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# Considerations for the use of a startling acoustic stimulus in studies of motor preparation in humans

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**Abstract**

Recent studies have used a loud (>120 dB) startle-eliciting acoustic stimulus as a probe to investigate early motor response preparation in humans. The use of a startle in these studies has provided insight into not only the neurophysiological substrates underlying motor preparation, but also into the behavioral response strategies associated with particular stimulus-response sets. However, as the use of startle as a probe for preparation is a relatively new technique, a standard protocol within the context of movement paradigms does not yet exist. Here we review the recent literature using startle as a probe during the preparation phase of movement tasks, with an emphasis on how the experimental parameters affect the results obtained. Additionally, an overview of the literature surrounding the startle stimulus parameters is provided, and factors affecting the startle response are considered. In particular, we provide a review of the factors that should be taken into consideration when using a startling stimulus in human research

*Keywords:* Startle response, preparation, EMG analysis, reaction time

## Introduction

Motor preparation and programming in humans has traditionally been investigated in studies involving methods such as reaction time (RT) measurement (e.g. Donders, 1969), mechanical blocking (Wadman et al., 1979), or via neurophysiological techniques such as electroencephalography (Kornhuber and Deecke, 1965) and transcranial magnetic stimulation (MacKinnon and Rothwell, 2000). In recent years, however, an acoustically evoked startle response has been employed as a tool to investigate preparatory motor processes. This has mainly been accomplished by presenting a loud (e.g., 124 dB) acoustic stimulus as a probe at some point in time prior to the required action in targeted RT tasks to look for changes in RT and movement production. In particular, when a startling acoustic stimulus (SAS) was presented simultaneously with the imperative "go" stimulus in RT and other movement tasks, the prepared and intended action was released at a significantly shorter latency than in control trials (Carlsen et al., 2003a, 2004a, 2004b, 2007, 2009a; Castellote et al., 2007; Cressman et al., 2006; Kumru and Valls-Solé, 2006; MacKinnon et al., 2007; Maslovat et al., 2008; Oude Nijhuis et al., 2007; Reynolds and Day, 2007; Siegmund et al., 2001; Tresilian and Plooy, 2006; Valls-Solé et al., 1995, 1999, 2005). It has been suggested that these short latency (<70 ms) reactions reflected the startle acting to release a pre-programmed response without the usual cortical trigger (Carlsen et al., 2004a, 2004b; Valls-Solé et al., 1999). While there exists continued debate with respect to the exact mechanism that results in the release of the response, this paper does not aim to resolve this issue (for more on these issues see Carlsen et al., 2004b, 2007, 2009a; Rothwell et al., 2002; Rothwell, 2006; Valls-Solé et al., 2008). Rather, here we will focus on methodological issues associated with the use of a startle paradigm, some of the recent findings with respect to its use, and how to best achieve a robust startle response in participants. In order to address some of these concerns and to propose a more standardized startle method, a discussion of factors related to the presentation of the startling stimulus and how these factors affect the response observed will be presented. Following this, some remarks pertaining to all of the relevant findings will form the basis of some recommended parameters for stimulus presentation and data analysis when using startle to investigate motor processes. First, however, this paper will review the literature investigating the nature of the startle response in humans, including the neurophysiological structures and pathways involved and will summarize some

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of the recent literature with respect to the use of a SAS in movement tasks. These topics will be presented under the headings below:

- Section 1: Early release of a movement by startle
- Section 2: The startle response
- Section 3: Detecting a startle response: EMG
- Section 4: Acoustic stimulus parameters: Effects on the startle response
- Section 5: Other factors affecting the startle response

### 1. Early Release of a Movement by Startle

Using a RT paradigm, it was reported that premotor RT (time from stimulus presentation to EMG onset) was significantly reduced when participants were startled by an unexpected loud acoustic stimulus (130 dB) presented in conjunction with a visual "go" signal (Valls-Solé et al., 1999). Based on these findings, Valls-Solé et al. suggested that "the whole motor programme can be triggered [by the startle] without the expected command from the cerebral cortex" (1999, p. 937). This conclusion was based on two results from the startle condition: First, the observed premotor RT was very short compared to the control (no startle) condition, and second, EMG activity appeared to be unmodified from this control condition. In the fastest reactions observed by Valls-Solé et al., premotor RT was 65 ms. In a typical RT paradigm, RTs of 180 ms are commonly observed in response to visual stimuli, while RTs of 140 ms or more are commonly observed in response to auditory stimuli (Brebner and Welford, 1980). It was argued by Valls-Solé et al. (1999) that actions initiated in less than 70 ms (i.e. premotor RT < 70 ms) were unlikely to have involved the cortex in their initiation due to fixed amounts of time needed to convert the acoustic stimulus to neural signals and for neural transmission. Thus, they suggested that sufficient details of a prepared movement may have been stored in the brainstem and spinal centres so that it could be, in some cases, initiated subcortically and released early.

Since the EMG activity for wrist movements (flexion or extension) retained their characteristic triphasic profile through both control (no startle) and experimental (startle) trials, the prepared movements appeared to be elicited unaltered. More importantly, since the observed EMG patterns were unchanged, Valls-Solé et al. (1999) believed that the observed fast response was not produced by an early startle reflex adding on to a later voluntary response. More evidence for this viewpoint was provided by Carlsen et al. (2004b), who showed that neither EMG patterns nor response kinematics were significantly changed in startle-speeded responses compared to their control counterparts in a RT task involving an arm extension to fixed targets located at 20, 40, or 60 degrees of angular extension. Irrespective of the fact that the observed RT was significantly shortened to a mean of 70.2 ms in the presence of a startle, no modifications to the response kinematics or EMG patterns for any of the target distances were evident. Example kinematics and EMG data for arm movements to a 60 deg target under control and startle conditions are shown in Figure 1. These data confirmed the suggestion that the response that was elicited by the SAS at short latencies was in fact the intended prepared response. This pattern of response speeding by startle is not limited to tasks involving the upper limb (e.g., Carlsen et al., 2004a, 2004b, 2007; 2009a, 2009b; Cressman et al., 2006; Kumru and Valls-Solé, 2006; Maslovat et al., 2008, 2009a; Tresilian and Plooy, 2006; Valls-Solé et al., 1995, 1999), but has also been shown for tasks such as for stepping and anticipatory postural adjustments prior to stepping (MacKinnon et al., 2007; Nieuwenhuijzen et al., 2000; Reynolds and Day, 2007) , rising onto toes (Valls-Solé et al., 1999), head rotations (Oude Nijhuis et al., 2007; Siegmund et al., 2001, 2008), eye movements (Castellote et al., 2007) and sit-to-stand movements (Queralt et al., 2008).

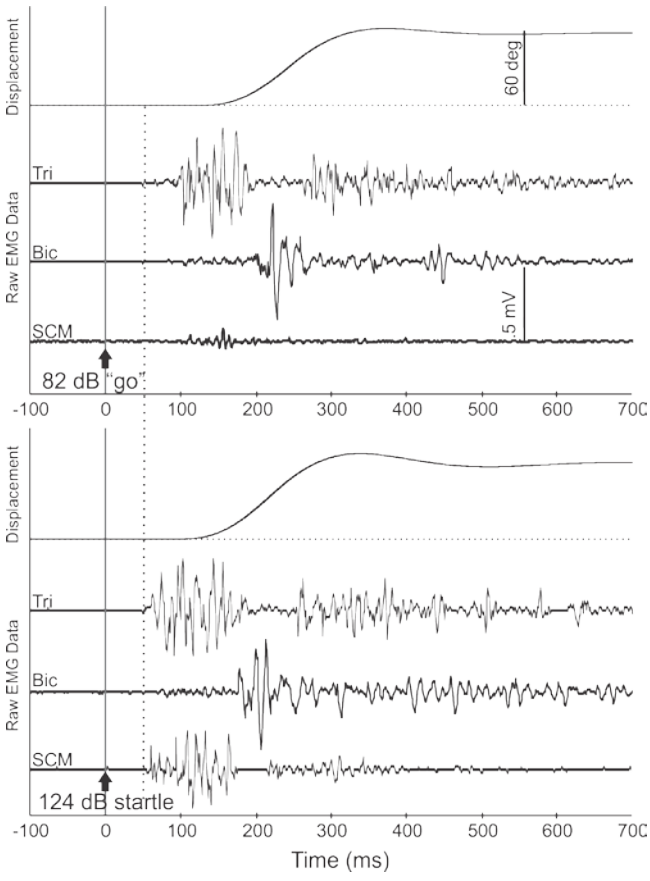


***Figure 1. Raw kinematic and EMG data from a single participant making a 60 deg arm extension movement to a target in an auditory cued simple reaction time task. Top panel (A) is a control trial where an 82 dB tone served as the "go" signal, and bottom panel (B) is a startle trial where a 124 dB tone was given in place of the normal "go" signal. Time zero is stimulus onset. Displacement (deg) and raw EMG from triceps (Tri), biceps (Bic), and sternocleidomastoid (SCM) are shown. Note that although RT latency was shortened in the ST trial, triphasic***

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***EMG configuration and kinematics were unaffected. Adapted from: Carlsen, A.N., Chua, R., Inglis, J.T., Sanderson, D.J., Franks I.M., 2004a. Prepared movements are elicited early by startle. J. Motor Behav. 36, 253-264. Copyright ©2004 Heldref Publishing Limited.***

A simple RT task theoretically allows an individual to complete response selection and programming in advance of the "go" signal since the required response is certain. However, in choice RT tasks the required response is provided by the "go" signal, making it necessary for response selection and response programming to occur during the RT interval (i.e. following the "go" signal). In some cases it may be possible to prepare part of an uncertain response in advance (e.g., Rosenbaum, 1980), however, in a pure choice RT paradigm (particularly involving mutually exclusive movements) this is not generally the case (see Carlsen et al., 2009b). In order to confirm that only responses that were organized in advance of the "go" signal (i.e. pre-programmed) could be speeded by a SAS, Carlsen et al. (2004a) conducted an experiment where a SAS was presented in both simple RT and choice RT tasks. Results of this experiment showed that during a simple RT task, premotor RT was dramatically shortened from 142 to 86 ms when a startle response was present. In contrast however, in a choice RT condition where the correct response had to be selected during the RT interval, a startle did not shorten RT (Control: 204 ms, Startle: 203 ms). Several studies have shown that some limited response speeding due to startle can occur in a choice RT situation (Kumru et al., 2006; Oude Nijhuis et al., 2007; Reynolds and Day, 2007), suggesting that some preparation can occur in these situations (e.g., increased excitability of subcortical pathways, Kumru et al., 2006). Nevertheless, these results strongly suggest that the majority of the facilitatory effect of the startle during simple RT was not due to faster response propagation, and was more likely due to the involuntary triggering of a prepared response (Carlsen et al., 2004a).

Importantly, the use of a SAS in a choice RT paradigm also showed that startle could be used as a probe to determine under what stimulus conditions a response is pre-programmed or not. For example, the aforementioned results provided evidence that during a choice RT task participants did not prepare a response in advance due to the uncertainty regarding the required upcoming movement, as no early movement was elicited by the SAS. However there has been some evidence provided that response preparation can occur when there are multiple response possibilities. Using a precuing RT paradigm Carlsen et al. (2009b) showed that when a subset of possible responses was cued in advance of the go signal, participants adopted a strategy whereby multiple movements were pre-programmed in order to allow for the quickest possible response. Possible responses involved targeted flexion or extension with either the left or right wrist. When there were only two response options and they were lateralized (e.g., either flexion with the left hand or extension with the right), both movements were elicited by a SAS at short latency. Importantly, the movements elicited by SAS always reflected the precued response options. However, on the contrary, there is evidence that knowing the required response in advance does not guarantee advance preparation. For example, in a task often termed a "go/no-go" RT task, the required response is known in advance (and thus can be theoretically pre-programmed), and the imperative stimulus simply informs the subject whether or not to make the movement (see Donders, 1969). While some response speeding was seen when a SAS was presented in this type of task (Kumru et al., 2006), the movements were not triggered at startle-like latencies (Carlsen et al., 2008a). This suggested that a go/no-go task was treated more like a choice RT task where the response was not generally pre-programmed, but programming occurred following the "go" signal.

For many simple RT situations, the use of a SAS probe has shown that movements appear to be programmed in advance of the "go" signal regardless of the type of movement involved (Carlsen et al., 2004b; Castellote et al., 2007; MacKinnon et al., 2007; Oude Nijhuis et al., 2007; Siegmund et al., 2001, 2008). However, a SAS has also recently been used to determine *when* (or if) a response is pre-programmed depending on the type of temporal information provided. For example, in the timing of the "go" signal with respect to a warning cue (i.e. foreperiod) has been thought to modulate when attention is directed toward the signal. Specifically, previous studies have found that for a given range of foreperiods, comparatively short or long foreperiods result in the longest RTs, whereas the shortest RT is achieved when the "go" signal is presented in the middle of the range of possible foreperiods (Cressman et al., 2006; Drazin, 1961; Mowrer, 1940; Niemi and Näätänen, 1981). This RT effect was thought to result either from a change in response preparation based on estimates of the required time to respond, or from processes other than response programming, such as readiness to perceive the stimulus and / or attention. However, when a SAS was presented in a RT task involving a range of different foreperiods, the RT differences were eliminated (Cressman et al., 2006). The authors concluded that the movement was fully prepared throughout the variable foreperiod, and thus RT differences observed must be due to effects other than changes in response programming. Similarly, in a fixed (3 sec) foreperiod or variable (2-3 sec) foreperiod simple RT task, a planned movement was elicited at short latency when the SAS was presented up to 1500 ms prior to the "go" signal (Carlsen and Mackinnon, 2010; MacKinnon et al., 2007). However, the incidence of movement release was reduced compared to when a SAS was presented simultaneous with the imperative "go" signal, but increased as the timing of the SAS approached the "go" cue. These findings suggest that for a semi-unpredictable simple RT tasks the planned movement is progressively constructed starting well in advance of the intended movement onset in order to allow for quick

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reactions. In contrast, however, a very different result was seen when a SAS was presented in a task where the response was to be timed accurately with the arrival of a clock hand at a target. Specifically, a SAS presented at various times in advance of the target in an anticipation-timing task, did not trigger an early response even when applied between 500 ms and 200 ms prior to the target (see also Carlsen et al., 2003b, 2008b), suggesting that for these types of timing tasks it may not be critically important or strategically beneficial to prepare a response well in advance of the required target. Thus, a SAS can not only inform about what is pre-programmed, but also *when* it is programmed.

Finally, startle has recently been used to infer changes to pre-programming as a result of learning. For example, Maslovat et al. (2008) required participants to practice a bimanual movement of asymmetrical amplitudes, whereby participants simultaneously extended their right arm 20 deg to a target and their left arm 10 deg to a target. Prior to and following practice, startle trials were interspersed with control trials to examine the effects of practice on the preparation of this movement. The comparison of startle to control trials indicated that a different amplitude movement could indeed be prepared in advance for each limb, and this preparation improved with practice. Importantly, a more accurate movement was produced following practice, and the startle stimulus triggered a similar movement compared to control trials (in kinematics and muscle activation pattern) both early and late in the acquisition process. In a follow up study, Maslovat et al. (2009a) examined preparation changes with practice of an asynchronous bimanual movement, whereby participants extended both limbs to a 20 deg target but the left limb was delayed by 100ms relative to the right limb. Again, startle trials were interspersed during the acquisition process to determine how preparation changed with practice. The results indicated that although a timing delay was still present in startle trials, it was consistently shorter than control trials. With practice the delay in both startle and control conditions became closer to the target but the difference between trial types was maintained. This was taken as evidence that movements involving between-limb timing can be prepared in advance and improved with practice; however, the response triggered by the startle is not the same as that in control trials. The authors explained the timing disparity by hypothesizing that the startle acted to speed up the pulse accumulation of the participant's internal timekeeper (Block and Zakay, 1996) and thus produce a movement with a shortened delay interval. Collectively, these studies show that the effects of a startle can also be used to investigate changes in response programming that occur during motor skill acquisition. Thus, considering the above experiments, it is clear that the effect of startle to trigger a movement is not a limited epiphenomenon, but is a useful tool that can be used to investigate motor pre-programming, motor system activation, and even motor learning.

To ensure that SAS is used correctly as an investigative probe tool, it is important to have consistency in the methodology and application as well as interpretation of results. Since the use of a startle in many experimental situations is novel (as is the technique to many behavioural neuroscientists), many questions have been raised and have yet to be addressed with respect to the optimal experimental parameters and manipulations when using the startling acoustic stimulus (SAS). Additionally, interactions between instructions to participants, timing and amplitude of stimuli, and types of responses elicited can have an effect on the results. These considerations will be discussed in the following sections.

## 2. The Startle Response

When using the startle as a research tool, it is important to determine whether or not the acoustic stimulus actually elicited a startle response in the participants. In the case that it does not, the experiment is not testing the effect of a startle, rather it is simply testing the effect of a (more) intense stimulus. The startle response (or startle reflex) in and of itself has long been the subject of scientific investigation (e.g., Landis et al., 1939), with many results elucidating the nature of the startle response both in humans (Brown et al., 1991b; Jones and Kennedy, 1951) and animals (e.g., Davis, 1974; Davis, 1984; Shnerson and Willott, 1980). However, before using the acoustic startle as a research tool, it is important to first understand what a startle is, how it is produced and recorded, what the effects are on humans, and what factors may affect the startle response (e.g. either increasing or decreasing the startle response latency and / or amplitude). The following is a discussion of these considerations.

The startle response is a diffuse, whole-body physiological response consisting of a characteristic set of muscle actions initiated by a sudden and unpredictable intense acoustic, visual, mechanical, electric, or vestibular stimulus (Blumenthal et al., 2005; Davis, 1984; Scott et al., 1999; Shnerson and Willott, 1980; Yeomans and Frankland, 1996). Enoka (2008) refers to the startle reaction as "the most extreme example of a distributed set of automatic responses to an [environmental] disturbance" (p.275). This response involves a pattern of muscle flexion (although extension has also been observed, see Brown et al., 1991b), as well as an increase in central nervous system and autonomic activity (Thackray et al., 1972). While several stimulus perturbations can be used to elicit a startle (Blumenthal et al., 2005; Yeomans and Frankland, 1996), the most commonly employed stimulus modality has been the loud acoustic stimulus, due to the ease and reliability with which it can be implemented.

Landis et al. (1939) described the startle response as a patterned response consisting of several bilateral stereotyped muscle movements. This response started with blinking of the eyes and a characteristic facial expression, along with dorsiflexion of the head and neck. In addition,

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the response included a curling of the shoulders in a ventro-caudal direction, flexion of the elbows and fingers, bending of the trunk, and bending of the knees. This "generalized flexion" response has been hypothesized to be an adaptive defence response in terrestrial mammals to a predatory attack from the rear, as it may result in reduced exposure of the dorsal surface of the neck, a vulnerable point of attack (Yeomans and Frankland, 1996). Landis et al. used high-speed photography, which they described as "tedious, demanding, and expensive" (1939, p. 156), to visually capture and later describe the patterned response in humans. Because of this, other measures have been employed which are more economical and easily analyzed. For example, increases in heart rate as well as significant increases in skin conductance (O'Gorman and Jamieson, 1977; Ornitz et al., 1996; Shalev et al., 1997; Thackray and Touchstone, 1970) have been reported in response to an intense auditory stimulus. Thackray (1972), however, cautioned that stimuli low enough in intensity (as low as 40 dB) to only evoke "orienting" responses and not startle responses, were also accompanied by increases in galvanic skin response, and that these differences in skin conductance between stimulus intensities were not abrupt enough to allow for discrimination between the responses. While measures such as heart rate and galvanic skin response can be used to assess startle, the startle response has been primarily measured as an electromyographic (EMG) response since the work of Jones and Kennedy (1951). This is due to the short latency of the EMG bursts observed in response to a startling stimulus, the reliability of the response (Brown et al., 1991b), and the practicality of the method (Jones and Kennedy, 1951).

In response to a 124dB acoustic stimulus, Brown et al. (1991b) described EMG patterns from eye closure, facial grimacing, neck flexion, trunk flexion, abduction of the arms, flexion of the elbows, and pronation of the forearms. They reported a large range in the latencies of onset EMG activity in the various muscle groups (25 – 199 ms), however, median EMG onset times were all found at short latencies ranging from 36.7 ms in orbicularis oculi (OOc) to 98.8 ms in first dorsal interosseous (FDI). In addition, while activity in OOc was always seen in response to the acoustic stimulus, other components of the startle response were less reliable. Startle elicited EMG latencies that increased with increasing segmental distance from the brainstem, with facial muscles being the first to be activated, followed in order by neck and paraspinal muscles, upper arm, lower arm, trunk, and finally leg muscles (Brown et al., 1991b). Interestingly, activation in the intrinsic hand muscles (e.g. FDI) was disproportionately long (median onset 98.8 ms), activating well after the forearm muscles and even the abdominals (82.3 ms). Importantly, Brown et al. (1991b) noted that activity in sternocleidomastoid (SCM) was the first recordable EMG response to a startle after the eyeblink. This SCM activity was found to be the most consistent EMG response after the eyeblink, and the last to disappear due to repeated startle stimulation (described as habituation). Finally, the authors noted that due to the activation pattern of cranial nerve innervated muscles, the pattern of activation was in a caudal to rostral direction, starting from approximately the eleventh cranial nerve (Brown et al., 1991b).

### *2.1. Startle response pathways*

Since the startle response onset latency is very short (e.g. median onset for many muscles is <75 ms, see above), Yeomans and Frankland (1996) suggested that the primary startle response circuit cannot include many synapses, and emphasized the role of the pontine reticular formation, specifically, the nucleus reticularis pontis caudalis (nRPC). Although many studies have reported that the nRPC is of central importance in the startle circuit (Davis, 1984), Yeomans and Frankland (1996) specifically implicated the giant neurons of the nRPC. As such, the grading seen in the amplitude of the startle response with the intensity of the stimulus (described below) was suggested to be the result of the number of nRPC giant neurons recruited. As the stimulus intensity increases, the number of nRPC neurons activated increases, leading to a larger startle response. In this way, the nRPC giant neurons may act as "command" neurons of the acoustic startle response. The nRPC neurons conduct to the various levels of the spinal cord, along the reticulo-spinal tract, and activate motorneurons with both weak monosynaptic connections, and strong disynaptic connections involving interneurons (for more detail see Yeomans and Frankland, 1996). This motor activation then produces the measurable EMG response and movement associated with the startle.

### *2.2. Startle vs. acoustic blink reflex*

As previously noted, an eyeblink (characterized by EMG activity in OOc) has been observed as a response to loud auditory stimuli in many experiments (Blumenthal, 1996; Brown et al., 1991b; Carlsen et al., 2003a, 2003b, 2007; Miwa et al., 1998; Säring and von Cramon, 1981; Valls-Solé et al., 1995, 1999). As such, due to its short latency and reliable elicitation (Blumenthal, 1996; Blumenthal et al., 2005; Brown et al., 1991b), OOc EMG burst activity has been the most widely used indicator of a startle response. This widespread usage of the startle-elicited eyeblink led the editors of Psychophysiology to appoint a committee to publish a report outlining guidelines for using the startle eyeblink in human research (see Blumenthal et al., 2005). However, evidence published by Brown et al. (1991b), suggests that the eyeblink may not always be a valid indicator of a startle response in and of itself (see also Rothwell, 2006). Two lines of evidence were given to support this position. First, the auditory blink response does not habituate in the same manner as the rest of the startle response: Although other components of the startle response in the participants were no longer seen in response to the loud startling stimulus, the eyeblink was seen even after presenting the acoustic stimulus at regular

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intervals (every 1 min) for 20 minutes. This indicated that although the participant had habituated to the stimulus and was no longer being startled, the OOc was still activated by a separate auditory blink reflex (Brown et al., 1991b; Kofler et al., 2001b). The second line of evidence concerns the configuration of the EMG activity from the OOc following habituation. When a startle response was elicited (prior to habituation), the EMG activity in the OOc was much longer in duration compared to following habituation. It was suggested that the response seen during a true startle was simply an early auditory blink response with a separate OOc startle response grafted onto the end, and that the two responses arise from two physiologically separate circuits (Brown et al., 1991b; Meincke et al., 2002). The auditory blink reflex was reported by Brown et al. (1991b) to occur at a short latency (36.7 ms) and to be of a relatively brief duration (a range of OOc EMG response durations from 63.3 to 149.2 ms). Säring and von Cramon (1981) also reported short duration blink responses, with mean blink EMG response duration of 114 +/- 18 ms. This is in contrast to the much longer (110 – 400 ms) duration EMG responses found by Brown et al. (1991b) when other startle response indicators were present. In addition, Brown et al. (1991b) suggested that in 36% of startle trials, two distinct components were visible.

Similar findings were evident in research from our own laboratory (Carlsen et al., 2007), where the configuration of the OOc EMG was substantially different depending on whether or not startle-related SCM activity was present. Specifically, our data indicated that when SCM EMG activity was observed in response to a loud acoustic stimulus, a two-component (early + later) EMG pattern was seen in OOc. However, when no SCM activity was observed only the early single EMG component was present (Fig. 2). It appeared that OOc activity alone was qualitatively different than OOc activity when SCM activity was also present. Further analysis of integrated EMG analysis showed that there was significantly more OOc EMG activity when SCM was also detected. Together, these data suggested that the OOc response was both qualitatively and quantitatively different depending on the presence of SCM activity, and that OOc activity detected in the absence of SCM activity was different and not necessarily indicative of a startle response (Carlsen et al., 2007).

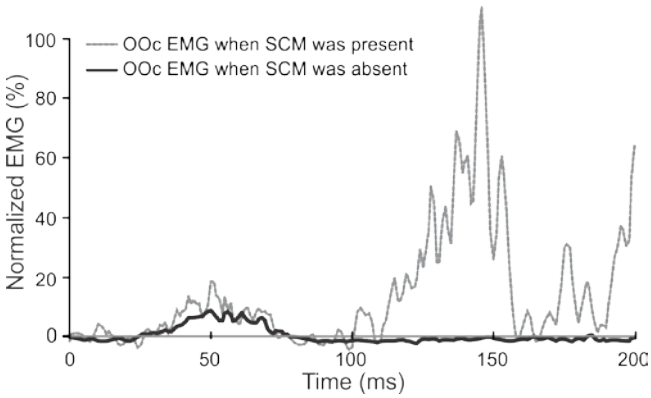


***Figure 2. Ensemble averages of rectified EMG from orbicularis oculi (OOc) from a single subject expressed by whether sternocleidomastoid (SCM) activity was either detected (grey) or not (black). Adapted from: Carlsen, A.N., Dakin, C.J., Chua, R., Franks, I.M., 2007. Startle produces early response latencies that are distinct from stimulus intensity effects. Exp. Brain Res. 176, 199-205. Copyright ©2007 Elsevier Limited..***

If these two blink components are physiologically separate, the neural pathways should also be different. The auditory blink reflex pathway is similar in many respects to the general auditory pathway (Hudspeth, 2000). However, in the midbrain, the auditory blink reflex pathway deviates from that of the normal auditory pathway. Lesioning studies have shown that axons mediating the blink reflex project from the inferior colliculus to the midbrain reticular formation (Hori et al., 1986). Axons from the midbrain reticular formation synapse at the facial nucleus (Hinrichsen and Watson, 1983; Hori et al., 1986), and then continue on through the facial nerve (VII) where they innervate OOc (Brown et al., 1991b). As mentioned, this is a somewhat different pathway than the normal auditory pathway. In addition, this pathway is different than the acoustic startle response pathway (Yeomans and Frankland, 1996), giving strength to the assertion by Brown et al. (1991b) that the auditory-blink and startle-blink responses are physiologically separate. As mentioned, several studies have reported that the pontine reticular formation is of central importance in the startle circuit (Davis, 1984; Yeomans and Frankland, 1996). This is in contrast to the midbrain reticular formation pathway described in the acoustic blink reflex. A potential explanation of the chain of events initiated when the startle occurs is outlined in the following segment:

The acoustic startle stimulus is presented and transduced by the receptors of the cochlear nerve. The acoustic signal is passed along three or more pathways. One pathway passes through the midbrain reticular formation which leads to a short latency blink reflex. Another pathway connects with the pontine reticular formation, which (if the signal is strong enough) activates the giant neurons of the nucleus reticularis pontis caudalis (nRPC). This leads to a generalized startle response consisting of activation of motor pathways at increasingly longer latencies as

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segmental distance from the lower brainstem increases (Brown et al., 1991b). Additionally, the cranial nerves are activated in a caudal to rostral direction of propagation leading to activation of the SCM (innervated by cranial nerve XI), followed by activation of OOc (innervated by cranial nerve VII), and masseter (innervated by cranial nerve V) (see also Valls-Solé et al., 2008 for a review of these pathways, specifically Figure 1 p.498). A third pathway follows the normal auditory pathway to the primary auditory cortex. In this way, it is possible that the blink response is activated twice in short succession, first by the midbrain reticular formation, and secondly by the pontine reticular formation, prior to conscious awareness of the stimulus.

In light of this evidence, one must question the utility of using the eyeblink as the sole startle response indicator, yet many experiments have indeed used only the eyeblink response as an indication that a startle response had occurred. Instead, the above evidence appears to suggest that the best measure of whether a startle has been elicited may be derived from a short latency burst of EMG activity in the SCM, and secondarily from the C4 paraspinals (C4P), as these show the shortest latency and the highest reliability (Brown et al., 1991b). Similar conclusions were drawn from studies of patients with movement disorders such as Parkinson's disease (Kofler et al., 2003; Williams et al., 2008), anxiety disorder (Bakker et al., 2009), and dystonia (Muller et al., 2003). For example, it was shown that in children with hyperactive startle responses, the eyeblink alone may not be indicative of a full acoustic startle response and that a whole body measure may be more appropriate (Bakker et al., 2009). It should be noted, however, that the EMG response from the OOc might be an acceptable indicator of startle, as long as the distinction is made (through measurement of EMG burst duration) between a simple auditory blink response and a longer duration EMG response due to a startle (Brown et al., 1991b; Carlsen et al., 2007).

One potential confound with the use of SCM as a startle indicator is that often head movements are a part of the required voluntary response (Oude Nijhuis et al., 2007; Siegmund et al., 2001). Thus researchers need to be able to distinguish between SCM activation that occurs as a result of the startle response versus activation due to movement of the head itself. One solution is to investigate differences in bilateral SCM activation. Although the startle response is generally considered to be bilaterally synchronous (Brown et al., 1991b) it has been shown that lateralized differences occur in the amplitude of the response. For example, differences in OOc activation have been shown that were attributed to differences in hemispheric gating (Cadenhead et al., 2000). Similarly, lateralized differences in the startle response amplitude were shown in SCM and biceps that were consistent with hand dominance (Kofler et al., 2008). However, in order to separate out startle-related and voluntary responses, EMG from the right and left SCM can be compared through a coherence analysis which can be used to look for lateralized differences in the frequency components of EMG signals (Farmer et al., 1997). As the startle reflex is a bilateral response, the left and right SCM would normally be expected to show a high coherence as was shown by Grosse and Brown (2003) for proximal upper limb muscles. Alternately, when the required voluntary response involves a head movement to one side (Oude Nijhuis et al., 2007; Siegmund et al., 2001), the startle evoked SCM activity should show low bilateral coherence.

### 3. Detecting a Startle Response: EMG

Once it is determined what muscle response indication will be used to verify whether a true startle response has occurred, it is important that reliable and valid methods be used to analyze EMG data. Thus an appropriate determination can be made on a trial-by-trial basis to either accept the trial as a "startle trial" or reject it. An excellent description of EMG collection procedures including participant preparation, electrode attachment, signal amplification and filtering, and analog to digital conversion, is provided by Blumenthal et al. (2005). Although these procedures are intended for analysis of the OOc eyeblink response, most of the recommendations can be generalized to the SCM response as well. Of particular relevance to the present discussion are scoring parameters, and the quantification of the response. As suggested by Blumenthal et al. (2005), EMG onset detection can be performed either manually, or by using a computer algorithm that can detect onsets based on an objective criterion. For example, one method involves displaying the EMG pattern for each muscle on a computer monitor with a superimposed marker indicating the point at which activity increased to more than two standard deviations above a baseline level (e.g., a mean of 100 ms of EMG activity preceding the "go" signal). Onset is then verified by visually locating and manually adjusting the onset mark to the point at which the activity first increases (see Carlsen et al., 2004b). An allowable time window (e.g., from 30ms to 120ms) following the SAS for SCM onset, can be used to distinguish startle SCM activity from other SCM activity (e.g. postural; Carlsen et al., 2004b). In this way, a decision can be made as to whether or not a SCM startle response was elicited by the loud stimulus on a trial to trial basis. This scoring is important since it has been shown that RT for the intended movement differs based on whether or not a startle response was elicited (Carlsen et al., 2007). Specifically, how the presence of a SCM startle response affected RT was the focus of a study involving different stimulus intensities. It was shown that for any stimulus intensity over 90dB RT was shortened to ~80 ms if a SCM burst was observed. Thus, while only a small number of trials with stimulus intensities of 93 dB or 103 dB resulted in an observed SCM EMG response, these trials all had substantially shortened RTs, similar to those observed at much higher intensities (e.g. 123 dB). As such, it was suggested that only if startle related SCM activity was observed (irrespective of stimulus intensity) there was

sufficient activation to trigger the pre-programmed response, leading to early triggered RTs. These results indicate that trials in which no SCM response was observed should be treated separately or removed from the analysis of "startle" trials in which a SCM startle response was elicited (Carlsen et al., 2003a, 2007). This separation of trials may be beneficial when attempting to distinguish the simple effect of stimulus intensity (where higher stimulus intensities lead to shorter RTs, presumably as a result of faster perceptual processing, Kohfeld, 1971; Luce, 1986; Woodworth, 1938), from the involuntary release of a prepared motor program by the startle (Carlsen et al., 2007, 2009a). In order to increase the probability of eliciting a startle response within the context of a RT task, stimulus parameters and testing environment must be taken into account.

## 4. Acoustic Stimulus Parameters: Effect on the Startle Response.

Although various stimulus modalities can be used to elicit a startle (see above), here we will discuss the parameters surrounding a startling *acoustic* stimulus (SAS), as it is the most widely employed method. Landis et al. (1939) favoured this method as it capitalized on two of the requirements for eliciting a startle: intensity and "surprise." A comprehensive review of many of the stimulus properties that affect the acoustic (as well as visual, electrical and mechanical) startle response has been provided by Blumenthal et al. (2005) and will not be repeated here. However, we will summarize, and expand upon, some of the key stimulus features that lead to a startle response with the largest amplitude and shortest latency. These include SAS frequency, rise time, duration, and of course, intensity. Note that unless otherwise stated, all intensities reported involve the A-weighted decibel (dB) scale, dB(A) measured with a dB meter (e.g. Cirrus Research - model CR:252B) at the distance that the ears will be from the noise source during the experiment.

Since a startle can be elicited at all frequencies in the audible range (Pilz et al., 1987), it has been argued that a broadband noise pulse (white noise) may be a more effective startling stimulus than a single tone (Blumenthal and Berg, 1986). This may due to broadband noise activating a larger portion of the basilar membrane resulting in the generation of increased activation in the acoustic nerve and increased input to the auditory pathway. However, many recent experiments looking at the interaction between motor acts and the startle response have elicited a startle response successfully in a majority of trials regardless of whether the SAS consisted of a single frequency (e.g., 750 Hz, 1kHz, 1.5kHz: Carlsen et al., 2003a, 2004a, 2008b, 2009a, 2009b; MacKinnon et al., 2007; Maslovat et al., 2008, 2009a; Reynolds and Day, 2007; Siegmund et al., 2001; Walsh and Haggard, 2008) or multiple frequencies (e.g., Castellote et al., 2007; Kumru et al., 2006; Oude Nijhuis et al., 2007; Valls-Solé et al., 1995, 1999, 2005). Thus, while Blumenthal et al. (2005) argued that all other things being equal, white noise results in shorter latency and larger amplitude startle responses, stimulus frequency may not be of paramount importance. Nevertheless, to maintain the novelty of the stimulus and perhaps decrease the rate of habituation, several researchers have varied both the frequency and intensity of the stimulus from trial to trial (Kofler et al., 2001a, 2003, 2006; Muller et al., 2003; see also Valls-Solé et al., 2008).

SAS rise time (the time it takes for the stimulus to reach maximum intensity), however, is a critical startle stimulus feature for eliciting a startle response. The rise time of the stimulus intensity that is produced by the headphones or speaker (e.g., MG Electronics – model M58-H) should be less than 12ms in order to elicit a startle response. If longer rise times are used, even extremely intense sound levels (140 dB) fail to produce a startle response (Davis, 1984), as the startle appears to reflect a response to a sudden change in the stimulus environment (Blumenthal and Berg, 1986; Blumenthal et al., 2005). In addition to rise time, SAS duration can also have an effect on startle responses. It has been shown that stimulus durations of up to 50 ms are associated with larger startle responses (Blumenthal et al., 2005), with little benefit from longer duration stimuli. Akin to this, summation of two shorter acoustic stimuli also results in an increase in the magnitude of startle, which has been attributed to temporal summation (Marsh et al., 1973). More recent evidence suggests that acoustic stimulus summation effects are most pronounced at inter-stimulus intervals (ISIs) of 4 - 6 ms (Li and Yeomans, 1999). Using short burst duration stimuli (e.g., 40 ms, see Carlsen et al., 2004b) poses some challenges for measuring and verifying the stimulus intensity. The use of a sound level meter capable of measuring *impulse* dB level is important (e.g. Cirrus Research - model CR:252B).

While acoustic stimulus parameters have an influence on both the probability of eliciting a startle response as well as its magnitude, stimulus intensity is one of the most important factors in achieving a robust and valid startle response (Carlsen et al., 2007; Landis et al., 1939). That said, acoustic stimulus intensity is also one of the most variable parameters between experiments in the literature. For example, it has been reported that the startle blink response can be evoked with acoustic stimuli as low as 70dB (Blumenthal et al., 2005), while intensities of up to 150dB have also been used (Valls-Solé et al., 1995). However, while low intensity stimuli may sometimes produce a measurable "blink," more intense stimuli produce larger startle responses with shorter response latencies (Blumenthal, 1996; Davis, 1984). As such, many experiments have utilized acoustic stimuli with intensities ranging from 103 dB (Walsh and Haggard, 2008) to 130dB (Valls-Solé et al., 1999), with many studies using stimuli in the 113dB – 124dB range (Abel et al., 1998; Brown et al., 1991b; Carlsen et al., 2004a, 2004b, 2009a, 2009b; Cressman et al., 2006; MacKinnon et al., 2007; Maslovat et al., 2008, 2009a; Oude Nijhuis et al., 2007; Reynolds and

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Day, 2007; Siegmund et al., 2001). A recent study examining the interaction between acoustic stimulus intensity, startle, and reaction time (RT) in a simple RT task showed that the probability of observing a startle response increased with increasing stimulus intensity (Carlsen et al., 2007). Of course, this in itself is not entirely surprising; rather, how the probabilities of observing an OOc response alone vs. observing SCM along with OOc changed with increased intensity is more interesting (see Fig. 3). In particular, it was shown that the probability of observing a majority of SCM "startle" responses only occurred when stimulus intensity was 123 dB (Carlsen et al., 2007). Note that it was also determined above that in many cases SCM may be a better indicator of a startle response that is sufficient to trigger a pre-programmed action than an OOc "blink." In contrast, even with a stimulus intensity of 103 dB, "no response" (neither SCM activity nor blink) was observed more than 50% of the time (Fig. 3). Since the probability of observing a SCM response increased with intensity, it was suggested that for RT experiments involving the use of a startle, the highest possible SAS intensity should be used short of risking auditory damage to participants (Carlsen et al., 2007). However, prolonged exposure to sound levels above 120 dB is not recommended, and even short bouts of these stimulus intensities may be unpleasant for participants. While standards will vary both between and within countries, the standard outlined by the National Institute for Occupational Safety and Health (NIOSH) in the United States of America prohibits any exposure to noise levels above 140 dB (A-weighted scale). Additionally, exposure over 130 dB is limited to less than 1 sec. total noise dose (where the remainder of an eight hour workday averages less than 85 dB; see NIOSH, 1998). As such, stimulus intensities below this level are advised both for participant comfort and to minimize risk, in addition to strict adherence to local standards.

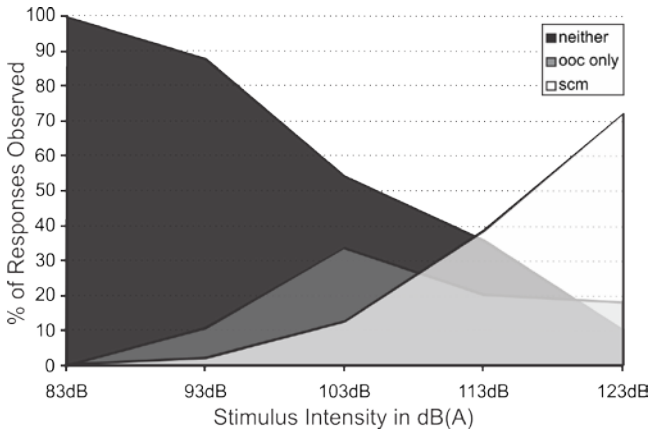


***Figure 3. Percentage of trials in which the startle indicators were observed for each stimulus intensity. Black is the percentage of trials in which neither sternocleidomastoid (SCM) nor orbicularis oculi (OOc) activity was observed. Grey represents the percentage of trials in which OOc activity was observed but not SCM. White represents the percentage of trials in which SCM activity was observed. Adapted from: Carlsen, A.N., Dakin, C.J., Chua, R., Franks, I.M., 2007. Startle produces early response latencies that are distinct from stimulus intensity effects. Exp. Brain Res. 176, 199-205. Copyright ©2007 Elsevier Limited.***

## 5. Other Factors Affecting the Startle Response

Having outlined the previous research involving the optimal stimulus parameters for the acoustic startle stimulus, and how to best measure whether a startle response has been elicited, there remain several factors (such as external environment and prior experience), which may affect the startle response. These may also lead to differences in startle response latency, amplitude, or both. Some of these factors will be discussed below.

### *5.1. Habituation and motor preparation*

Response habituation, which involves a decrease in behavioural response to a repeated stimulus (Kandel et al., 2000), has been observed in most studies involving the use of a SAS (Abel et al., 1998; Davis and Heninger, 1972; Davis, 1984; Leaton et al., 1985; Schicatano and Blumenthal, 1998; Valls-Solé et al., 1997). While a decrease in EMG response amplitude is observed with habituation, response latency is unaffected (Schicatano and Blumenthal, 1998). Evidence has been shown in humans that most components of the startle response are no longer seen after only 2 to 6 random presentations of a startling stimulus, eventually declining to the point where no overt response to a startling stimulus is seen apart from the ever-present "blink" reflex (Brown et al., 1991b). The pattern of habituation is not random, however, nor is it all-or-none, as the response tends to decline in amplitude with repeated exposure (Abel et al., 1998) and disappear in peripheral regions first (Davis and Heninger, 1972). Response reductions observed during habituation are thought to be caused by depressed synaptic transmission in the involved neural circuits (Kandel et al., 2000), although the exact neural mechanism of startle habituation is not well understood (Jordan and Strasser, 2000). However, since startle habituation has been observed in decerebrate rats (Leaton et al., 1985), habituation is thought to be a process that occurs in the brainstem, specifically due to synaptic depression of the reticular formation at the level of the pons (Chokroverty et al., 1992).

There are some measures that may be undertaken to counter the habituation normally observed. For example, Brown et al. (1991b) suggested that habituation may be decreased by increasing the time between presentations of the SAS. More importantly in the context of the current discussion however, Valls-Solé et al. (1997) reported a large reduction of startle habituation in participants who were preparing to react to a "go" signal. Participants were exposed to 5 startling stimuli in each of 4 different conditions. Participants were resting quietly, or resting in a busy environment, or preparing to react in a reaction time

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task, or focusing on an upcoming visual stimulus. The rate of habituation was only significantly decreased when participants were preparing to react to a visual "go" signal in a RT task. In this condition, peak EMG amplitude in the SCM did not decrease below 60% of initial amplitude, whereas in all other conditions, EMG amplitude fell below 20% of initial values by the fifth presentation of the stimulus. The authors suggested that reduced cortical inhibition of the startle response as well as increased excitability of the motor pathway due to readiness to perform a motor act might have been the reason for the decreased habituation (Valls-Solé et al., 1997). Similar results to these were presented in a study by Siegmund et al. (2001), where little habituation of the startle response was observed over 14 trials in which a startling stimulus was presented in a RT task involving head flexion and rotation. As suggested previously, readiness to perform a motor act was argued to be the most likely candidate for the decrease in habituation (Siegmund et al., 2001) but since the required movement in the Siegmund et al. study involved the activation of the muscles used for startle response detection (SCM), this specific muscle activation may have contributed to the pattern observed.

In order to resolve the question of how habituation progressed when participants were involved in a RT task, Carlsen et al. (2003a) performed an experiment where a startling stimulus (124 dB) was presented in 20 out of 100 RT trials. No significant reduction in SCM EMG amplitude was observed from the first to 20$^{th}$ startle trial across participants. However, in several trials for each participant, no SCM activity was seen (see Fig. 4 top panel), although it returned in later trials. Additionally, the incidence of startle trials in which SCM activity was observed was no higher in the first ten trials than in the last ten trials across participants (Fig. 4, bottom panel). It was suggested that in contrast to a decrease in habituation processes, the increased neural activation associated with readiness to perform a motor act (Brunia, 1993; Coxon et al., 2006; MacKinnon and Rothwell, 2000) led to an overriding of habituation, or "dishabituation" of the startle circuits (Carlsen et al., 2003a; see also Kandel et al., 2000). Thus, when used in the context of a motor task requiring advance preparation, it appears that it may be possible to elicit a startle response indefinitely (Carlsen et al., 2003a).

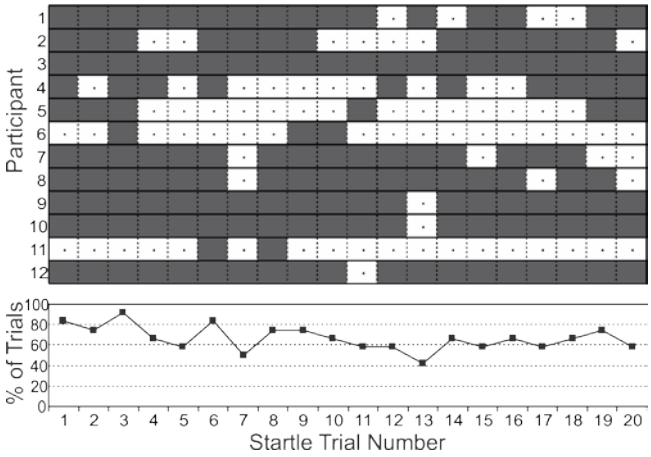


***Figure 4. Individual participant (top) and group (bottom) data indicating startle trials in which a sternocleidomastoid (SCM) burst was observed. In the top panel, each box represents a startle trial (in order of presentation) and whether SCM activity was observed (grey) or not (white). The bottom panel represents the percentage of trials across subjects for each startle where SCM was observed. Adapted from: Carlsen, A.N., Chua, R., Inglis, J.T., Sanderson, D.J., Franks I.M., 2003a. Startle response is dishabituated during a reaction time task. Exp. Brain Res. 152, 510-518. Copyright ©2003 Elsevier Limited.***

Interestingly, it seems that even though readiness to perform a motor act delays the rate of habituation, an ongoing motor act does not. In investigating the effects of a startling stimulus on rifle timing, Foss (1989) found that the disrupting effects in aiming proficiency caused by the startling stimulus decreased rapidly with repeated exposure to the stimulus. Recovery of the effect (a decrease in the amount of habituation) was seen after a 24-hour rest period. It should be noted that although various ratios of control to startle trials have been used (e.g., 4:1 by Carlsen et al., 2003a; 2:1 by Oude Nijhuis et al., 2007), no systematic test of this parameter has been carried out and it remains unclear how this may affect habituation.

### *5.2. Prepulse inhibition*

While it has been shown that temporal summation of two shortly spaced startling stimuli (e.g. ISI of 5 – 10ms) can lead to increased startle response amplitudes and reduced startle latencies (Graham, 1975; Li and Yeomans, 1999), lower intensity acoustic stimulus prepulses (not able to produce a startle response on their own), at longer intervals prior to the startling stimulus (20 - 500 ms) have been shown to decrease startle amplitude (Graham, 1975; Hoffman and Searle, 1965). However, this effect decays as the prepulse-SAS interval lengthens (Graham, 1975). This type of reflex modification has become known as prepulse inhibition (PPI) of startle (Davis, 1984; Fendt et al., 2001; Hoffman and Searle, 1965; Hoffman, 1984; Ison and Hammond, 1971; Lehmann et al., 1999; Swerdlow et al., 2001; Valls-Solé et al., 2008). This PPI has been suggested to reflect the ability of higher brain centres to gate or filter incoming sensory information (Abel et al., 1998;

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Blumenthal, 1996; Swerdlow et al., 2001; Zhang et al., 1999). PPI effects can occur even when the prepulse and startling stimulus modalities are different (e.g., tactile or visual prepulse in conjunction with an auditory startle, Lipp et al., 2000; Zhang et al., 1999), although effects of PPI are most pronounced if the prepulse and startle stimulus are in the same modality (Balaban et al., 1985). Interestingly, while pulsed background noise can reduce the startle response, it was shown that increased constant background noise (85 dB) led to a doubling of the startle response possibly due to decreased cortical gating (Hoffman and Searle, 1965).

A large body of literature is associated with the study of PPI, its neural connections and mechanisms, and how it relates to higher brain function as well as brain disorders (see Fendt et al., 2001; Swerdlow et al., 2001). Furthermore, a review of how PPI may interact with startle in RT tasks has been provided by Valls-Solé et al. (2008). As such a full review of these issues is beyond the scope of this article. More importantly with respect to the current paper is the implication that in order to increase the likelihood of eliciting a startle response (as well as achieving the largest amplitude response), the sensory environment should be kept sufficiently constant so that PPI does not take place. Furthermore, care should be taken in the design of the task so that any warning / ready stimulus is given with sufficient lead-time prior to the startle to avoid PPI. On the other hand, it has been shown that if PPI was induced during a startled RT task, the startle response (EMG in OOc and SCM) was significantly attenuated while the RT speeding effect of the startle was unaffected (Valls-Solé et al., 2005). This, they suggested, showed that the early release of a prepared movement by startle and the startle response itself were separable, thereby implicating different physiological mechanisms. In the work by Valls-Solé et al. (2005), the prepulse was presented via a weak electrical shock to the finger 100ms prior to an auditory “go” signal. A similar recent study investigated how the modality and timing of the prepulse affected both the startle response and early release of a prepared movement (Maslovat et al., 2009b). An auditory prepulse of 84dB was given at various time intervals (100ms, 500ms or 1000ms) prior to the auditory “go” signal. Similar to the findings of Valls-Solé et al. (2005), an auditory prepulse presented 100ms prior to the imperative stimulus resulted in decreased SCM activation (i.e. attenuation of the startle response) but did not affect the reaction time for the prepared response (i.e. early release still occurred). However, prepulses delivered at 500ms or 1000ms prior to the “go” did not result in a significant decrease in the SCM activation. Thus it appears the timing of the prepulse is more critical than modality in its modulation of the startle response. Nevertheless, irrespective of its interactions with RT, PPI can result in a decrease in startle response and if the detection of a measurable startle response is an important factor, PPI should be considered when developing an experimental protocol involving startle.

### *5.3. Other considerations*

Although we have already reviewed many of the elements that influence both the probability of eliciting a startle response and its amplitude, there are several other factors that may interact in a complex way to modulate the startle response. For example, there is some evidence to suggest that in females and in older individuals, there may be a small but significantly larger probability of observing a startle response, coupled with larger response amplitudes (Kofler et al., 2001a, 2001b). Additionally it has been suggested that PPI is greater in females than in males (Abel et al., 1998; Lehmann et al., 1999). Other evidence suggests that posture and the level of background muscle contraction may modulate the size of the measured startle response: For example, it was shown that when standing, the probability of observing a startle response in the lower leg muscles was much greater than when sitting (Brown et al., 1991a).

While it has been shown that anxiety can affect the startle response (Bakker et al., 2009; Coombes et al., 2007), general arousal levels can also change the measured startle response (Andrews et al., 1998; Schicatano and Blumenthal, 1998). How the startle response is modulated by directed attention, however, may be of more importance to the purposes of the current review since in traditional RT tasks participants typically attend to some type of “go” signal. This “go” signal may vary in timing as discussed above, but may also vary in modality. While presenting a “go” signal in multiple modalities has been shown to reduce reaction times (Nickerson, 1973), it has been suggested that intersensory facilitation has no effect on the RT speeding effect of startle (Valls-Solé et al., 1999, 2008). However, it may be of interest when using the startle to know how directed attention may affect the startle response depending on the modality of the “go” signal. Several experiments have investigated this interaction directly (although not in the context of a RT task), and attention seems to affect the startle response in an interesting way. If attention is directed at a stimulus in the same modality as the startling stimulus, the startle response is enhanced (e.g., larger amplitude and shorter latency). However, attention directed toward a different sensory modality than the startle stimulus, may result in a decreased startle response (Anthony and Graham, 1985; Schicatano and Blumenthal, 1998) or may have no effect on the startle response, neither enhancing nor reducing it (Richards, 2000). Specifically, in one study investigating the effect of the attentional process on the startle response (Schicatano and Blumenthal, 1998), subjects were instructed to attend to the startle stimulus, a visual task, or not given instruction to attend to anything in particular. Results showed that startle latency, detected by an eyeblink response, was significantly longer, and that startle amplitude was significantly smaller when attending to the visual task. This, the authors suggest, provides evidence that attentional modulation of the startle response

is sensitive to the attended sensory modality. This is due to the suggestion that the afferent sensory pathways are enhanced with increased attention to a particular sensory modality leading to an increased startle response from a startle stimulus in the same modality (Richards, 2000). Thus, when using startle in a RT task, the modality of the "go" signal may play an important role. It should be noted that the above effects on the startle response are relatively small compared to the effects resulting from changes to the stimulus parameters discussed above. Furthermore, this review is more concerned with using a startle to elicit a prepared movement, rather than the actual startle itself. While the effects of these factors individually may be small, it is unclear how they may interact to increase or decrease the probability of eliciting a startle that is sufficient to trigger a prepared response. However, in the extreme case where all the possible effects that may lead to a smaller startle response coincide (younger male, low arousal level, no anxiety, attention directed at another stimulus modality), these factors may play a decisive role in the ability to elicit a startle response, and thus a pre-programmed action. Currently, however, this possibility remains untested.

## Summary and Conclusions

Recently, there has been renewed interest in the use of a startle response in humans with respect to voluntary movement. Rather than simply investigating its effect on ongoing motor control, however, a SAS has been used to probe motor preparation prior to movement onset. The aim of this methodological review paper was to survey some of the recent literature involving the use of a SAS to investigate motor processes in humans, and to re-evaluate some of the methodological considerations with respect to the use of a SAS. It was shown that the movement task employed and the timing of the SAS probe influence the results, and thereby can provide insight into processes of early motor preparation (section 1: Early release of movement by startle). However, in order to infer that any changes in behavioural response are due to startling the subject, a startle response must be measured independently of the focal response and best practices for detecting and analyzing a startle response (section 3: Detecting a startle response: EMG) was presented. In particular, it was suggested that the best muscle for detection of startle-related EMG activity is SCM (e.g., Blumenthal et al., 2005; Brown et al., 1991b; Carlsen et al., 2003a, 2007) and some considerations with respect to EMG collection and analysis were presented. In order to obtain a robust startle response in a majority of trials it was suggested that a narrow or broadband frequency SAS of 50 ms duration and 124 dB(A) intensity be used, presented in up to 25% of trials (section 4: Acoustic stimulus parameters). Finally some of the external factors that may affect the startle response were discussed, including startle habituation, prepulse inhibition (PPI), and directed attention, although some of these factors appear to have comparatively little effect on the startle response in isolation (section 5: Other factors affecting the startle response).

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