Exp Brain Res (2014) 232:3797–3802
DOI 10.1007/s00221-014-4074-y

RESEARCH ARTICLE

The early release of actions by loud sounds in muscles with distinct connectivity

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Received: 16 June 2014 / Accepted: 8 August 2014 / Published online: 21 August 2014

Abstract The presentation of an unexpected and loud auditory stimulus (LAS) during action preparation can trigger movement onset much sooner than normal. Recent research has attributed this effect to the activation of reticulospinal connections to the target muscles. To our knowledge, no studies have investigated the effects of LAS presentation in tasks requiring the simultaneous activation of muscles with different connectivity to motor areas of the brain. Here, we sought to establish the importance of muscle connectivity by asking participants to contract the orbicularis oris and abductor pollicis brevis muscles simultaneously. A LAS was randomly presented at 200 ms prior to the expected time of movement onset in an anticipatory timing task. We show that muscles controlled via bulbar connections to reticular formation can be triggered early by sound as much as muscles with spinal connections to the reticular formation.

Keywords Motor preparation · Muscle connectivity · Loud auditory stimuli

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Introduction

The presentation of a loud auditory stimulus (LAS) during action preparation can cause our movements to be initiated much sooner than normal. The latency of these reactions can be so quick that Valls-Sole et al. (1995, 1999) proposed that motor plans could be stored subcortically and triggered by the same circuits responsible for the startle reflex, in the reticular formation (Davis et al. 1982). This phenomenon has been termed the StartReact effect and is allegedly different from the well-known effects of stimulus intensity on reaction time (Cattell 1886), where movement initiation also becomes quicker as the intensity of the Go signal increases.

While the two response facilitatory phenomena may involve separate circuits (Carlsen et al. 2007), it is well documented that both share other commonalities. For example, it has been reported that acoustic stimulus intensities effects affect both response time and vigour systematically (Jaskowski et al. 1995). The same behavioural effect has been reported in the StartReact literature (Kumru and Valls-Sole 2006; MacKinnon et al. 2007; Marinovic et al. 2013; Rogers et al. 2011; Tresilian and Plooy 2006). This suggests that while separate, the mechanisms of response production may overlap more than previously believed for the two phenomena. Thus, we make no distinction between the StartReact effect and the stimulus intensity effect in the studies reported here. Rather, we were interested on how muscle connectivity could impact movement onset when a non-relevant LAS occurs.

Within the StartReact literature, Carlsen et al. (2009) reported that the effect of LAS on the release of motor actions is greater for arm muscles than it is for hand muscles. They attributed the larger effect on arm muscles to their stronger reticulospinal connections in comparison

with hand muscles. More recently, Honeycutt et al. (2013) provided evidence that the differential effect between arm and hand muscles may be due to task functionality as hand muscles can also have strong reticulospinal connections (Riddle et al. 2009). More specifically, Honeycutt et al. (2013) demonstrated that the effect on hand muscles can be larger if the task is more ecologically relevant (e.g. grasp vs. abduct the index finger). They suggested that more functionally relevant tasks may activate reticulospinal connections more strongly than less functional actions, an explanation that again emphasizes the importance of this particular pathway.

Interestingly, it has been demonstrated that functional movements typically engage distributed cortical networks (Graziano 2011; Graziano and Aflalo 2007; Graziano et al. 2005). Thus, functional actions may engage larger neural networks than single finger movements (Flament et al. 1993; Kouchtir-Devanne et al. 2012). The earlier release of actions reported by Honeycutt et al. (2013) when they used a grasping task as opposed to a finger abduction could therefore be related to the more distributed representation of grasping movements in the cortex. Alternatively, one could speculate that grasping movements are stored subcortically, whereas individuated fingers movements are not. At present, however, we are unaware of more direct evidence in favour of this alternative hypothesis.

While the subcortical triggering of motor plans is still under dispute (Alibiglou and MacKinnon 2012; MacKinnon et al. 2013; Marinovic et al. 2013, 2014; Nonnekes et al. 2014b; Stevenson et al. 2014), the suggestion that reticulospinal connections are important for the facilitation of movement initiation has been less debated (Carlsen et al. 2009; Honeycutt et al. 2013). Here, we tested this assumption by measuring the impact of loud sounds on the release of anticipatory timing actions requiring the simultaneous contraction of muscles with distinct connectivity (orbicularis oris and abductor pollicis brevis). We hypothesized that if muscle connectivity was important for the early released of prepared action, response initiation should differ for muscles with distinct connections to the reticular formation.

Methods and materials

Participants

Ten volunteers (two women) participated in the experiment reported here ($M = 26$ years). Participants gave written informed consent prior to commencement of the study, which was approved by the local Ethics Committee of the University of Queensland and in conformity with the Declaration of Helsinki on the use of human subjects in research. All of them stated that they were right-handed, had normal of corrected-to-normal vision and were free of any known neurological diseases.

Task

Participants were required to make a brief lip muscle contraction ("press your lips") at the same time they pressed a button on a response box using their right thumbs when the last of a sequence of four (500 ms apart) flashes (red, 200 × 200 pixels) appeared on a 19-in. monitor screen (60 Hz refresh rate, 1,280 × 1,024 resolution) as shown in Fig. 1. Participants were positioned 0.9 m away from the monitor. The duration of the flash was three frames (≈50 ms). Visual stimuli and sounds were generated with Cogent 2000 Graphics running in MATLAB 7.5. Feedback about temporal error was provided after all practice trials and only after Control trials during the experiments (i.e. trials without LAS). Feedback was based on the time difference between the appearance of the last flash and the onset of lip contraction. We choose to give feedback about the lip contraction in order to restrain variability along that aspect

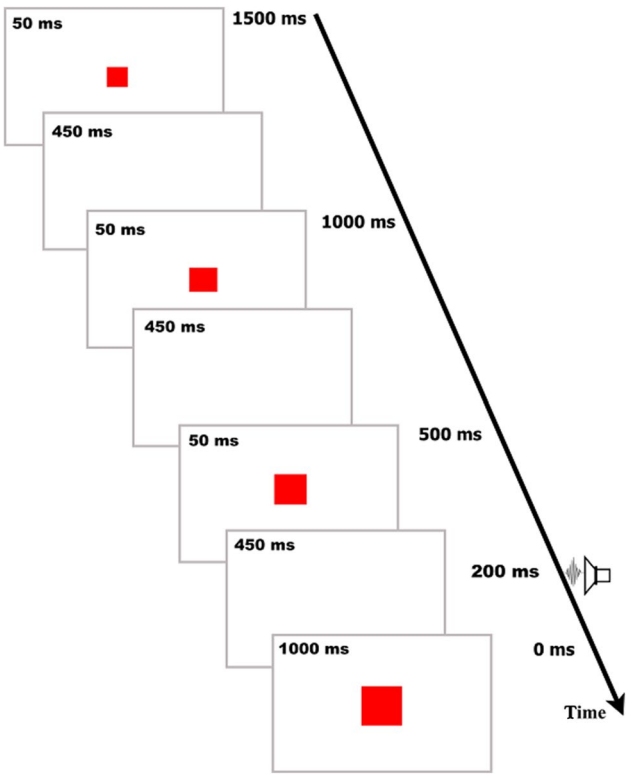

Fig. 1 Sequence of events during a trial. The first *flash* served as a warning signal and lasted for 50 ms. Subsequent *flashes* were presented 500 ms apart. The second and third *flash* also lasted 50 ms. The last *flashed rectangle* remained on the screen for 1,000 ms to mark the end of the trial. Unexpectedly, in some trials a LAS was presented between the third and fourth *flash* in shown in the diagram (color figure online)

of the task, which is probably less intuitive than button pressing responses that are more frequent in daily activities (e.g. playing computer games).

Procedures and design

Prior to practice trials, participants were given feedback about the type of contraction to be performed (quick and brief) on an oscilloscope. They were also given feedback about the EMG-relaxed state of the lips and right thumb. This biofeedback was provided to avoid background co-contractions that would make difficult to detect EMG onset reliably. Participants performed 40 trials during practise before the experimental block of trials. Feedback about the temporal error in relation to the lips contraction was provided to help participants to learn the correct time of movement onset. This feedback was based on the onset of voluntary EMG activity on the orbicularis oris muscle, and it is analogous to the premotor reaction time reported in other studies on the StartReact effect (Kumru and Valls-Sole 2006; Valls-Sole et al. 1999). During practice trials, we also made biofeedback available to the participants after each trial so that they were aware when the temporal error feedback was induced by background co-contractions. We did not enforce perfect synchronicity between lip and button pressing. Instead, we asked participants to perform the task so as that they perceived synchronicity was achieved. The participants performed 100 trials where they tried to synchronize their contractions with the last of a sequence of four flashes. On 10 of those, a LAS was presented, and participants were asked to ignore it and perform the task normally. Note that participants were to anticipate the occurrence of the last flash and, therefore, should not respond to it, making the Go signal an internal event (e.g. movement onset is not externally dictated). The order of presentation of trials was pseudo-randomized so as to avoid the fact that the LASs were presented sequentially. Feedback about temporal errors was provided in all trials except for those in which the LAS was presented. The onset time of the LAS was always 200 ms prior to appearance of the last flash as depicted in Fig. 1. This LAS presentation time is very close to the timing at which Carlsen and Mackinnon (2010) reported that responses were "startled" in virtually 100 % of the trials using similar anticipatory timing action.

Loud auditory stimuli

The LAS were bursts of 50 ms broadband white noise with a rise/fall time shorter than 1 ms. Stimuli were generated on a digital computer and presented binaurally via high-fidelity stereophonic headphones (Sennheiser model HD25-1 II; frequency response 16 Hz–22 kHz; Sennheiser Electronics GmbH and Co. KG, Wedemark, Germany). The input signal to the headphones had a bandwidth of approximately 10 Hz–30 kHz. Auditory stimuli had a peak loudness of 124 dB. The LAS intensity was measured with a Bruel and Kjaer sound level metre (type 2205, A weighted; Brüel and Kjaer Sound and Vibration Measurement, Naerum, Denmark) placed 2 cm from the headphone speaker.

Data analysis

EMG signals were recorded from the orbicularis oris (OO), right abductor pollicis brevis (APB) and sternocleidomastoid (SCM) muscles using disposable Ag–AgCl electrodes. The electromyogram (EMG) signal was amplified (1,000×), band-pass filtered between 30 and 1 kHz (Grass P511 isolated amplifier), sampled at 2,000 Hz, and stored on computer. The variables of interest were constant temporal error, peak EMG and time to peak EMG on the OO and APB muscles. SCM EMG activity was monitored to check whether or not "true startle responses"—SCM activation from 50 to 120 ms after LAS presentation—were observed. Temporal error was defined as the difference between appearance of the last flash and contraction of the OO and APB muscles (negative = early contraction). Movement onset was detected from the EMG signal of both muscles by a simple algorithm that measured when their activation exceeded two standard deviations from the rectified baseline activity. This movement onset detection algorithm used during the experiment gave similar results in comparison with an offline analysis of the filtered data using the movement onset detection algorithm developed by Teasdale et al. (1993). Peak EMG was defined as the maximum value of the rectified and filtered EMG signal (second-order Butterworth low pass in forward and reverse with a 50 Hz cut-off frequency) of the target muscles (APB and OO). Time to peak EMG was defined as the time between EMG onset and the time when the filtered EMG signal reached its peak. Nine trials (0.009 % of the total number of trials) were discarded from analysis because either the participants responded before the LAS stimulus (two trials) or we could not reliably identify EMG onset in one of the muscles (seven trials). The Shapiro–Wilk test was used to test for normality of the variables analysed. Means of temporal error, peak EMG and time to peak EMG obtained in Control trials and in LAS trials were found to be normally distributed and compared using paired *t* tests.

Results

As shown in Fig. 2, EMG activity in the OO muscle occurred sooner in trials where a LAS was presented ($t_9 = 5.21$, $p = 0.0005$, $r = 0.86$). The same pattern was observed for the APB muscle ($t_9 = 7.50$, $p = 0.00003$,

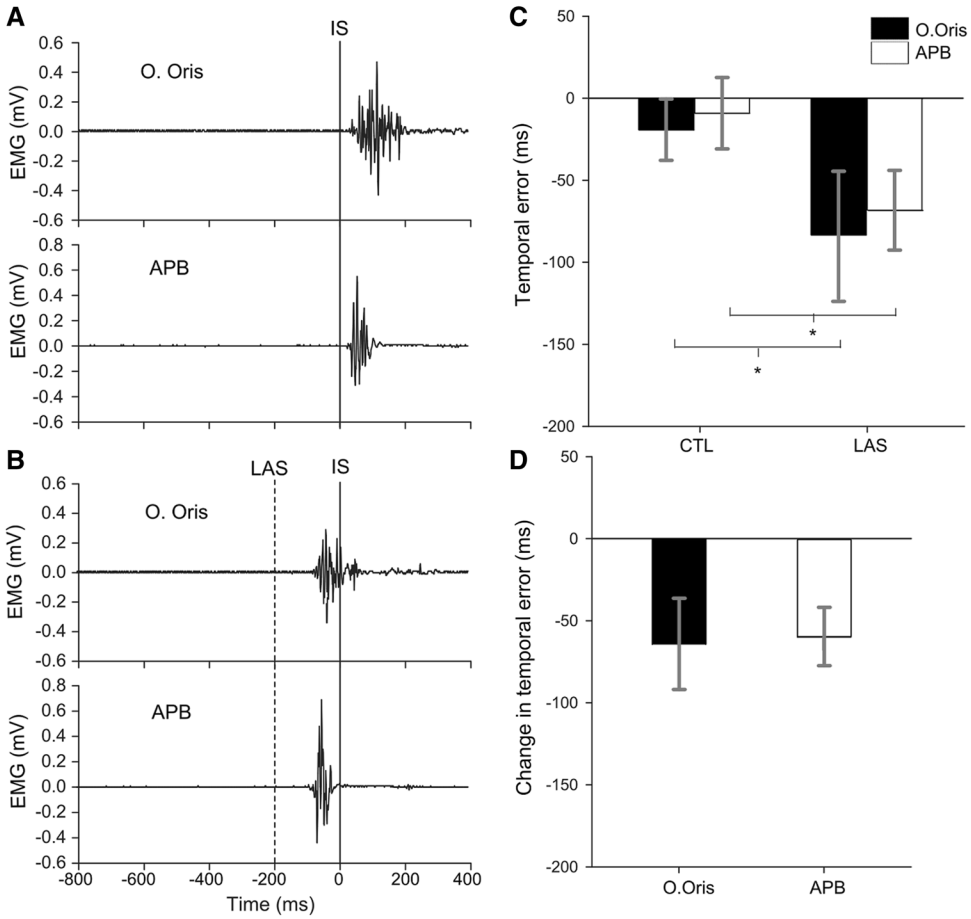

Fig. 2 Example of EMG activity in Control (**a**) and LAS trials (**b**). **c** Temporal error for OO and APB during Control and LAS trials. **d** Difference between LAS and Control trials for OO and APB. *Error bars* represent the 95 % CIs for the mean. *Statistically significant differences between means, $p < 0.001$

$r = 0.92$). Moreover, a comparison between the change in temporal error (LAS temporal error—control temporal error) for the OO and APB muscles failed to reveal a statistically significant difference between means ($t_9 = 0.56$, $p = 0.58$, $r = 0.18$, 95 % CI [−15.04, 25.04]; Fig. 2b). Furthermore, changes in temporal error for the OO muscle are linearly correlated with changes in temporal error on the APB muscle ($\Delta e_{OO} = 1.08\ \Delta e_{APB} + 0.02$, $R^2 = 0.48$, $p = 0.026$).

Table 1 provides the results for peak EMG and time to peak EMG for both OO and APB in Control and LAS trials. As shown in Table 1, we found no differences between Control and LAS trials regarding time to peak EMG. These results suggest that differences in temporal error found for OO and APB could not be explained by the combination of an initial reflexive response preceding a voluntary contraction as this would predict a longer time to peak EMG for LAS trials. For peak EMG, in contrast, we found a statistically significant difference between means for the APB muscle: As typically observed in other studies, the EMG amplitude of the responses was larger in LAS trials than in Control trials (see (Kumru and Valls-Sole 2006; Marinovic et al. 2013).

From the 10 participants, five had SCM activity between 50 and 120 ms after LAS onset. The proportion of trials in which SCM activity was observed for these participants ranged from 0.1 to 1 (mean = 0.44, SD = 0.42). Thus, for half of our participants, some of the responses were truly startled responses. Another three participants had SCM activity at slightly longer latencies ranging from 129 to 212 ms after LAS onset (proportions ranged from 0.4 to 0.6, mean = 0.5, SD = 0.1). The two remaining participants showed no signs of SCM activity. While the number of trials in which SCM activity was rather variable for some participants, further analysis of the seven participants that showed activation of the SCM muscle showed that the

Table 1 Means of EMG and temporal variables analysed

Variable	Control mean (SD)	LAS mean (SD)	*t*	*p*	*r*
O. Oris					
Time to peak EMG (ms)	106.2 (36.5)	116.9 (58.3)	0.77	0.46	0.24
Peak EMG (mV)	0.47 (0.05)	0.44 (0.07)	1.40	0.17	0.42
APB					
Time to peak EMG (ms)	65.8 (21.16)	68.9 (29.9)	0.60	0.56	0.19
Peak EMG (mV)	0.31 (0.22)	0.42 (0.28)	3.18	0.01	0.73

pattern of results was similar to our main analysis. For the OO muscle, trials with SCM activation (SCM+) where in average 14.5 ms (SD = 30.13, 95 % CI [−13.37, 42.36]) faster than those without SCM activation (SCM−). This small difference between means was not statistically significant ($t_6 = 1.27$, $p = 0.25$, $r = 0.48$) and the direction of the difference was not the same for all participants. For the APB muscle, SCM+ trials resulted in responses that were in average 22 ms (SD = 42.93, 95 % CI [−17.7, 61.7]) faster than SCM− responses. This difference between means (SCM+ vs. SCM−) for the APB was also non-statistically significant ($t_6 = 1.35$, $p = 0.22$, $r = 0.51$) and again the direction of the difference was not consistent across participants.

Most important to the purpose of our study, however, is the comparison between muscles. More precisely, the comparison between muscles for the differences in SCM+ and SCM− trials. While the APB muscle was activated 7.8 ms (SD = 34.4) earlier than the OO muscle in trials with SCM+, the difference between means for the two muscles was not statistically significant ($t_7 = 0.64$, $p = 0.53$, $r = 0.22$) and the 95 % confidence interval for the difference between means included zero as a possible value for the true difference (CI [−20.89, 36.63]). Moreover, for the eight participants (one participants more than the analysis for SCM+ vs. SCM− as one participants had SCM+ in all trials) that had SCM+ trials, and three had faster responses on the OO muscle than on the APB muscle. The reversed result was observed in the other five participants, and the differences between APB and OO onsets ranged from 1 to 64 ms for them, suggesting no clear cut advantage for a particular muscle to have its initiation facilitated by sound.

Discussion

Recent research has emphasized the importance of the reticulospinal pathway for the early release of motor actions by LAS (Carlsen et al. 2009; Honeycutt et al. 2013; see also Nonnekes et al. 2014b). This suggestion has never been tested directly, although Castelotte et al.'s (2007) showed early release of saccadic eye movements that do not rely on reticulospinal connections. Saccades, however, are a very particular case in that they are produced by brainstem pattern generators that are laid down during foetal development (Sparks 2002) and can be activated reflexively by acoustic and visual stimuli without forebrain involvement (see Leigh and Zee 2006, for a review). Thus, saccades can be produced by mechanisms entirely different from those involved in the production of other voluntary behaviours. Here, we investigated the reticulospinal hypothesis by comparing muscles with different connectivity in the same task. Participants performed an anticipatory timing task which required synchronous activation of the *orbicularis oris* and *abductor pollicis brevis*, which have different types of connections with the primary motor cortex. The OO muscle is controlled primarily via the corticobulbar and reticulobulbar pathways, while the APB is controlled mainly via corticospinal and reticulospinal pathways (Augustine 2008; Kuypers and Martin 1982). This allowed for a test of the importance of the muscular connectivity to the magnitude and/or manifestation of the effect.

We found that the OO muscle was activated much sooner than normal when a LAS was presented. More importantly, however, we observed an effect of similar magnitude for the APB muscle, and comparison of the change in timing revealed that the effect for the OO muscle was not statistically different than for the APB muscle. Furthermore, changes in temporal error for the APB muscle could predict changes in temporal error for the OO muscle, suggesting that these muscles were indeed affected similarly by the loud auditory stimulus. The same conclusion was achieved when we analysed responses with SCM+ and SCM− separately, indicating that the time advantage obtained in different muscles was not contingent upon the presence of an indicator of a startle response (SCM+) (see also Campbell et al. 2013; MacKinnon et al. 2007; Nonnekes et al. 2014a; Nonnekes et al. 2013; Reynolds and Day 2007; Rogers et al. 2011, for similar findings). These results suggest that muscle connectivity is not imperative for the manifestation, and for the magnitude, of the early release of responses elicited by sound. By doing so, our results motivate the need to reassess the interpretation of previous findings that were based on this connectivity hypothesis. For instance, earlier responses observed by Honneycutt et al. (2013) with grasping movements in comparison with isolated finger movements could be conditional upon the functionality of the task rather than on stronger reticulospinal activation. Functional movements typically engage distributed cortical networks (Graziano 2011; Graziano and Aflalo 2007; Graziano et al. 2005) and may thus engage larger neural networks than single finger movements (Flament et al. 1993; Kouchtir-Devanne et al. 2012). The earlier release of actions reported by Honeycutt et al. (2013) when they used a grasping task could therefore be related to the more distributed representation of grasping movements in the cortex. It is important to note that instead of diminishing the importance of the results reported by other groups, our results suggest an alternative interpretation that relies less on the specific connections to target muscles. Moreover, our results cannot be interpreted as evidence that the brainstem is not involved in the early activation of the OO muscle. It is safe to assume, however, that the particular pathways to the muscles studied here differ. The orbicularis oris can elicit reflex like responses as does the orbicularis oculi (Stevenson et al. 2014), but these

connections do not go through the spinal cord (e.g. reticulobulbar connections). The connections from the reticular formation to the abductor pollicis brevis are reticulospinal rather than reticulobulbar and mandatorily pass through the spinal cord. Moreover, we are unaware of any reports showing that LAS stimulation can elicit activation of this muscle within short latencies (<120 ms). Thus, even though these muscles have distinct pathways, they can be released similarly sooner than normal by acoustic stimulation. This indicates that advanced responses induced by sound may not rely on the type and/or strength of the connections to the reticular formation.

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