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Startle neural activity is additive with normal cortical initiation-related activation☆

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HIGHLIGHTS

- Subjects performed a simple reaction time (RT) task in response to an auditory cue.
- A startling acoustic stimulus (SAS) was presented during the RT interval.
- Results indicated that both voluntary and SAS initiation process jointly occur.
- We argue that an additive model of initiation-related activation can explain the results.

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ABSTRACT

The current study examined the process of response initiation in a simple reaction time (RT) task using a startling acoustic stimulus (SAS), which has been shown to trigger a prepared movement through an involuntary initiation pathway. The SAS was presented within the RT interval (concurrent with, and 25, 50, 75, 100, and 125 ms following the "go" signal), with the observed response latency used to examine the relative contributions of voluntary and involuntary activation to response initiation. Our results clearly indicate that both voluntary and startle-related initiation activation jointly contribute to the observed RT. The data support a model in which startle-related neural activity is additive with voluntary cortical initiation-related activation. This result also provides indirect support for the hypothesis that both voluntary and SAS-related involuntary activation involve a similar process of response output.

1. Introduction

In a simple reaction time (RT) paradigm it is unknown exactly when the response is to be performed, but knowing the required response in advance allows response selection and preparation to occur prior to the "go" signal. However, in these situations, RT values are considerably longer than what would be expected for pure stimulus detection, with the additional time interval thought to involve response initiation processes. The processes of response preparation and initiation have been recently described using a neural accumulation model [1] in which the preparation of a movement can be conceptualized as increasing activation of a neural network of cortical neurons to some level below threshold [13]. Initiation of the movement is then achieved through additional activation of the network beyond the "ignition point," leading to motor output (see [4] for a similar model involving saccade initiation).

The purpose of the current experiment was to probe the neural activation underlying the process of response initiation in a simple RT paradigm by using a loud acoustic stimulus, capable of eliciting a startle reflex. Previous work involving a startling acoustic stimulus (SAS) has shown that a pre-programmed movement can be triggered at a shorter latency by a SAS presented concurrent with the "go" signal via a faster, brainstem-mediated initiation process. In a normal (non-SAS) RT trial, the "go" signal is processed in sensory structures such as the primary auditory or visual cortices, leading to movement initiation through voluntary increases in neural activation. However, in a SAS trial, it is thought that a response that has been prepared in advance is initiated involuntarily by activation provided by neural circuits associated with the startle reflex. Thus a SAS can be used to determine if and

Abbreviations: ECR, extensor carpi radialis; FCR, flexor carpi radialis; RT, reaction time; SAS, startling acoustic stimulus; SCM, sternocleidomastoid.

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when substantial response preparation has occurred by examining whether the expected response was triggered at short latency (see [1,2,11] for recent reviews).

In the current study we presented the SAS at regular intervals *after* the "go" signal but before response onset (i.e., during the RT interval) to examine the effect of a SAS presented during the voluntary initiation process. Although the neural pathways involved in the startle reflex are well known, it is currently unclear how the SAS interacts with neural circuits to trigger the prepared response. One explanation for a shortened response latency in SAS trials involves increased activation of the reticular structures that are responsible for the startle reflex, suggesting sufficient detail of the movement characteristics are stored and triggered from subcortical structures including the brainstem and spinal centres [12]. For response initiation this subcortical triggering hypothesis would predict a "horse-race" between processes where response initiation would either occur from brainstem structures (resulting in startle-like RTs, relative to when the SAS was presented) or from cortical structures (resulting in control-like RTs), depending on whether the voluntary or SAS activation reached the prepared response first. Alternatively, it has recently been proposed that SAS may result in shortened response latency by the startle increasing motor cortical activation via an ascending reticulo-thalamo-cortical circuit; a faster pathway that results in the movement being initiated earlier from the same cortical neural network [1]. This hypothesis would predict that the voluntary and involuntary initiation processes may occur simultaneously, jointly contributing to response initiation-related activation.

2. Materials and methods

2.1. Participants

Data are presented from fifteen healthy participants (9F, 6 M; 24 ± 5 years) with no sensory or motor dysfunctions, who showed a consistent reflexive reaction to the SAS (see below). All participants gave written informed consent and reported normal hearing. This study was approved by and conducted in accordance with the ethical guidelines set by the Behavioural Research Ethics Board at the University of Ottawa and conformed to the latest revision of the Declaration of Helsinki.

2.2. Apparatus and task

Participants sat in a chair facing a 17 in. LCD computer monitor with their right arm resting in a custom manipulandum that restricted movement to wrist flexion and extension, with the forearm parallel to the floor and the palm facing inwards. The shoulder was abducted approximately 15°, and the arm was secured using Velcro straps placed proximal to the wrist and distal to the elbow. The task for the participant was to perform a ballistic 20° wrist extension movement from neutral (wrist neither flexed nor extended) "as quickly as possible" following an auditory imperative "go" stimulus. Feedback was provided on the computer monitor after each trial consisting of RT on that trial and accuracy with respect to the target. A points scheme was also provided to encourage fast RTs.

2.3. Instrumentation and stimuli

A warning tone (100 ms, 200 Hz) was followed by a variable foreperiod (2000–2500 ms), and finally an imperative "go" signal consisting of an 82 dB, 25 ms, 1000 Hz sine wave that was generated using digital to analog hardware (PCI-6024E, National Instruments). The signal was amplified and presented via a loudspeaker (MG Electronics M58-H, frequency response 300 Hz–11 kHz, rise time <1 ms) located 30 cm directly behind the participant at head height. Participants performed 5 blocks of 30 RT trials that emphasized fast reaction times in response to the sound.

In 20% of trials a startling acoustic stimulus (SAS), consisting of a 120 dB, 25 ms, white noise waveform (equal power from 1 Hz to 22 kHz), was presented at six different delay intervals (0, 25, 50, 75, 100, 125 ms) *following* the "go" signal. Stimulus intensity was confirmed using a precision sound level metre located at the same distance from the loudspeaker to the ears (Casella model CEL-254, A-weighted scale, impulse setting). Participants were told that on some trials they would hear a loud "static noise" sound that could be ignored. The SAS was presented pseudorandomly such that no two consecutive trials included a SAS, no SAS was presented in the first 2 trials of each block, and each SAS delay interval occurred in a random order, once in each 30 trial block. Participants performed up to two practice blocks of 10 trials (without SAS) to familiarize themselves with the task and equipment.

Surface electromyographic (EMG) data were collected from the muscle bellies of the right extensor carpi radialis longus (ECR), right flexor carpi radialis (FCR), and left sternocleidomastoid (SCM) muscles using bipolar preamplified surface electrodes connected to an external amplifier system (Delsys Inc.). Wrist angular position data were collected using a potentiometer attached to the central axis of the manipulandum. On each trial, unfiltered EMG and position data were digitally sampled at 1 kHz (National Instruments PCI-6024E via BNC-2090) for 3 s beginning 500 ms prior to the "go" signal using a customized programme written with LabVIEW software (National Instruments Inc.).

2.4. Data reduction and analysis

Peak displacement and velocity were defined as the points at which displacement and velocity decreased following displacement onset (angular displacement of more than 0.2°). Surface EMG burst onsets in all muscles were defined as the point at which the EMG first began a sustained rise 2 standard deviations above baseline levels (see [2] for details). Premotor RT was defined as EMG onset in the ECR muscle. To determine startle response incidence, trials were separated by whether or not an EMG burst was observed in SCM within 120 ms following SAS onset (indicative of startle related activity, see [2]). In order to investigate the effect of a startling stimulus on kinematic and EMG variables, only SAS trials where a startle response was observed in SCM were included in these analyses [2].

2.5. Statistical analyses

The proportion of trials in which an EMG response in SCM was elicited by the SAS was analyzed using a one-way, 6 factor (SAS delivery: 0, 25, 50, 75, 100, 125 ms), repeated measures analysis of variance (ANOVA), to determine if SAS presentation time led to any differences in startle response incidence. Prior to analysis proportion data were subjected to an arcsine square root transform to correct for violations to normality [7]. Similarly, premotor RT, peak displacement, time to peak displacement, peak velocity and time to peak velocity were analyzed using one-way, 7 factor (SAS delivery: none, 0, 25, 50, 75, 100, 125 ms), repeated measures ANOVA, to determine if there were differences in EMG onset and quality of movement produced. Greenhouse–Geisser corrected degrees of freedom were used to correct for any violations of sphericity. Differences with a probability of less than .05 were considered to be significant. Partial eta squared (η_p^2) is reported to provide an estimate of the proportion of the variance that can be attributed to the tested factor. Tukey's HSD