RESEARCHERS HAVE LONG EXAMINED the processes associated with response preparation in an attempt to determine how we perform the many complex tasks in day-to-day life. During this examination, there has been evidence that the preparation and performance of a movement is dependent on whether the goals of the task are expressed spatially or temporally. For example, the relationship between speed and accuracy of movement appears to be different depending on how movements are defined. In the original investigation of the speed-accuracy trade-off, spatial characteristics of targets were manipulated and participants were required to tap back and forth as quickly as possible (Fitts 1954). This manipulation produced a logarithmic relationship between the “index of difficulty” of the target and speed of movement. Alternatively, asking participants to perform a rapid aiming movement to a given target in a required movement time produced a linear relationship between speed and accuracy, whereby faster movements produced more variability of endpoint and thus less accurate performance (Schmidt et al. 1978, 1979). One reason given for the difference in the speed-accuracy trade-off was a difference in the movement goal, because in one scenario movement time is controlled, whereas in the other spatial accuracy is maintained and controlled (Zelaznik et al. 1988). The use of a spatial target encouraged participants to use a time-minimization control strategy vs. a temporal-precision strategy (Carlton 1994). More recently, through the collection of functional MRI and EEG data, it has been found that different neural activation patterns are involved in the timing of movement initiation compared with planning of the specific sequence of motor output (Bortolotto and Cunnington 2010; Bortolotto et al. 2011). These results provide additional evidence that the processes associated with when to produce a movement (i.e., temporal characteristics) are different to those associated with how to produce a movement (i.e., spatial characteristics).

Despite evidence that the process of preparation of a timed movement is different to that of a spatially defined movement, the underlying mechanisms of movement preparation under temporally controlled conditions are still unclear. It has been suggested that the “strategy” underlying the control of single-joint movements is dependent on how the goals of the task are represented (Gottlieb et al. 1989b). When movements of varying distance are required to be performed as fast and accurately as possible (known as speed-insensitive movements), the organizing principle of the nervous system involves modulation of the duration of motor neuron excitation (Gottlieb et al. 1989a). This is reflected in the duration of the electromyogram (EMG) burst of the agonist muscle. Conversely, when movements are required to be performed at different velocities (known as speed-sensitive movements), the organizing principle of the nervous system involves modulation of the amplitude or intensity of motor neuron excitation (Cortecos et al. 1989). This is reflected in a change to the initial slope of the rise of the EMG burst in the agonist muscle. Although there is evidence that different EMG variables appear to be modulated depending on the instructions given and nature of the movement, it is not known how this modulation is achieved by the nervous system. Rather than an explicit strategy being “chosen,” one suggestion is that the parameter modulations are “emergent properties of a particular motor program and experimental protocol” (Gottlieb 1993, p. 160). That is, when asked to perform a movement at maximal or submaximal velocity, the output of the movement is a consequential effect of a speed-insensitive or speed-sensitive strategy, respectively. Although this view is supported by examination of the motor output for

Motor preparation of spatially and temporally defined movements: evidence from startle

Dana Maslovat, Nicola J. Hodges, Romeo Chua, and Ian M. Franks
School of Kinesiology, University of British Columbia, Vancouver, British Columbia, Canada

Submitted 25 February 2011; accepted in final form 23 May 2011
given movements, it is not known whether the modulation of the EMG parameters is prepared and stored in advance of movement initiation or “emerges” due to some sort of feedback control. The purpose of this study was to compare preparation differences in temporally defined vs. spatially defined movements to determine if the modulation of EMG parameters is preprogrammed.

One of the more recent methodologies used to study advance preparation involves the use of a loud acoustic stimulus capable of eliciting a startle response (for recent reviews, see Carlsen et al. 2011; Rothwell 2006; Valls-Solé et al. 2008). During a simple reaction time (RT) task, where the required response is known in advance, replacing the “go” signal with a loud (≥124 dB) startling stimulus has been shown to elicit the prepared response at a much shorter latency. Given the dramatic reduction of premotor reaction times (<80 ms), it has been hypothesized that the startling stimulus can act as a trigger for a preprogrammed response, bypassing the usual voluntary command processes (Carlsen et al. 2004b; Valls-Solé et al. 1999). Studies employing a startling stimulus have consistently shown that the movement triggered during startle trials is similar in movement kinematics and EMG configurations to that of control trials. This has been shown for such diverse tasks as upper arm and wrist movements (e.g., Carlsen et al. 2004b, Maslovat et al. 2008, 2011; Valls-Solé et al. 1999), stepping and gait initiation (MacKinnon et al. 2007; Queralt et al. 2010; Reynolds and Day 2007), head rotations (Oude Nijhuis et al. 2007; Siegmund et al. 2001), sit to stand (Queralt et al. 2008), and rise to tiptoes (Valls-Solé et al. 1999). However, most of these experiments have used a spatially defined movement whereby participants move to a predetermined target as fast as possible. When a timing requirement is added to the movement, it appears that the startling stimulus triggers a movement with different characteristics than control trials (Maslovat et al. 2009). In the study by Maslovat et al. (2009), a startling stimulus was used during practice of a bimanual arm movement that required a 100-ms delay period between initiation of the limbs. Participants were able to perform this delay accurately in control trials, but the timing delay was dramatically shorter in startle trials. Examination of the muscle activation patterns revealed a difference in within-limb EMG timing for startle vs. control trials whereby the triphasic muscle burst (i.e., between initial agonist onset and antagonist onset and between antagonist onset and second agonist onset) was compressed in startle trials.

To explain why the startle trials produced movements that were compressed in time, the authors hypothesized that the addition of a precise timing requirement changed how the movement was prepared. To accurately delay a limb by 100 ms, participants would have relied on some sort of internal timer that was likely affected by the startling stimulus. Although most internal clock models are based on timing of relatively long intervals, evidence from event-related brain potentials suggests that at the neural level, timing can be regulated within the millisecond range (for a review, see Macar and Vidal 2004). The results and hypothesis of this study by Maslovat et al. (2009) were consistent with Block and Zakay’s (1996) attention-gate model of timing, whereby a pacemaker produces pulses at a given rate. When attention is focused on timing, a gate is “opened” to monitor the pulses of the pacemaker, which are accumulated until a threshold is reached. However, the rate of these pacemaker pulses is affected by the participant’s arousal level (Block and Zakay 1996; Triesman 1963). During startle trials, arousal is expected to increase, thus causing an underestimation of time duration (Maslovat et al. 2009).

Although a startling stimulus has been used to examine the preparation of between-limb timing in a bimanual movement, it has not been employed to investigate the preparation of time-constrained movements. In the current studies we used a startling stimulus with a unimanual movement performed at submaximal velocity as a new way to examine the modulation of EMG variables and determine whether different control parameters were prepared in advance for spatial vs. temporally based movements. If control parameters are prepared before movement initiation, we would expect to see modulation in both control trials and startle trials, because the startling stimulus is thought to trigger the prepared movement. We also examined whether there was evidence of reliance on a timekeeper for the time-constrained movements, because this would be reflected by movements compressed in time. Participants performed movements of different spatial amplitudes and temporal requirements, with startle trials interspersed with control trials.

For the spatially defined movements, we expected participants to preprogram motor commands that would result in different agonist burst durations for the various movement amplitudes (Gottlieb et al. 1989a). Because participants would not require a timekeeper to complete these movements, it was expected that the startling stimulus would trigger a movement with similar kinematics and EMG pattern compared with control trials but at much shorter onset latencies (Carlsen et al. 2004b). We predicted that the increased agonist burst duration with increasing movement amplitude would be present during control and startle trials, because these commands would be prepared in advance of movement execution.

For the temporally defined movements, we expected participants to preprogram motor commands that would result in a different rate of increase of agonist activation for the different movement velocities (Corcos et al. 1989). In addition, to perform the timed movement accurately, participants would be required to rely on an internal timekeeper whose pacemaker pulse would be accelerated on startle trials due to increased arousal level. Thus it was expected that the startling stimulus would trigger a movement at short onset latency with condensed kinematics and EMG pattern compared with control trials (Maslovat et al. 2009). Although we expected the modulation of agonist rise rate with changing movement velocity, we were unsure whether this effect would be observable on startle trials. Previous research has reported that a startling stimulus increases neural activation levels (Carlsen et al. 2004a), which has been shown to increase the rate of agonist rise (Maslovat et al. 2008, 2009). This increased activation may overshadow the differences in prepared intensity. In addition to independently examining the spatially and temporally defined movements, we also directly compared movements that were defined by different goals. We predicted that the use of a timekeeper for the temporally constrained movements would result in movement compression on startle trials compared with the spatially defined startle trials.
METHODS

Participants. Fifteen right-handed volunteers with no obvious upper body abnormalities or sensory or motor dysfunctions participated in the study after giving informed consent. However, only data from 12 right-handed volunteers [4 male, 8 female; mean age 20.9 (SD 1.5) yr] were employed in the final analysis. Three participants did not show consistent activation in the sternocleidomastoid (SCM) muscle during startle trials (which is thought to be the most reliable indicator of a startle response) and thus were excluded from the analysis (see Carlsen et al. 2011 for more detail regarding the exclusion criteria for participants). All participants signed an informed consent form and were naïve to the hypothesis under investigation. This study was conducted in accordance with ethical guidelines established by the University of British Columbia (UBC) and approved by the UBC Behavioral Research Ethics Board.

Apparatus and task. Participants sat in a height-adjustable chair in front of a 22-in. color monitor (Acer X233W, 1152 × 864 pixels, 75-Hz refresh) resting on a table. Attached to the table on the right side of the monitor was a lightweight manipulanda that participants used to perform horizontal flexion-extension movements about the right elbow joint. Participants’ arms and hands were secured with Velcro straps to the manipulanda with the elbow joint aligned with the axis of rotation and the hands semisupinated to grasp a vertical metal rod. The home position for each arm was located such that a 30° extension movement resulted in the arms being straight ahead (i.e., perpendicular to the monitor on the table) and was defined as 0°. Targets were located on the table top at 20°, 40°, and 60° of extension from the home position and were visible throughout the experiment.

In response to an auditory go signal, the participants were instructed to perform either a spatially defined movement to one of the three targets (day 1) or movement to the 20° target with a given timing requirement (day 2). Participants were instructed to look straight ahead at the monitor and respond by initiating a movement as fast as possible and performing the movement as accurately as possible. Accuracy was defined in terms of spatial error on day 1 and timing error on day 2. The timing requirement on day 2 for each participant was based on their performance on day 1. We took the mean time to peak displacement for each of the three targets as timing goals for the second day of testing. Thus the short movement on day 1 was comparable to the fastest movement on day 2, although the goal of the movement was represented in a different manner (spatially on day 1, temporally on day 2). The movement times to the medium and long targets on day 1 resulted in a moderate and slow movement to the short target on day 2.

Participants often perform rapid forearm extension movements by overshooting the target and rebounding slightly to end displacement at the intended position (e.g., Carlsen et al. 2004b; Maslovat et al. 2008, 2009). In addition, it often takes participants considerable time after peak displacement to reach a resting position due to oscillation around the target (i.e., over 350 ms; see Carlsen et al. 2004b). Our concern was that these extended movement times could possibly change the kinematics of how the movements would be performed on day 2, thus not allowing a between-day comparison of the short movement. For this reason we used time to peak displacement rather than the full movement time for the timing requirement on day 2. Participants were informed that feedback would be provided based on peak displacement of the movement produced.

Participants performed two testing sessions (~60 min) on consecutive days. Each testing session began with a maximal voluntary contraction of the agonist (triceps) and antagonist (biceps) muscles of the right arm to allow for between-day comparisons of EMG activity. Next, participants performed 3 blocks of trials, which included 10 practice trials and 46 testing trials. Each block contained movements to a single target (day 1) or time goal (day 2), and the order of blocks was counterbalanced between participants. The order of movements was maintained between days for each participant. Thus, if a participant practiced the short (20°), medium (40°), and long (60°) movement in order on day 1, their presentation order on day 2 would have been fast, moderate, and slow. During the testing phase of each movement, 6 of the 46 trials were startle trials, with no startles presented in the first 10 trials and no 2 consecutive startle trials. Augmented feedback was not provided during the trial; however, terminal feedback was provided for all trials on the monitor for 5 s following the trial, which included RT (in ms) and an accuracy score, expressed as a constant error (CE) of either peak displacement (day 1, in degrees) or movement time to peak displacement (day 2, in ms). To encourage fast and accurate responses, a monetary bonus was offered for fast RTs (both day 1 and 2) and accurate movements (spatially accurate on day 1 and temporally accurate on day 2).

All trials began with a warning tone consisting of a short beep (80 ± 2 dB, 100 ms, 100 Hz) presented simultaneously with the word “Ready!” and a visual precue in the form of text presented on the computer screen and visible throughout the trial. The visual precue on day 1 consisted of “SHORT (20°),” “MEDIUM (40°),” or “LONG (60°),” and on day 2 appeared as a specific time goal to reach the short target (based on day 1 movements; i.e., “Movement Time Goal = 200 ms”). The go signal followed the warning tone by a random period of 2,500–3,500 ms and could consist of either a control stimulus (80 ± 2 dB, 100 ms, 1,000 Hz) or a startling stimulus (124 ± 2 dB, 40 ms, 1,000 Hz, rise time <1 ms). All auditory signals were generated by a customized computer program and were amplified and presented via a loudspeaker placed directly behind the head of the participant. The acoustic stimulus intensities were measured using a sound level meter (Cirrus Research model CR:252B) at a distance of 30 cm from the loudspeaker (approximately the distance to the ears of the participant).

Recording equipment. Surface EMG data were collected from the muscle bellies of the following superficial muscles: right lateral head of the triceps brachii (agonist), right long head of the biceps brachii (antagonist), and right and left SCM (startle indicator) using preamplified surface electrodes connected via shielded cabling to an external amplifier system (Delsys model DS-80). Recording sites were prepared and cleansed to decrease electrical impedance. The electrodes were oriented parallel to the muscle fibers and then attached using double-sided adhesive strips. A grounding electrode was placed on the participant’s right ulnar styloid process. Angular displacement of the forearm was measured using potentiometers (Precision model MD157) attached to the central axis of the manipulanda, which had a precision of 0.07°/bit. A customized LabView computer program controlled stimulus and feedback presentation, initiated data collection at a rate of 1 kHz (National Instruments, PC-MIO-16E-1) 500 ms before the presentation of the go signal, and terminated data collection 2,000 ms following the go signal.

Data reduction. Analysis was restricted to the testing trials only (practice trials were not analyzed). A total of 42 of the 3,312 trials were discarded (1.3%). Reasons for discarding trials included displacement reaction time less than 80 ms (i.e., anticipation, 25 trials) or in excess of 500 ms (2 trials), movements to an incorrect target (13 trials), and startle trials in which no detectable startle response (SCM activity) was observed (2 trials). Because these trials were identified during data marking procedures, they were not repeated in the experiment; however, the low error rates ensured a sufficient number of trials for analysis in each condition. Overall, participants performed the movements with a high degree of accuracy on control trials as shown by the low overall grand mean of absolute error for peak displacement on day 1 (short = 0.8°, medium = 1.0°, long movement = 1.1°) and temporal error on day 2 (fast = 13 ms, moderate = 17 ms, slow movement = 21 ms).

Surface EMG burst onsets were defined as the point at which the EMG first began a sustained rise above baseline levels. The location of this point was determined by first displaying the EMG pattern with a superimposed line indicating the point at which activity increased to more than 2 SD above baseline (mean of 100 ms of EMG activity...
premiering the go signal). Onset was then verified by visually locating and manually adjusting the onset mark to the point at which the activity first increased. This method allowed for correction of errors of the algorithm. EMG offsets were marked in a similar fashion, with the activity between EMG onset and EMG offset being defined as the duration of a muscle burst.

Initial movement onset was defined as the first point of change of more than 0.2° of angular displacement from the starting position following the go stimulus, whereas peak displacement was defined as the first point at which displacement decreased following movement initiation. Final position was defined as the point at which angular velocity fell below 8°/s and remained below this value for 50 ms. As previously mentioned, we chose to use time to peak displacement as our criterion for movements on the second day because participants often require considerable time to slow their movements to under 8°/s for 50 ms. We felt this marker would ensure that the short movement on day 1 would be similar in kinematics to the fast movement on day 2. To calculate velocity, displacement data were passed through a digital, fourth-order Butterworth low-pass filter (cutoff frequency of 10 Hz) and then differentiated. Time to peak velocity and time to peak displacement were calculated from the time of displacement onset to maximal velocity and displacement, respectively. Total movement time was considered from displacement onset to final position.

**Dependent measures and statistical analyses.** All dependent measures were analyzed separately for the spatially based movements on day 1 and the temporally based movements on day 2 via a 2-stimulus type (control, startle) × 3-movement (short/fast, medium/moderate, long/slow) repeated-measures analysis of variance (ANOVA). We also compared the short movement on day 1 to the fast movement on day 2 via a 2-stimulus type (control, startle) × 2-day repeated-measures ANOVA. These movements were of the same amplitude (20°) but were defined spatially on day 1 and temporally on day 2. We did not compare the other movements between days because they were movements of different amplitudes.

The EMG pattern of rapid, single-joint movements is characterized by an initial burst of the agonist muscle (AG1) to provide the impulsive force to start the movement, followed by an antagonist burst (ANT) to provide the braking force, followed by a second agonist burst (AG2) to clamp the limb at the correct position (for a review, see Berardelli et al. 1996). Whereas AG1 and ANT bursts are typically consistent for a given movement, AG2 onset and duration are more variable and difficult to quantify, and this burst is not always present for slower movements (Berardelli et al. 1996; Wadman et al. 1979). For this reason, and because we were primarily interested in the movement to peak displacement, we chose to only examine the latency and duration of the AG1 and ANT bursts. EMG-dependent measures included time from stimulus onsets to AG1 onset (i.e., premotor RT) to determine whether the startling stimulus initiated the movement at latency values that would suggest the movement was prepared in advance and triggered by the startling stimulus. We also measured the relative timing between the onset of the first agonist burst and onset of the antagonist (AG1-ANT) and the duration of both burst durations. To quantify intensity of motor neuron excitation, we integrated the rectified raw EMG trace for the first 30 ms of the first agonist burst, which represents the initial slope of the rise in EMG (Q30) (Cordos et al. 1989; Gottlieb et al. 1989a; Khan et al. 1999; Maslov et al. 2008; 2009). To compare kinematics of the movements, we examined time to peak velocity, time to peak displacement, and total movement time.

For the repeated-measures ANOVAs, the Greenwood-Geisser ε factor was used to adjust the degrees of freedom for violations to sphericity. Uncorrected degrees of freedom are reported, with the corrected P values. Partial eta squared (η²) values are reported as a measure of effect size. The α level for the entire experiment was set at 0.05, and where appropriate, significant results were examined via Tukey’s honestly significant difference test and simple effects tests to determine the locus of the differences.

### RESULTS

A summary of the results for all dependent measures, including means and SD, are provided in Table 1. Rather than showing a single trial of an exemplar participant, we created ensemble averages for each condition (Fig. 1) showing dis-

---

### Table 1. Experimental results for each day, stimulus type, and movement

#### Day 1: Spatially Defined Movements

<table>
<thead>
<tr>
<th>Variable</th>
<th>Control</th>
<th>Startle</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Short (20°)</td>
<td>Medium (40°)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Premotor RT, ms</td>
<td>127.6 (17.9)</td>
<td>130.7 (17.0)</td>
</tr>
<tr>
<td>AG1-ANT time, ms</td>
<td>86.4 (25.5)</td>
<td>108.3 (27.7)</td>
</tr>
<tr>
<td>AG1 duration, ms</td>
<td>96.8 (16.2)</td>
<td>114.5 (15.7)</td>
</tr>
<tr>
<td>ANT duration, ms</td>
<td>92.4 (9.7)</td>
<td>106.1 (17.3)</td>
</tr>
<tr>
<td>AG1 Q30, mV · ms</td>
<td>1.52 (0.60)</td>
<td>1.64 (0.73)</td>
</tr>
<tr>
<td>Time to peak velocity, ms</td>
<td>74.5 (12.2)</td>
<td>96.3 (14.7)</td>
</tr>
<tr>
<td>Time to peak displacement, ms</td>
<td>161.1 (27.9)</td>
<td>206.8 (40.0)</td>
</tr>
<tr>
<td>Total movement time, ms</td>
<td>242.4 (32.4)</td>
<td>285.0 (42.3)</td>
</tr>
</tbody>
</table>

#### Day 2: Temporally Defined Movements

<table>
<thead>
<tr>
<th>Variable</th>
<th>Control</th>
<th>Startle</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Fast</td>
<td>Moderate</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Premotor RT, ms</td>
<td>124.6 (18.5)</td>
<td>128.5 (19.6)</td>
</tr>
<tr>
<td>AG1-ANT time, ms</td>
<td>81.5 (22.4)</td>
<td>91.4 (21.2)</td>
</tr>
<tr>
<td>AG1 duration, ms</td>
<td>100.6 (12.5)</td>
<td>107.2 (11.6)</td>
</tr>
<tr>
<td>ANT duration, ms</td>
<td>102.8 (12.1)</td>
<td>106.7 (11.7)</td>
</tr>
<tr>
<td>AG1 Q30, mV · ms</td>
<td>1.56 (0.87)</td>
<td>1.29 (0.62)</td>
</tr>
<tr>
<td>Time to peak velocity, ms</td>
<td>77.0 (10.3)</td>
<td>86.0 (12.7)</td>
</tr>
<tr>
<td>Time to peak displacement, ms</td>
<td>171.2 (26.4)</td>
<td>195.8 (36.8)</td>
</tr>
<tr>
<td>Total movement time, ms</td>
<td>233.1 (25.3)</td>
<td>256.0 (31.3)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Variable</th>
<th>Slong</th>
<th>Medium (40°)</th>
<th>Long (60°)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Premotor RT, ms</td>
<td>124.6 (18.5)</td>
<td>128.5 (19.6)</td>
<td>126.7 (16.2)</td>
</tr>
<tr>
<td>AG1-ANT time, ms</td>
<td>81.5 (22.4)</td>
<td>91.4 (21.2)</td>
<td>101.1 (21.8)</td>
</tr>
<tr>
<td>AG1 duration, ms</td>
<td>100.6 (12.5)</td>
<td>107.2 (11.6)</td>
<td>113.0 (14.0)</td>
</tr>
<tr>
<td>ANT duration, ms</td>
<td>102.8 (12.1)</td>
<td>106.7 (11.7)</td>
<td>117.9 (13.2)</td>
</tr>
<tr>
<td>AG1 Q30, mV · ms</td>
<td>1.56 (0.87)</td>
<td>1.29 (0.62)</td>
<td>1.08 (0.45)</td>
</tr>
<tr>
<td>Time to peak velocity, ms</td>
<td>77.0 (10.3)</td>
<td>86.0 (12.7)</td>
<td>96.5 (16.7)</td>
</tr>
<tr>
<td>Time to peak displacement, ms</td>
<td>171.2 (26.4)</td>
<td>195.8 (36.8)</td>
<td>239.0 (42.6)</td>
</tr>
<tr>
<td>Total movement time, ms</td>
<td>233.1 (25.3)</td>
<td>256.0 (31.3)</td>
<td>279.5 (34.1)</td>
</tr>
</tbody>
</table>

Values are means and SD (in parentheses). AG1, initial agonist burst (triceps); ANT, antagonist burst (biceps); Q30, rise in EMG activity; RT, reaction time.

---

**Note:** Values are means and SD (in parentheses). AG1, initial agonist burst (triceps); ANT, antagonist burst (biceps); Q30, rise in EMG activity; RT, reaction time.
placement (left) and velocity curves (right) to represent data from all trials from all participants (i.e., 480 trials for control, 72 trials for startle). This was achieved by normalizing each trial in time to displacement onset, which was considered time 0. These normalized averages are shown for control and startle trials for all three movements for spatially defined movements on day 1 (top), temporally constrained movements on day 2 (middle), and a comparison of the short movement on day 1 with the fast movement on day 2 (bottom). For all movements, the startling stimulus triggered movements with compressed kinematic profiles such that they were performed faster than control trials.

In addition to kinematic data, we have also provided ensemble group averages for rectified raw agonist and antagonist EMG activation for all conditions (Fig. 2). These graphs represent trials normalized to displacement onset (time 0) and EMG activation normalized as a percentage of the maximal voluntary contraction trials for control (left) and startle trials (right). On day 1 (spatially defined movements, top), movements were modulated by agonist duration, and on day 2 (temporally constrained movements, bottom), movements were modulated by agonist amplitude.

Premotor RT. As expected, the startling stimulus caused participants to initiate all movements at significantly shorter
Premotor RT values on both day 1 [F(1, 11) = 88.05, P < 0.001, η² = 0.89] and day 2 [F(1, 11) = 44.20, P < 0.001, η² = 0.80], as shown in Fig. 3A. Startle trials were performed at latencies short enough (M = 86 ms for both days) to suggest that a preprogrammed response was triggered, bypassing the usual voluntary command and cortical processing pathways (Carlsen et al. 2004b; Valls-Solé et al. 1999). Premotor RT was not significantly different for the various movements during control or startle trials, as shown by the lack of main effect for movement on day 1 (P = 0.342) and day 2 (P = 0.334). The analysis between the short movement on day 1 and the fast movement on day 2 showed only a main effect of stimulus type [F(1, 11) = 55.34, P < 0.001, η² = 0.83], because the premotor RTs did not differ for the two types of movement between days (F < 1).

Spatially based movements. A significant main effect for movement amplitude and stimulus for all EMG pattern-dependent measures (AG1-ANT, AG1 duration, ANT duration) revealed that the muscle activation pattern was compressed in time as movement amplitude decreased and by the startling stimulus. The lack of significant interaction effects showed that the startling stimulus produced a similar compression for all movements. We were most interested in the duration of the AG1 burst, because this is thought to be the variable that is modulated for movements of different amplitudes (Gottlieb et al. 1989a). As predicted, the main effect for movement amplitude [F(2, 22) = 80.01, P < 0.001, η² = 0.88] was due to a significant difference between all three movements (Fig. 3B, day 1). The lack of movement × stimulus type interaction confirmed this effect was present for both control and startle trials. For the analysis of EMG rise time, Q30 showed a main effect for stimulus [F(1, 11) = 13.43, P = 0.004, η² = 0.55] due to higher activation for startle trials compared with control trials (Fig. 3C, day 1). As expected, no effect of movement amplitude was found for Q30 (P = 0.723), confirming that agonist rise time does not appear to be a control parameter for movements performed as fast as possible.

Consistent with the EMG pattern results, analysis of the kinematic variables confirmed that time to peak velocity and time to peak displacement were performed faster for startle trials compared with control trials and for short-amplitude movements compared with long-amplitude movements (i.e., main effects for stimulus and movement). In addition, time to peak displacement showed a significant movement × stimulus type interaction [F(2, 22) = 4.58, P = 0.029, η² = 0.29], confirming that startle trials were compressed more for the long...
The movements were compressed at the kinematic markers of time to peak velocity and time to peak displacement, they were completed in a similar time course in both startle and control trials.

**Temporally based movements.** As with the spatially defined movements, a significant main effect for movement speed and stimulus was found for all EMG pattern-dependent measures, revealing that the muscle activation pattern was compressed in time for faster movements and by the startling stimulus. Although not predicted to be a control variable for temporally constrained movements (Corcos et al. 1989), the duration of AG1 showed a main effect for movement speed \(F(2, 22) = 7.47, P = 0.005, \eta_p^2 = 0.40; \text{Fig. 3B, day 2} \) due to a significant difference between the fast movement and slow movement, with the moderate movement not significantly different from the other movements.

As expected, the rise of EMG activity (Q30) was modulated to produce the various movement velocities, as shown by a significant effect of movement speed \(F(2, 22) = 4.92, P = 0.017, \eta_p^2 = 0.31 \) due to a higher Q30 for the fast movement compared with both the moderate and slow movements. Although the movement \(\times\) stimulus type interaction was not significant \((P = 0.122)\), examination of means (Table 1 and Fig. 3C, day 2) suggested that the main effect for movement speed was more prevalent on control trials. Q30 also showed a main effect for stimulus \([F(1, 11) = 21.56, P = 0.001, \eta_p^2 = 0.66] \) due to higher overall activation for startle trials compared with control trials.

In addition to confirming EMG rise time as a control parameter for timing-based movements, we were also interested in the effect of stimulus type and movement speed on the movement kinematics. Both time to peak velocity and time to peak displacement were differentially affected by the startling stimulus, as shown by a significant movement \(\times\) stimulus type interaction \([\text{time to peak velocity: } F(2, 22) = 6.65, P = 0.006, \eta_p^2 = 0.37; \text{time to peak displacement: } F(2, 22) = 6.76, P = 0.007, \eta_p^2 = 0.38]\). Both interaction effects were due to startle trials being compressed more for the slow compared with the fast movement, with the moderate movement not different from the other two movements.

Although the startling stimulus compressed the slow movements more than the fast movements, it did not result in participants performing all movements at the same velocity values (see Fig. 1, middle). To confirm this, we separately analyzed the startle trials of the three types of movements on day 2 and determined that all three movements were significantly different from each other with respect to time to peak velocity and time to peak displacement \((\text{both } P \text{ values } < 0.001)\). Total movement time showed a main effect for type of movement \([F(2, 22) = 9.53, P = 0.004, \eta_p^2 = 0.46]\) but was not affected by the startling stimulus \((P = 0.852)\). Thus, although the movements were compressed at the kinematic markers of

![Fig. 3](http://jn.physiology.org/)

Fig. 3. Mean (SE) data for premotor reaction time (RT; A), duration of first agonist burst (AG1; B), and the AG1 rise of EMG activity (Q30; C), separated by testing day, type of movement, and stimulus. *Main effect of stimulus type; ** main effect of movement type. Although all movements were triggered at short latencies (A), note the modulation of AG1 duration for day 1 (B) and the modulation of AG1 Q30 for day 2 (C).
between the testing days and, hence, the movement goals. The AG1-ANT time interval showed a main effect for day \([F(1, 11) = 5.71, P = 0.036, \eta^2 = 0.34]\), which was due to a significantly shorter time between AG1 and ANT for temporally constrained movements compared with spatially based movements. Most importantly, the time between AG1 and ANT showed a day \(\times\) stimulus type interaction effect \([F(1, 11) = 6.36, P = 0.028, \eta^2 = 0.37]\), which was due to more temporal compression during startle trials on day 2 (temporally based movements) compared with day 1 (spatially based movements) (Fig. 4). Because this difference in time between muscle burst onsets for startle and control trials was relatively small (8 ms on day 1 vs. 16 ms on day 2), and because the movement times were not identical for control trials between days, we performed a further post hoc analysis of the time between AG1 and ANT expressed as a percentage of the total movement. Again, a significant day \(\times\) stimulus type interaction was seen \([F(1, 11) = 8.38, P = 0.015, \eta^2 = 0.42]\), whereby the startling trials caused a significant reduction in time between AG1 and ANT for temporally defined movements (from 35% of the total movement time for control trials to 29% for startle trials), whereas no difference was found for spatially defined movements (from 35% of the total movement time for control trials to 33% for startle trials). For the kinematic variables, a comparison between the short movement on day 1 and the fast movement on day 2 showed a main effect for stimulus type only \((P < 0.001)\) for both time to peak velocity and time to peak displacement. No significant effects were found for total movement time.

**DISCUSSION**

We compared the preparation of temporally and spatially defined movements through the use of a startling stimulus and manipulation of the goals of the task. We predicted that spatially defined movements would be modulated by agonist burst duration, whereas temporally constrained movements would be modulated by agonist burst rise time (Gottlieb et al. 1989b). Overall, the results supported the use of different control parameters depending on how the movement was defined (Fig. 3, B and C). For spatially defined movements, the duration of the agonist burst was varied for the different movement amplitudes, and this effect was maintained in both startle and control trials. Rise time of the agonist burst did not appear to be a controlling variable for spatially based movements, because no differences were found between movement amplitudes. Conversely, for temporally based movements, the rise time of the agonist burst was modulated, because differences were found for the different movement velocities. Although this effect was not statistically dependent on the type of stimulus, there did appear to be a more consistent relationship between movement velocity and Q30 values for control trials compared with startle trials. This is not surprising, because EMG rise time was increased during startle trials, likely due to the increased activation associated with being startled (Carlsen et al. 2004a; Maslovat et al. 2008, 2009).

Temporally based movements also showed differences in agonist duration, which was not predicted; however, the magnitude of duration differences between movements was much smaller in the timing-based movements (8 ms) compared with the spatially based movements (30 ms). The startling stimulus triggered all movements, irrespective of how they were defined, at very short latencies (~85 ms, see Fig. 3A) that were consistent with other studies involving upper arm movements (e.g., Carlsen et al. 2004a, 2004b; Maslovat et al. 2008, 2009). Thus, although it has been suggested that modulation of EMG parameters is an emergent property of the experimental protocol (Gottlieb 1993), our results provide evidence that the preparation of motor commands that result in different control parameters occurred before the go signal such that they were prepared in advance and triggered by the startling stimulus.

We also tested for evidence in favor of a model of movement control for temporally based movements that utilizes an internal timekeeper. We expected startle trials for the spatially defined movements to trigger a movement with kinematic and EMG characteristics similar to those for control trials (Carlsen et al. 2004b), because no timekeeper would be required. For temporally constrained trials, we expected startle trials to trigger movements with condensed kinematics and EMG characteristics due to the use of an internal timekeeper whose pulse rate is accelerated by a startling stimulus (Maslovat et al. 2009). We found that the startling stimulus compressed both spatially and temporally defined movements (Fig. 1), thus only partially supporting our hypothesis. For the spatially defined movements, the startling stimulus affected all movements in a similar manner; however, for the temporally defined movements, the startling stimulus had a differential effect on the various movements, with slower movements compressed to a greater extent than faster movements.

Although we have attributed the proportional compression of timed movements (Fig. 1, **middle**) to the reliance on a timekeeper whose pulse rate is accelerated by the startling stimulus, it is possible that the differential effect of the startling stimulus was instead due to the nature of the slower movements. Increased activation caused by the startling stimulus could have had a greater effect on the lower velocity movements compared with the higher velocity movements due to a ceiling effect (i.e., the faster movements would already be performed closer to maximal velocity). To further examine whether reliance on a timekeeper was responsible for the observed effects, we directly compared the performance of the short movement
on day 1 and day 2, because these movements differed only in terms of how the movement goal was presented to the participants. On day 1, participants were asked to move as fast as possible to a spatial target of 20°, whereas on day 2, they were asked to move to a 20° target with a time goal similar to how they performed the movement on day 1. Consistent with the utilization of a timekeeper whose pacemaker pulse rate is dependent on arousal level, the startling stimulus had a differential effect when the movement was performed with spatial vs. temporal goals. The time between the first agonist burst and the antagonist burst was shorter for startle trials when movements were defined in a temporal manner (Fig. 4). The time difference between the first agonist burst and antagonist is a critical component of performing the movement correctly, because it determines when the braking force is applied to stop at the proper position and time (Wadman et al. 1979). It may be argued that differences in type of feedback provided between days (amplitude vs. timing error) and order effects (spatial movements were always performed on day 1) partially contributed to this effect. However, the difference in AG-ANT time showed robust differences whether examined in terms of absolute differences or as a percentage of total movement time.

An unexpected result was that the startling stimulus also compressed the spatially defined movements. Based on previous research (Carlsen et al. 2004b), we expected similar movement kinematics and EMG patterns for startle and control trials for spatially defined movements. In the current experiment the time to peak displacement for control trials was almost identical to those seen for spatially defined movements by Carlsen et al. (2004b). However, Carlsen et al. (2004b) did not show any movement compression for startle trials compared with control, although their variability for peak displacement was high on startle trials, which may have masked any effects of the startling stimulus. Similar to Carlsen et al. (2004b), the startling stimulus did not affect total movement time for spatially defined targets. This means that participants exhibited a longer time between peak displacement and movement completion on startle trials, which may have been due to participants attempting to slow their movements after the transient effects of the startling stimulus, which is thought to affect cortical processing for a brief period of time (Carlsen et al. 2004a).

The fact that both spatially and temporally defined movements were compressed for startle trials suggests that the startling stimulus affected movements with a specific timing requirement and those that were performed as fast as possible. This may be due to participants using a timing reference standard even when movements were performed without a specific timing goal or due to the startling stimulus having a different effect for the different types of movements. For spatially targeted movements, it has been suggested that a startling stimulus involves either a lowering of activation threshold for the involved muscles or increasing the activation accumulation rate such that the threshold is reached more quickly (Maslovat et al. 2011), which may explain the movement compression observed on day 1. However, when a timing component is required for the movement (day 2), there appears to be an additional effect of the startling stimulus on the timekeeper pulse rate, as shown by the differential effect for startle trials between the spatially and temporally defined movements (Fig. 4).

Support for a timekeeper that is affected by the state of the participant is consistent with the psychological literature examining factors that affect our internal timing mechanism. A number of studies have shown that when arousal level is increased, participants are prone to underestimating time intervals, which has been attributed to an increase in pacemaker pulse rate (Gruber and Block 2005; Meck 1996; Penton-Voak et al. 1996). In addition, distortions in time have also been reported due to attentional shifts by a distractor or unexpected event that changes the rate of information processing and thus the perception of elapsed time (Eagleman et al. 2005; Tse et al. 2004). Whereas these studies have typically involved perceptual discrimination or replication of a time interval, the current study indicates that a similar effect can be seen for movement preparation and execution.

In conclusion, we have provided evidence that temporally defined movements are prepared differently than spatially defined movements. Consistent with the theory presented by Gottlieb et al. (1989b), spatial movements were modulated by agonist duration, whereas timed movements were modulated by agonist rise time. The introduction of a startling stimulus compressed both spatially and timing-based movements; however, a greater effect was found for movements defined by a temporal goal and for slow movements compared with fast movements. These results are consistent with the hypothesis that timing-based movements rely on an internal timekeeper in which the pacemaker pulses are affected by the participant’s arousal level. Although it may not be surprising that movements with different goals are prepared differently, our results indicate that the control parameters were prepared before movement execution. With manipulation of the task goal and the use of a startling stimulus, we were also able to provide evidence that temporally defined movements not only involve different control parameters but also implicate an internal timekeeper, thus adding to our understanding of the preparation of timed movements.

ACKNOWLEDGMENTS

We acknowledge the assistance of Chris Forgaard for data collection and EMG marking and Paul Nagelkerke for technical support.

GRANTS

This study was supported by a Natural Sciences and Engineering Research Council of Canada grant (to I. M. Franks).

DISCLOSURES

No conflicts of interest, financial or otherwise, are declared by the author(s).

REFERENCES


