Hand Muscle Activity for Digital Coartulation in Pianists

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Introduction

This study explored the variability across a group of normal, healthy pianists to provide a comprehensive description of the patterns of muscle activity used for various sequences of keypresses. The goal was to determine which features or combinations of features change across the various skill levels.

Table 1. Subjects’ ages, trainings, and performances

<table>
<thead>
<tr>
<th>Subject</th>
<th>Age</th>
<th>Experience with Keyboard</th>
<th>Time before and after keypress (ms)</th>
<th>Performance</th>
<th>Score</th>
</tr>
</thead>
<tbody>
<tr>
<td>Subject 1</td>
<td>18</td>
<td>10 years</td>
<td>50-100</td>
<td>Beginner</td>
<td>50</td>
</tr>
<tr>
<td>Subject 2</td>
<td>20</td>
<td>15 years</td>
<td>100-150</td>
<td>Intermediate</td>
<td>75</td>
</tr>
<tr>
<td>Subject 3</td>
<td>22</td>
<td>20 years</td>
<td>150-200</td>
<td>Advanced</td>
<td>90</td>
</tr>
</tbody>
</table>

Methods

Subjects: Ten pianists participated in the experiment: 4 professionals and 6 amateurs. Ages and training backgrounds varied across these subjects.

EMG: Electromyographic signals were recorded from 7 channels to characterize the activities of action potentials in spinal motoneurons. EMG data were recorded and averaged across 10 repeats of the same sequence.

Sequences: Pianists played several musical scores from which we selected 100 three-key sequences (Table 2). Sequences varied in pitches (P) as well as fingerings (F) causing the hand to change shape during the sequence. To characterize this, we calculated the change in postural index (PI) using the equation above.

IKI: The sequences were played with the right hand as a metronome signaled an inter-keypress interval of 125 ms.

PCA: Principal components analysis combined data from each EMG channel across the 22-15 sequences centered on each digit.

Table 2. Keypress sequences and postural index change (Amplitude of F + P on + P – F)

<table>
<thead>
<tr>
<th>Sequence</th>
<th>PI Change</th>
</tr>
</thead>
<tbody>
<tr>
<td>1-2-3</td>
<td>0.05</td>
</tr>
<tr>
<td>2-3-4</td>
<td>0.10</td>
</tr>
<tr>
<td>3-4-5</td>
<td>0.15</td>
</tr>
</tbody>
</table>

Results

The multi-muscle EMG pattern for each sequence could be reconstructed as a weighted sum of PCs. PC2 weighting coefficients differed for sequences involving hand opening or closing, providing evidence that the central agonist burst differed depending on which digits/keys were used before and after the central keypress.

Figure 1. Average EMG (sequence 2.3.4) for All Muscles.

Figure 2. Time before and after keypress (ms).

Figure 3. Averaged time before and after keypress (ms).

Figure 4. Time before and after keypress (ms).

Figure 5. PI change.

Figure 6. EMG bursts.

Conclusion

Ten normal subjects were found to be remarkably consistent in their patterns of coartulation. There were slight differences in playing speed (inter-keypress interval, IKI) that were associated with changes in burst duration and levels of antagonist/agonist coartulation (differing).

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Coarticulation

The duration of the central agonist burst, and the amount of antagonist/agonist coartulation tended to increase with the slight increases in inter-keypress interval that occurred across subjects (especially across the amateur subjects, open symbols).

Coactivation

The multi-muscle EMG pattern for each sequence could be reconstructed as a weighted sum of PCs. PC2 weighting coefficients differed for sequences involving hand opening or closing, providing evidence that the central agonist burst differed depending on which digits/keys were used before and after the central keypress.

Figure 7. Central agonist burst.

Figure 8. Central antagonist burst.

Figure 9. Central antagonist burst.

Figure 10. Central antagonist burst.

Figure 11. Central antagonist burst.

Figure 12. Central antagonist burst.

Figure 13. Central antagonist burst.

Figure 14. Central antagonist burst.

Figure 15. Central antagonist burst.

Figure 16. Central antagonist burst.

Figure 17. Central antagonist burst.

Figure 18. Central antagonist burst.

Figure 19. Central antagonist burst.

Figure 20. Central antagonist burst.