</td>
</tr>
</tbody>
</table>


**CORRELATION COEFFICIENTS.** For each task $d$, for each muscle pair $(m_i, m_j)$ with selected electrodes ($e_i, e_j$), the correlation coefficients of mean peak EMG across the subjects were calculated (Table 5)

$$cc_{d,i} = \text{correlation coefficient}(MP_{s,d,e}, MP_{s,d,e})$$

**ABSOLUTE AND RELATIVE CROSS-SUBJECT MEANS.** Further calculated were the cross-subject means and SD of the mean peaks (Fig. 3A)

$$MP_{s,d} = \text{mean} (MP_{s,d,e}) \quad (a)$$

$$StdP_{s,d} = \text{std} (MP_{s,d,e}) \quad (b)$$

**FIG. 2.** Raw EMG in *subjects 5 and 6* in the six tapping tasks (*x* axis: seconds). Clear EMG bursts with finger lifting, interspaced by silent periods when finger touches the table. *Subject 5*: T2: ED2 and EI equally active, significant accessory EMG (AEMG) only in EPL, some in ECRB, T3: minor AEMG in ED4, even less in others, but more in ECRB and ECRL. T4: major AEMG in EDM, significant in ED2, lesser in ECRB. T5: less AEMG in ED4 than in EDM in T4. ECU show also nonphasic activity. T1V: moderate AEMG in EDM, and ECU, T1H: moderate AEMG in EDM, ECU and ED2 (the latter presumably crosstalk from APL, see RESULTS). *Subject 6*: atypical results. T2: more AEMG in ED3 than in ED4, and considerably more in EDM compared with *subject 5*. D2 tapping of moderate strength, ED2 and EI equally active, in contrast with light or vigorous tapping (Fig. 5). T3: more AEMG in ED2 than in EDM in T4. T4: very large AEMG in EDM and ED4, than in *subject 5*. T4: very large AEMG in EDM and EI.
However, Eq. 6a gives a lumped average of two independent inter-subject variations: in absolute EMG amplitudes due to intersubject tapping intensity differences and in relative amplitudes of PEMG and AEMG, which is the interest of this study. To mitigate the first variations the cross-subject means were calculated for the ratios of the mean AEMG peaks over the mean PEMG peaks of the task finger extensor \((e_d)\) (Fig. 3B)

\[
MNP_{d,e} = \text{mean}
\left(\frac{MP_{e,d}}{MP_{e,d,e}}\right), \quad \text{StdNP}_{d,e} = \text{std}
\left(\frac{MP_{e,d}}{MP_{e,d,e}}\right)
\]

**RESULTS AND INTERPRETATION**

In the following, the experimental results and their interpretations are presented together. The interpretations are based on a multidimensional data set consisting of absolute and relative EMG amplitudes (Fig. 3, A and B, respectively), \(t\)-test results (indicated in Fig. 3A), EMG correlation coefficients (Table 5), selected scatterplots and trendlines (Fig. 4), anatomic proximity and variability in the muscles’ length and position (Leijnse et al. 2008) and location of their electrodes. The interpretations may serve as hypotheses for further investigations. Numeric AEMG values in text as percentage of PEMG are from Fig. 3B. Statistical values with discarded outliers are indicated (*).

**RAW EMG.** Two examples are given of raw EMG in the six tasks, representative of the generally obtained data (Fig. 2). Subject 5 was selected for closely reflecting the statistical average. Subject 6 presents a “worst case,” showing higher and atypical AEMG. Both subjects show clear extensor EMG bursts interspaced by silent periods, typical for the recorded EMG in all subjects.

**GENERAL FINDINGS.** For each task, a signature EMG pattern emerged in which amplitudes of PEMG were greater than of AEMG (Fig. 3, A and B). In tasks T2 and T1H, the synergists ED2 and EI, and EPL and APL, respectively, were both most active. However, AEMG in certain muscles can be substantial and may in individual cases exceed PEMG (e.g., EDM in T4, Fig. 3).

**STATISTICAL SIGNIFICANCE.** In all tapping tasks, for all ED parts and almost all other extensors, the null-hypothesis of undiversified EMG activity was rejected at the Bonferroni corrected significance \(\alpha = 0.0056\). The few non-significant values, indicated in Fig. 3A, correspond to, by hypothesis, synergistic muscles (mutually redundant ED2 and EI in T2, synergistic EPL and APL in T1H) or very large crosstalk (EI to EPLc in T2, ED4 to EDMc in T4) (see further).

**Mutual redundancy of ED2 and EI**

In index tapping (T2), great intersubject differences were observed in relative ED2c and EIc PEMG (Fig. 5). In some subjects, almost no ED2c PEMG was recorded, in others, almost no EIc PEMG, in others equal amounts. Remarkably, large intrasubject ED2c/EIc PEMG variability was also observed. In the same...
subject, relative ED2/EI, PEMG amplitudes remained generally constant for a given tapping modality. However, one or the other could become relatively greater with more or less vigorous tapping and/or changes in tapping trajectory (interphalangeal joints more or less flexed), and/or with practice, after which possibly sensitivity to precise electrode placement (e.g., on narrow muscle bellies), from underlying anatomic variability bringing muscles closer or further from electrodes on other muscles or from interindividual greatly variable coactivation. Generally, EMG correlations were least in T3 and highest in T1 and T1H. In T3, the low correlations support other data pointing to independent, isolated ED3 EMG activity. In the thumb tasks, the higher correlations may result from the greater complexity of thumb movement eliciting coactivations, and/or from more general crosstalk from the deep, central EPL (see further).

AEMG in individual muscles

**INTERPRETATION CRITERIA.** AEMG consisting of systematic coactivation and/or crosstalk in the population would between subjects positively correlate with PEMG (more vigorous tapping would induce higher AEMG across subjects).

**AEMG in ED parts**

**ED3** recorded no more than a very small 14 ± 10% AEMG in any of the given tasks (Fig. 3B). **ED2** received...
mild AEMG in T3 (23 ± 14%, and even half that without outlier, see Fig. 4A, T3), T5 (27 ± 8%), and T1V (22 ± 8%), but almost double in T1H (42 ± 15%; Fig. 4B, T1H) and 53 ± 22% in T4 (cc = 0.59, Fig. 4A, T4). ED4, received moderate AEMG in T2 (30 ± 19% of ED2 PEMG; Fig. 4A, T2) and T3 (28 ± 9%; Fig. 4B, T3), but about half of EDM PEMG in T5 (48 ± 14%; cc = 0.85, Fig. 4A, T5).

**INTERPRETATION.** First, the very low AEMG in ED3e means that ED3 was not significantly coactive in any tasks and that
FIG. 4. Cross-subject scatter plots, trend lines, and correlation coefficients (Eq. 5) of subject mean peak RMS EMG of selected muscles. Rows: finger tests. *Correlation coefficients without the outlier indicated by enlarged symbol. Positive associations point to systematic crosstalk (not necessarily from the x axis muscle) or coactivation. Large scatter (e.g., T4.D and T5. B–D) may correlate with variable anatomic proximity, sensitivity to electrode positioning on narrow muscles affecting crosstalk to the electrodes, and/or with highly individually different coactivation (see RESULTS).
its electrodes were by anatomic distance virtually isolated from crosstalk from other ED parts and finger extensors. Second, the 53 ± 22% ED2e AEMG in T4 and 30 ± 19% ED4, AEMG in T2 would be mainly mutual crosstalk due to anatomic proximity, as ED2 and ED4 are adjacent (Leijnse et al. 2008). If this AEMG was coactivation of ED2 with ED4 or inversely, ED3 would also likely be coactive at the same level because the middle finger is situated between index and ring fingers. However, ED3, AEMG in T2 and T4 is virtually nil. Crosstalk from EI to ED4 in T2 is unlikely, as they correlate negatively (cc = −0.36, Fig. 4B, T2). The large difference between the AEMG (crosstalk) from ED2 to ED4 in T2 (30%; cc = 0.22, Fig. 4A, T2) and inversely in T4 (53%; cc = 0.59, Fig. 4A, T4) might correlate with differences in muscle surface width. ED2 surface width is smaller than ED4 (Leijnse et al. 2008), so that ED4e could be placed further from ED2 than inversely. Third, the 42 ± 15% ED2e AEMG in T1H (Fig. 4B, T1H) would be mainly crosstalk from APL. Evidence was provided by the fact that of the two ED2e pairs, the distal pair recorded systematically more AEMG in T1H, sometimes almost double of the proximal pair (these distal ED2e were rejected for the final results). The distal ED2e were situated at ED2 more above where the APL crosses beneath ED2. The sensitivity of ED2e positioning to APL crosstalk also reflects in the ED2e AEMG scatter (Fig. 4B, T1H). Fourth, the 48 ± 14% ED4e AEMG in T5 (cc = 0.85, Fig. 4A, T5) would be mainly crosstalk from EDM. However, this AEMG may also contain signal from the ED5. The ED5 might be coactive in little finger lifting, while its muscle belly may proximally partly overlap with the ED4e (Leijnse et al. 2008).

**AEMG in other finger extensors**

**EDM, AEMG.** Although narrow, EDM is anatomically superficial and EDM PEMG could be well recorded. EDM AEMG was very small in T3 (13 ± 6%), mild in T1H, T1V, and T2 (29 ± 10, 33 ± 11, and 35 ± 12% of ED2 PEMG, respectively) but very large in T4 (93 ± 35%; cc = 0.41, Fig. 4C, T4).

**INTERPRETATION.** First, the EDM arises from the reverse side of the origin tendon sheet from which the ED4/5 muscle bellies arise. With the EDM on the narrow EDM close to ED4, large crosstalk from ED4 can be expected in T4, which would explain the main portion of the 93 ± 35% EDM AEMG. The weak correlation may reflect crosstalk sensitivity to EDM placement. Electrodes have been placed more or less ulnarily, i.e., more or less close to ED4, and were also placed somewhat variably in proximal-distal direction. Second, EDM AEMG in T2 correlates negatively with ED2e PEMG (cc = −0.34) but positively, although weakly, with EI PEMG (cc = 0.43; Table 5). This suggests that the EDM AEMG in T2 is mainly crosstalk from EI, which lays beneath EDM in the distal half of the forearm.

**EI, AEMG.** EI AEMG was very low in T3 (13 ± 7%), increasing to up to half of PEMG in T1H and T1V (39 ± 19 and 46 ± 20%, respectively), T4 (47 ± 30%), and T5 (52 ± 37%).

**INTERPRETATION.** First, the EIe AEMG (and EPLe AEMG) in T4 and T5 displayed large scatter with no correlation (Fig. 4, D, T4, and B and C, T5). We hypothesize this scatter is crosstalk correlating with the variable distal reach of the EDM and ED5 muscle bellies (Fig. 9 in Leijnse et al. 2008). Anatomically, the EDM and ED5 may not or may reach up to the EI. In the latter case, their muscle bellies lie between the deep EI and its electrodes (or near the EPL) and will cause large crosstalk. Second, the 46 ± 20 and 39 ± 19% EI AEMG in T1V and T1H, respectively, also has a relatively large scatter but higher correlation coefficients (cc = 0.46 and cc = 0.67, Fig. 4C, T1V/T1H). This EI AEMG is highly consistent between T1V and T1H tasks (cc = 0.84, Fig. 4D, T1V/T1H). Because the EI is unrelated to thumb function, we hypothesize that this EI AEMG is not coactivation but mainly crosstalk from EPL. The scatter would correlate with more or less ulnarily placed EI (further from or closer to EPL) and/or more or less distal reaching EPL muscle bellies (more EPL muscle bulk close to EI).

**EPL, AEMG.** The EPL muscle belly runs beneath the ED except for a short length radial to the ED tendons just proximal to the wrist. Thus it may amaze that generally at the latter location good PEMG could be obtained. However, equally high EPL AEMG was recorded (except in T3): 47 ± 25% in T4 (Fig. 4D, T4), 42 ± 22% in T5 (Fig. 4C, T5), and 55 ± 34% of ED2e PEMG in T2. The latter AEMG has no correlation with ED2e PEMG (cc = 0.05, Fig. 4C, T2), but a high correlation with EI PEMG (cc = 0.86, Fig. 4D, T2), so that crosstalk from the adjacent EI can be hypothesized (at a high level: trend line slope = 0.79).

**APL, AEMG.** The APL as placed in Fig. 1 are not in anatomic proximity of other finger extensors. This reflects in mild AEMG in all tests, with a maximum of 32 ± 15% in T1V.
ECRB<sub>e</sub> and ECRL<sub>e</sub>. ECRB<sub>e</sub> and ECRL<sub>e</sub> AEMG Radial wrist extensors AEMG was greatest in T3 at, respectively, 43 ± 25 and 43 ± 20%; a mild 27 ± 18 and 19 ± 11% of ED2<sup>2</sup> PEMG in T2, 35 ± 30 and 19 ± 11% in T4, and <20% in other tasks. In all tasks, ECRB<sub>e</sub> and ECRL<sub>e</sub> AEMG was remarkably highly correlated, ranging from cc = 0.80 in T5 to cc = 0.97 in T1V, although their relative amplitudes varied significantly between tasks (Fig. 3B). In contrast, ECRB<sub>e</sub> and ECRL<sub>e</sub> AEMG correlations with PEMG were relatively weak, or when higher, AEMG was small (T1V, T1H), except in T3 (cc = 0.70<sup>0</sup> and 0.75<sup>0</sup> of ED3 PEMG after exclusion of a common outlier, Fig. 4C, T3, while the insert between ECRB<sub>e</sub> and ECRL<sub>e</sub> AEMG was cc = 0.92, Fig. 4D, T3).

INTERPRETATION. The high ECRB<sub>e</sub> and ECRL<sub>e</sub> AEMG in T3 contrasts with their lower AEMG in all other tasks. Biomechanically no reason exists for significant coactivation of radial wrist extensors in T3. Therefore the T3 AEMG would be mainly crosstalk from the short, stout ED3 belly which is adjacent to both radial wrist extensor muscle bellies. Similarly, ED2 is adjacent to ECRB but not to ECRL, which agrees with greater AEMG in ECRB<sub>e</sub> than in ECRL<sub>e</sub> in T2. However, anatomic proximity does not explain the 35 ± 30% ECRB<sub>e</sub> AEMG in T4 (larger than in T2) as ED4 and ECRB are not adjacent. The scatter is large and there is no correlation with ED4<sub>e</sub> PEMG (cc = 0.02). One explanation might be that subjects compensated to various degree with wrist fixation while trying to independently lift the ring finger, which many found difficult.

EUC<sub>e</sub> AEMG. EUC<sub>e</sub> AEMG increases with ulnar finger tasks, from 21 ± 7% in T2 and 17 ± 7% in T3 to 38 ± 22% in T4 (cc = 0.12) and 56 ± 32% in T5 (cc = 0.28, Fig. 4D, T5). In the thumb tasks, EUC<sub>e</sub> AEMG was also relatively high, and highly correlated: 46 ± 18% of EPL<sub>e</sub> PEMG in T1V (cc = 0.91), and 40 ± 22% of EPL<sub>e</sub> PEMG in T1H (with, however, the greatest correlation with the synergistic APL<sub>e</sub> PEMG: cc = 0.93, Fig. 4D, T1H).

INTERPRETATION. First, in T4 and T5 EUC<sub>e</sub> AEMG, large scatter is present with low correlation, which may be related to the individually variable limited independent extension of D4 and also D5. The ECU may be involved in variable amounts of wrist bracing in support of lifting of these fingers. Specific to T5, crosstalk to ECU<sub>e</sub> from the adjacent EDM is also expected with additional variability due to variable placement of ECU<sub>e</sub> more or less ulnar, further or closer to EDM (EUC<sub>e</sub> positioning is complicated by the closeness of ECU to the table with the forearm in rest). Second, the high ECU<sub>e</sub> AEMG correlations with PEMG in T1V and T1H suggest ECU coactivation to compensate the radial abduction moments at the wrist caused by thumb action. However, crosstalk from the EPL is not excluded as its muscle belly reaches proximal halfway the forearm deep ulnarly up to the ECU muscle belly and its electrodes.

AEMG WITH EPL<sub>e</sub> PEMG IN T1H. The AEMG in ED4, EDM, EI, and ECU as a proportion of EPL<sub>e</sub> PEMG is slightly less in T1H than in T1V (Fig. 3B). This is contra-intuitive, as in T1H two motors (EPL, APL) are primary active instead of one in T1V (EPL). We therefore hypothesize that the recorded EPL<sub>e</sub> PEMG in T1H contains some crosstalk from the coactive APL/EPB. The EPB is adjacent to EPL and was not individually recorded. However, the EPB is likely to be as active as the APL in T1H, so crosstalk from EPB to EPL<sub>e</sub> is expected. Reducing EPL<sub>e</sub> PEMG in T1H to 85% of the recorded level, shifts the relative amplitudes of AEMG at the above-mentioned muscles to the same level as in T1V (Fig. 3B, T1H). This would suggest that about 15% of the recorded EPL<sub>e</sub> PEMG in T1H would be accounted for by crosstalk from EPB/APL.

**Detection of absent EI by surface EMG**

In one potential subject the EI was found absent. Suspicion was raised when in T2 no EI<sub>e</sub> PEMG could be obtained under any circumstance. Only a single tendon (of ED2) could be palpated proximal to the MCP joint (the EI tendon runs in a separate tendon sheath compartment and can generally be palpated separate from the ED2 tendon). Diagnosis was confirmed by ultrasound at the Dept. of Radiology of the University of Louisville. Because of EI absence, this subject was excluded from the study.

**DISCUSSION**

**MEASURING INDIVIDUATED EMG IN ED PARTS TO INDIVIDUAL FINGERS.** This study investigated whether the anatomical constant organization of the ED in distinct muscle parts to different fingers would allow individuated surface EMG assessment. In a population of widely varying length, age, and forearm skin thickness, individual finger tapping tasks elicited distinct, consistent, statistically significant EMG patterns. ED3 EMG could be obtained without substantial AEMG to or from other ED parts, indicating ED3 functions practically as a separate muscle. The same holds for ED2, whose activity was not associated with substantially more AEMG than would be the case with a narrow, anatomically distinct muscle, such as a wrist extensor. The ED4 was equally well individually measurable, but its activity was associated with higher AEMG in ED2 (presumably crosstalk, see RESULTS), while the highest AEMG was not within ED, but in the adjacent, slender, anatomically separate EDM.

It can therefore be concluded that individual EMG in ED2, ED3 and ED4/5 can be recorded by surface EMG in a rested forearm in much the same way as in anatomically separate muscles of the same dimensions. In conjunction with anatomic findings (Leijnse et al. 2008), these EMG results support that the ED2, ED3, and ED4/5 can be considered as functional entities. On basis of anatomic observations, the ED5 with its common insertions in both ring and little fingers would not be consistently functionally distinct from ED4 (Leijnse et al. 2008).

**LIMITATIONS OF THE STUDY.** As stated in the introduction, the forearm was at rest with the wrist maximally relaxed during the finger tasks. Therefore the data do not show crosstalk from the wrist extensors and supinator as would occur with active wrist and arm movements. Further limitations are the constant position of forearm pronation in which electrodes were placed and tests performed.

**ABSENT EI.** The finding of an absent EI in a potential subject correlates with anatomic literature. The ED2 and its tendon to the index are anatomically constant. In numerous dissections, the first author did not find gross anomalies. In contrast, the EI is anatomically more variable and accessory tendons or split muscle bellies inserting in other fingers (e.g., medius) are not rare (von Schroeder and Botte 1991). This study illustrates that
EI absence could in principle be diagnosed by surface EI EMG in an index tapping test, but the variability in the activity of EI—when present—in index tapping due to the mutual redundancy with ED2 is a highly confounding factor.

APPLICATIONS. This study demonstrates the feasibility of more detailed finger extensor surface EMG measurements than is commonly considered feasible. In doing so, the study confirms areas where needle electrodes may consistently assess individual ED2, ED3, ED4/5 and EDM activity with the prospect of recording individuated ED EMG without the crosstalk measured with surface electrodes. This may lead to more detailed research in ergonomic applications and motor control. Clinically, this knowledge could be useful for detailed assessment of extensor function and even of functional anatomy in selected cases, for example, in certain hand problems in musicians. For neuropathologies, the study confirms the anatomical basis for optimization of extensor stimulation electrode locations for restoration of hand opening in grasp (Leijinse et al. 2008).

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