Warning signals induce automatic EMG activations and proactive volitional inhibition: evidence from analysis of error distribution in simple RT

Philippe Boulinguez¹*
Magali Jaffard²
Lionel Granjon³
Abdelrhani Benraiss¹

¹ Université de Poitiers, CeRCA, équipe Attention & Contrôle, CNRS, France
² Université de Poitiers, LPMC, EA 3814, France
³ Université de Poitiers, LMDC, UMR CNRS 6215, France

* corresponding author:
CeRCA, équipe Attention & Contrôle, CNRS
MSHS, 99 avenue du Recteur Pineau, 86000 Poitiers, France
+33 (0)5 49 45 46 97
pbouling@univ-poitiers.fr

Running head: Activation/inhibition in simple warned RT
Summary

Typical simple reaction time (RT) paradigms usually include a warning signal followed by a variable foreperiod before the presentation of a reaction stimulus. Most current interpretations suggest that the warning stimulus alerts the organism and so results in faster processing of either the sensory or motor components of the task. In this study, EMG was used to detect both covert and overt motor errors in a simple warned RT task. Results show that warning signals may trigger automatic motor activations which are likely to cause false alarms. Distribution analysis reveals that 77% of all errors detected with EMG are erroneous responses to the warning signal. Accordingly, we propose that movement triggering needs to be temporarily inhibited before the stimulus in order to prevent premature responses during the foreperiod. This proactive inhibition would be responsible for a paradoxical increase in RT for conditions with short foreperiods compared to control conditions in which no warning signal is presented. These results call for a reassessment of the theoretical framework used to interpret the effects of warning signals.

Keywords: EMG, Inhibition, Alertness, Visual detection, Motor preparation
INTRODUCTION

A number of behavioral, electrophysiological and neuroimaging experiments have demonstrated that visual attention and motor processes are intimately related (e.g. Rizzolatti et al. 1997; Brunia and van Boxtel 2001; Rushworth et al. 2003). Within this framework, it would seem reasonable that the presentation of a warning signal would speed up reaction time (RT) to a subsequent stimulus by changing neurophysiological activity at different levels of sensorimotor processing. However, more detailed analysis of this phenomenon demonstrates that the effects of warning signals on simple RT are likely more complex than this. Alternative interpretations to the benefits of warning signals are in fact possible and conflicting hypotheses about the locus of warning effects have been generated.

For more than a century the cognitive psychology of preparatory states has been studied. While virtually all studies agree that the effect of a neutral warning signal on RT are beneficial, they nonetheless disagree on the localization of this effect. Investigators have variously proposed that its origin lies in sensory/perceptual processing (e.g. Hackley and Valle-Inclan 1998), response selection/initiation (e.g. Hackley and Valle-Inclan 2003; Fernandez-Duque and Posner 1997) or motor events (e.g., Sanders 1983; Reddi et al. 2003). These hypotheses may not be mutually exclusive, however (e.g., Hackley et al. 2007). Recently, Fecteau and Munoz (2007) tested the neural mechanisms involved in warning by linking saccadic RT to the activity of visuomotor and motor neurons in the superior colliculus. They found both sensory warning effects in the form of an enhanced magnitude of sensory activity and motor warning effects in the form of a reduced threshold for initiating saccades as well as a faster rise in neuronal activity to reach this threshold. They proposed that motor events were the most important contributor to the warning effect.

Choice RT clearly requires substantial monitoring and inhibition to prevent incorrect responses (e.g., Burle et al. 2004; Stuss et al. 2005) and the traditional interpretation of simple RT is that it likely does not. However, an alternative interpretation of the warning effect on
RT may be made and we believe that monitoring and inhibition may also be at work in simple RT. This view is based on recent models which invoke temporal inhibitory control in simple RT (Brunia 1993; Los 2004; Narayanan and Laubach 2006; Narayanan et al. 2006) to simulate competition between activation and inhibition processes. They assume that inhibitory processes counteract both internal and external excitatory factors in order to prevent premature responses during the foreperiod.

In this study, we suggest that the warning signal is an important source of excitation that may induce a tendency to respond to the cue itself. Indeed Endo and colleaques have previously demonstrated that a visual stimulus which is not a target may automatically elicit increased activation in motor cortex (Endo et al. 1999). Recently, we demonstrated that a warning signal produces the same effect (Jaffard et al. 2007). This increase in activation may be compatible with motor hypotheses of the warning signal effect (Fecteau and Munoz 2007). However, it remains to be determined whether or not this relates to non specific motor preparation or to an erroneous reaction to the warning signal.

Here, we test the respective predictions of these hypotheses by analysing the distribution of errors in a classical, simple RT task in Humans. The standard hypothesis assumes a progressive increase in motor preparation related to expectation, as the probability that the stimulus will occur increases throughout the foreperiod (e.g., Näätänen 1970, Näätänen et al. 1974; Niemi and Näätänen 1981). More specifically, it predicts both a decrease in RT and a simultaneous increase in the amount of anticipation as the level of motor preparation rises throughout the foreperiod. Conversely, if the motor activations induced by warning signals are transient activations in response to these cues, anticipations will mainly be observed with a short fixed delay after the warning signal (i.e., anticipations will be distributed like RTs, but in relation to the warning signal as opposed to the stimulus).
METHODS

Errors represent a problematic but privileged means to understand and model information processing mechanisms (see Ratcliff et al. 1999 for review). In simple RT tasks, subjects may produce different reactions not directly evoked by the response signal: false alarms (RT are considered as invalid when the button is pressed even though no response signal was presented) but also other forms of anticipations (abnormally short RT considered as invalid, see Tiefenau et al. 2006 for discussion on data truncation and correction for false alarms and anticipations in simple RT). However, erroneous reactions may not systematically lead to overt false alarms and anticipations directly observable on RT data. Covert erroneous reactions may also be involved. In other words, analyzing errors solely on the basis of RT errors may be inconsistent. Alternatively, analyzing electromyographic (EMG) activations might provide an adequate and efficient tool to study both overt and covert errors (Allain et al. 2004; Burle et al. 2002a). Indeed, as assumed by the authors in choice RT experiments, subthreshold EMG activations are partial errors that were detected, aborted before reaching the overt response threshold, and successfully corrected. They provide evidence that an online, within-trial, executive control is involved. Obviously, EMG characteristics are also detectable on overt errors (i.e., when inhibitory mechanisms fail to stop the incorrect response in time). Thus, despite the fact that covert and overt errors may result from different cognitive processes (i.e., effective online inhibition vs. failure of inhibition), they can be collapsed together in the analysis of errors distribution because they are both supposed to index motor activation.

Subjects

Twelve right-handed males (aged 18 to 41 years), with normal or corrected to normal vision and without history of neurological or psychiatric disease, participated voluntarily in the experiment. The study was approved by the local ethical committee.
The paradigm consisted of a cued target detection task adapted from the classical studies of alertness and motor preparation (Fig. 1). Stimuli were projected onto a screen at a 50 cm distance from the participants’ eyes. The basic display was composed of a central fixation cross (1.2°). The warning signal consisted of two peripheral grey squares (1.37° wide, centered 10° on the left or right visual fields) presented during 50 ms. Target stimulus was a white X (0.57° wide, centered 10° on the left or right visual fields) present until subject’s response. Catch trials (without targets) were added (20%). Subjects were instructed to maintain fixation throughout the experiment and to respond as fast as possible once they detected the peripheral target, pressing a highly sensitive button with the right thumb. After target presentation, a 2000 ms delay was introduced before what is actually considered as the beginning of trial n+1 (starting with a variable 1100-1600 ms delay before possible cue presentation). However, no stimulus was associated with the start of trial n+1 in order to prevent it from acting as a supplementary warning signal. Subjects were instructed to comply with a maximum error rate of 5% on pain of being discarded from the analysis. When an overt response was given before target occurrence (false alarm) or too soon after target occurrence (anticipation: RT<100 ms), the trial was immediately aborted and an error signal was displayed on the screen informing subject about the amount of errors cumulated in the block.

Foreperiod duration is usually handled by means of stimulus onset asynchrony (SOA), which is the time between the onsets of the warning signal and the target. It was varied randomly between 100, 200, 300, 500, 700 and 900 ms. The use of several SOAs (at least three) is uncommon but fundamental for several reasons. First, the cue cannot be predictive of the exact moment of target appearance (with only two SOAs the cue may be used to predict precisely the time of target presentation and thus may serve instead as a temporal orienting cue as suggested by Posner and colleagues: Fan et al. 2005; Fernandez-Duque and Posner, 1997). Second, the precise temporal waning of the cueing effect can only be estimated when multiple SOAs are used. Third, it is important to provide short SOAs (what is not always the
case in cueing experiments) because studies using foreperiods as short as 100 ms clearly show that the decrease in RT is more prominent during the initial -100 to 400 ms- than during the last -400 to 1400 ms- part of a foreperiod (e.g., Fernandez-Duque and Posner, 1997). A total of 10 trials/SOA/target was presented (all in all, each block was composed of 145 trials: 60 for each target plus 25 catch trials). In order to avoid any bias related to mixing costs (see Jaffard et al. 2007), cued and no-cued trials were presented in two separate blocks of trials in a counterbalanced order across subjects. At this point, it is not trivial to notice that the variable SOA for no-cued trials is virtual (the actual start of a trial is not indicated by any stimulus). However, using this terminology allows comparing conditions which differ only with regard to the presence of the cue, all other events being strictly identical.

Electromyographic recordings

In order to facilitate the detection of EMG onsets for both correct and subthreshold activations, bipolar EMG recordings were performed (Fig. 1). Two Ag-AgCl electrodes (rochester), 11 mm in diameter were fixed 2 cm apart on the skin above the flexor pollicis brevis of the right thumb as described in Aldo and Perotto (2005). To ensure a unique contribution of the flexor pollicis brevis muscle to the thumb response, the handle was held such that the response button was placed on the line of the interphalangeal joint (i.e., insuring that only the proximal but not the distal phalanx of the thumb was involved in the movement). In addition, the forearm was placed in a splint in order to suppress postural emg activations and subjects were asked to relax. The EMG activity was monitored online during an experimental block. The automatic triggering of a trial could be suspended by the experimenter when the EMG signal was not stabilized. The signal was amplified (gain 250), filtered (10 Hz/1 khz for low/high frequencies cut-off, respectively), and digitized on-line (A/D rate 2 kHz).

Processing of EMG data
In order to optimize onset detection algorithms, data were further filtered offline with a second-order Butterworth filter (30 Hz lowpass cutoff frequency). We adapted a technique that allows the RT interval to be partitioned into premotor and motor components (e.g. Hasbroucq et al. 2003). *Premotor time* (PMT) is the time between the response signal and the onset of the voluntary EMG activity. PMT is supposed to reflect processes which occur prior to the activation of the motor system. *Motor time* (MT) is the time between the onset of the voluntary EMG activity and the closure of the switch (it just reflects peripheral motor processes and the speed of voluntary muscle contraction). For correct trials, we measured *premotor time* with this classical method. However, this experiment is intended to detect activations elicited by the warning signal. Therefore, for erroneous trials (false alarms and anticipations) we measured the latency of EMG activation with respect to warning signal presentation (*WS_EMG latency*), not target presentation. Accordingly, the different categories of errors observed in the experiment could be detected and collapsed together whatever EMG activations reached response threshold or not (Fig. 1). An automated algorithm inspired from Smid et al. (1990) was used. The EMG traces were also visually inspected off-line, trial by trial, as displayed on a computer screen. Since human pattern recognition processes are superior to automated algorithms (e.g., Van Boxtel et al. 1993), we hand-scored the EMG onset when the algorithm failed to detect it correctly, as it is usually done in experiments using this technique. Importantly, it must be emphasized that, at this stage, the experimenter was unaware of the type of trial he was looking at.

**** insert Fig. 1 about here ****

Even if our method allows detecting more false alarms than classical RT analyses do, the main and general problem of analyzing errors remains the limited number of trials on which the analysis is performed. Collecting a large amount of data from individual subjects does not guarantee to get stable results when collapsed because of interindividual variability.
Thus, for each individual trial we have normalized $WS_{EMG}$ latency with respect to the mean value of premotor time for correct no-cued trials:

$$Norm.WS_{EMG}\_\text{latency} = \frac{WS_{EMG}\_\text{latency}}{Mean.PMT_{(no-WS)}}$$

The rationale was the following: if the presentation of the warning signal elicits activations which are basically erroneous responses to warning signal presentation, $WS_{EMG}$ latency in erroneous trials should have the same characteristics as premotor time in correct no-cued trials, in other words should be close to 1. Thus, all data were collapsed together for distribution analysis (all in all, 202 values for erroneous trials which correspond to 11.6% of all trials).

We used the same rationale to collapse all data for correct cued trials on the basis of normalized premotor time values:

$$Norm.PMT = \frac{PMT}{Mean.PMT_{(no-WS)}}$$

**RESULTS**

*Correct trials*

A 2 Cue (cued, no-cued) x 6 SOA (100, 200, 300, 500, 700, 900 ms) ANOVA with repeated measures was applied to the data. Tukey tests were used for post-hoc analyses. A main effect of SOA ($F(5,55)=7.78, p<.001$) and a significant Cue by SOA interaction ($F(5,55)=9.3, p<.001$) were found. RT for cued trials with 100 ms SOA are i) greater than RT for no-cued trials with the same SOA (348 vs. 295 ms respectively, $p<.001$), and ii) greater than each cued RT with a longer SOA (348 vs. 305, 283, 291, 289 and 315 ms for, respectively, SOA 100 vs. 200, 300, 500, 700, 900 ms, $ps<.001$). All other comparisons failed to reach significant threshold. In other words, RT decrease with SOA lengthening is only observed from 100 to 200 SOAs and reveals interference rather than facilitation for short cue-target delays.
The same analysis was applied to *premotor time* and the same results were obtained ($F(5, 55)=10.65, p<.001$ for the Cue by SOA interaction, with $ps<.001$ for the same post-hocs). Conversely, no significant effect was found on *motor time*. In other words, *premotor time* and the underlying cognitive processes occurring prior to the activation of the motor system, rather than peripheral motor processes duration, may be responsible for the RT differences observed across cue and SOA conditions in correct trials.

**Correct no-cued trials, distribution analysis**

Correct no-cued trials serve as a reference for the forthcoming analysis of errors. The goal of this analysis is to characterize the RT distribution of our control trials in order to provide a reference for the analysis of errors distribution. *Normalized premotor time* distribution is presented in Fig. 2 (upper panel). This distribution is not normal, as confirmed by a Kolmogorov-Smirnov test ($d=0.08, p<.01$), but is asymmetric ($skewness=3.05$). It is clear that the increase of expectancy classically observed during non-aging foreperiods plays a direct role in the skewing of RT distribution (see Oswal et al. 2007 for recent convincing evidence). Thus, as expected, an Ex-Gaussian function was found to better fit the data (e.g. McGill 1963; Luce 1986). Accordingly, we used an adjustment algorithm based on the Simplex method (non linear optimisation algorithm). It was implemented with Matlab™ to find the parameters that best fit the following equation,

$$
\int x = \frac{1}{\tau} \exp \left( \frac{\mu + \sigma^2}{2\tau^2} - \frac{x}{\tau} \right) \phi \left( \frac{x - \mu - \frac{\sigma^2}{\tau}}{\sigma} \right)
$$

where $\phi$ is the normal cumulative function, and $\exp$ the exponential function. *Normalized premotor time* distribution was best fitted by an Ex-Gaussian function with $\mu = 0.827$, $\sigma =$ 0.099 and $\tau =$ 0.171 as parameters. A Khi² was used to test statistically the validity of this Ex-
Gaussian distribution. Observed and theoretical distributions were not significantly different (Chi square (11) = 5.26, \( p > .25 \)).

**Error distribution analysis**

The rationale was the following: If warning signals elicit automatic motor activations, then erroneous responses should be mainly observed with a fixed delay after cue presentation. More precisely, we expect errors to be distributed like control RT to targets (i.e., to be modelled by an Ex-Gaussian function centered on a warning signal-erroneous response delay similar to a target-correct response delay). In other terms, *normalized WS_EMG latency* should respect the same pattern of distribution as *normalized premotor time*. Accordingly, we tried to fit an Ex-Gaussian function to *normalized WS_EMG latency* data. However, no one was found that fits significantly the distribution. Errors rather seem characterized by a bimodal distribution. Data are presented in Fig. 2.

In order to provide a quantitative analysis, we have modelled a function intended to fit this bimodal distribution which is the sum of two Ex-Gaussian functions,

\[
\int(x) = \frac{ExGau(1) + C.ExGau(2)}{1 + C}
\]

where \( C \) is a coefficient taking into account the number of values belonging to each distribution.

Using the Simplex method, the parameters that best fit such a bimodal distribution are:

\( \mu_1 = 1.061, \sigma_1 = 0.140, \tau_1 = 0.155, \mu_2 = 2.602, \sigma_2 = 0.595, \tau_2 = 0.565 \) with \( C = 0.297 \). A \( \text{Khi}^2 \) was used to test statistically the validity of this model. Observed and theoretical distributions were not significantly different (Chi square (16) = 10.85, \( p > .25 \)). 95% confidence intervals were determined separately for each of the two Ex-Gaussian distributions. The first pool of data gathers 77% of all data (ranging from .7 to 1.98, that is from 159 to 450 ms), whereas the second one gathers 23% of all data (ranging from 1.38 to 5.32, that is from 313 to 1208 ms).

****insert Fig. 2 about here****
DISCUSSION

Recently, we have demonstrated with event-related fMRI that warning signals elicit increased activation of the sensorimotor cortex (Jaffard et al., 2007). However, it remains unclear if this rise in activation is related to anticipated and progressive non specific motor preparation, or to the transient activation of a response to the warning signal, or to the co-activation of inhibitory interneurons within the motor cortex. This study provides evidence in favour of the last two hypotheses. Indeed, while the ability to predict events and prepare a motor response would be expected to decrease RT, costs rather than benefits are observed when using an appropriate baseline as a control condition. In addition, the analysis of the distribution of errors suggests that warning signals trigger transient EMG activations within fixed short delays. We propose that these automatic activations require proactive volitional inhibition and that this inhibition may account for the observed RT effects.

Errors distribution

Despite the fact that subjects complied with the explicit instruction to respect a maximum overt error rate of 5% (based on RT analysis), the total number of errors (based on EMG analysis, both overt and covert errors combined) reached 11.9%. EMG onsets of erroneous trials reveal a clear bimodal distribution (Fig. 2). The first pool of data is composed of overt and covert responses to the warning signal. Indeed, these errors respect a RT-like distribution pattern which is nearly centered on the normalized value "1" of correct trials' premotor times (target_EMG onset delay). The second pool of data is composed of late errors which are likely to correspond to the anticipations described in the deadline model (Ollman and Billington 1972; Narayanan et al. 2006; Ratcliff et al. 1999). Importantly however, our quantitative analysis reveals that most of the errors observed in this simple RT experiment are warning signal-induced activations (77%). These false alarms observed on peripheral motor processes provide evidence that the warning signal is able to trigger automatic activations which may be responses to the warning signal (Fig. 2).
Obviously, inhibition is necessary at some point in order to avoid these activations provoking undesired responses to the warning signal (Picton et al. 2007). Inhibition can act through on-line executive control (Allain et al. 2004; Burle et al. 2002a). However, we assume that suppressing the current EMG activation is not the only inhibitory mechanism which is involved when a warning signal is presented (Jaffard et al. 2007). We suggest, rather, that covert and overt anticipations would reflect merely failures of standard inhibitory processes which would be proactively implemented precisely because any non-target stimulus would be able to trigger a premature response (i.e., when the activation induced by the warning signal would overcome proactive inhibition). The ability to inhibit a prepared action (volitional inhibition) has been investigated recently by assessing the excitability of the motor cortex during Go/NoGo tasks (e.g., Sohn et al., 2002; Coxon et al., 2006, 2007). These studies show that, while M1 excitability is known to be enhanced during preparation of a voluntary movement, it can be suppressed during volitional inhibition (see also Leocani et al., 2000). This can be explained by an increase in excitability of inhibitory interneurons within M1 acting to reduce the output of the corticospinal pathway. Since we have demonstrated that a warning signal may act as a NoGo stimulus (Jaffard et al., 2007), it is likely that this neural mechanism may directly contribute to the increase in RT observed when a target is presented very soon after a warning signal (i.e., before proactive volitional inhibition has been suppressed). In support of this hypothesis, Davranche et al. (2007) have recently suggested that the function of such inhibitory mechanisms was to secure the development of cortical activation during movement preparation in order to prevent erroneous responses. In their TMS experiment, the silent period that follows the MEP in the ongoing EMG was used as an index of intracortical inhibition and analyzed across different levels of preparation (see also Burle et al. 2002b). Since the removal of intracortical inhibition was more pronounced when preparation was optimal, the authors concluded that inhibitory and activation processes occur...
in parallel and that suppression plays an important role in time preparation and, hence, in RT effects.

**Relation to RT pattern**

This inhibition hypothesis is supported by RT analysis of correct cued trials. Indeed, when comparing trials with and without warning signals, interference rather than facilitation is observed for short foreperiods. If proactive volitional inhibition is implemented as soon as a trial starts in order to counteract the automatic activations which are likely to be triggered later by the visual warning signal, then, the role of the warning signal would consist in releasing this proactive inhibition when identified. Obviously, processing the visual signal and releasing inhibition is time consuming. Accordingly, when the target is presented soon after the warning signal, inhibition has not yet extinguished and RT is greater than the appropriate baseline value (i.e., a control condition without warning signal). After inhibition has been released (for longer foreperiods), RT simply returns to baseline (Jaffard et al. 2007).

**Resolving controversies about warning signal effects**

Results of this study suggest that a neutral warning signal does not provide a facilitation effect, neither at short nor at long foreperiods and thus clearly contradict the literature. However, the inhibition hypothesis resolves this controversy if careful attention is paid to the methods that have been used classically. First, most studies dealing with expectancy (momentary probability of the immediate delivery of the response signal) have

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1 Other sensory hypotheses (see Niemi and Näätänen for review) can be ruled out in this experiment. Indeed, as suggested by other behavioural data (Jaffard et al., 2005), neither the relative intensity of the warning stimulus nor to its location with regard to the target can fully account for the whole increase in RT with respect to the baseline. In other words, neither intensity as a criterion for stimuli discrimination nor forward masking effects can explain the strong RT increase observed at short foreperiods. Conversely, transforming the current experiment into a Go/NoGo task (Jaffard et al., 2007) clearly leads to an increase in Go RT (with respect to the baseline) which closely corresponds to the size of the cueing effect (SOA 100 ms minus baseline). Nevertheless, it is clear that the amount of perceptual stimulation from the warning stimulus may influence the amount of motor activation, as predicted by relations between stimulus intensity, response force and motor times (e.g., Jaśkowski et al. 1995; Ulrich et al. 1998). Accordingly, the risk that automatic motor activations reach movement threshold and trigger an undesired response may vary with the intensity of the warning signal with respect to the target. In other words, it is likely that the need to inhibit temporally inappropriate responses may differ with the amount of perceptual stimulation from the warning stimulus. This is in accordance with the observation that RT systematically increases with a corresponding increase in warning signal intensity (e.g., Kohlerfeld 1969).
focused only on the relation of foreperiod duration to reaction time (reviewed in Niemi and Näätänen 1981). In other words, most of these studies have focused on the warning signal effect only by considering the evolution of RT according to the WS-target delay and have not used a baseline to control for the cueing effect. Second, studies of alertness, which do use baselines, typically employ mixed experimental designs (e.g. Fan et al. 2005; Fernandez-Duque and Posner 1997; Fecteau and Munoz 2007) in which trials with and without warning signals are intermixed in the same block of trials. According to our interpretation, proactive inhibitory processes might have a critical effect on such a baseline (no-cued trials intermixed with cued trials). Indeed, when a target is presented without a preceding cue in a mixed design, proactive inhibition is maximal and cannot be released until the target is identified as a target, and RT remains maximal whenever the target appears. In other words, the baseline used to compute cueing effects would be biased by this inhibition and cueing benefits in mixed designs would just be a misinterpretation of the data (Jaffard et al. 2007). Conversely, in a block design, as used in this study, no proactive inhibition is required for no-cued trials and RT baseline is not biased. Accordingly, we challenge the classical view, interpreting short term warning signal effects as the benefits of alerting.

The idea that automatic motor activations may be elicited by visual information is by no means new (Sperry 1952); however, little interest has been paid to the secondary effects this may have on executive control. This study has provided simultaneously: i) direct evidence that warning signals are able to trigger activations as automatic responses to the warning signal, ii) direct evidence for the involvement of online control processes suppressing ongoing erroneous movements, and iii) indirect evidence for the involvement of earlier (proactive) and more efficient inhibitory mechanisms which account for short-term warning signal effects on simple RT.
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Authors Notes:

The authors are grateful to Giovani Berlucchi and Carlo-Alberto Marzi (Dipartimento di Scienze Neurologische e della Visione, Verona, Italia) for their help in conceiving the project and discussing data. They are also grateful to Sander Los for helpful comments and suggestions on an earlier version and Thomas Steeves for carefully reading and correcting the manuscript. This work was done at LPMC EA3814 and was funded by the French Ministry of Research (A.C.I. JC 6042 to PB). Magali Jaffard was supported by a doctoral fellowship from the French Ministry of Research.
Figures captions:

**Figure 1:** Schematic representation of the experimental procedure and illustration of the different types of EMG activations observed during the experiment.

**Figure 2:** Error distribution (*normalized WS EMG latency*) is bimodal (the sum of two Ex-Gaussian functions, black line). The earlier and higher distribution represents overt and covert false alarms (responses to the warning signal) whereas the smaller and later one would rather contain anticipations related to the increasing probability of stimulus occurrence as foreperiod duration increases (deadline model). The distribution of correct trials (*normalized premotor time*) in the control condition (no warning signal) is represented in the up right corner.
Variable delay (1100-1600 ms)  WS (50 ms)  Variable SOA (100-900 ms)  Target (150 ms)

Correct trial

Anticipation (abnormally short RT)

False Alarm (observed on subthreshold EMG activation)

False Alarm (observed on subthreshold EMG activation)

False Alarm (observed on subthreshold EMG activation)

Normal trials

Catch trials

RT=292 ms

RT=90 ms

RT=348 ms

RT=error