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Pre- and Poststimulus Activation of Response Channels: A Psychophysiological Analysis

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To examine mechanisms of response activation, we asked subjects to respond differentially to the central letter of one of four arrays—HHHHH, SSHSS, SSSSS, and HHSHH—and measured event-related brain potentials (ERPs) and electromyographic activity (EMG). For very fast responses, accuracy was at chance level for all arrays, suggesting that subjects were guessing. For intermediate latency responses, accuracy was above chance if the noise was compatible with the targets and below chance if it was incompatible, suggesting that these responses were based on partial stimulus analysis. For slow responses, accuracy was above chance for all arrays, suggesting that these responses were based on complete stimulus analysis. The occurrence and accuracy of fast responses could be predicted by examining motor potentials preceding the presentation of the array. Measures of the motor potentials in the period following the presentation of the array suggested that partial analysis of stimulus information could activate responses and that the level of response activation at the time of the EMG response was constant for trials with different response latencies. The data are discussed in terms of a response channel conception.

In the present article we report results of an investigation of the mechanisms that lead to the activation of responses in a choice reaction time (RT) paradigm. It is usual to define "responses" as the mechanical events (e.g., switch closures) that are recorded by the experimenter. However, these events are, in fact, the consequence of a complex chain of causally related phenomena that can be traced back through the spinal cord and the motor areas to various presetting mechanisms. Although closing the switch is the goal imposed on the subject by the experimenter, the real task of the subject is to accomplish the movement that will result in the switch closure. Thus, to understand how the switch comes to be closed, it is arbitrary to choose the point of switch closure as the level at which the analysis should be conducted. Indeed, as we hope to illustrate, we are led to a much richer understanding of the processes involved in a choice RT task when we broaden our research focus to include different levels of the response process rather than restricting ourselves to the switch closure.

Support for this approach can be found in studies of response preparation and response competition, which have both been interpreted in terms of "subthreshold" response activation, that is, activation of the response system that is not sufficient to trigger an overt response. Thus, response preparation can be conceptualized as an energizing phenomenon (Posner, 1978; Requin, 1985) by which response structures are activated at a subthreshold level. Indeed, when Requin (1985) recorded from the motor cortex of monkeys, he observed an increase in the firing rate of neurons during the foreperiod of RT tasks. Some of these neurons showed a further increase in firing rate during the execution of the overt response. Response competition, on the other hand, can be viewed as the reciprocal inhibition of competing response structures (see Eriksen & Schultz, 1979; Sherrington, 1906) in which the subthreshold activation of one response leads to a delay in the execution of another. This interpretation of response competition has been supported by the results of studies in which electromyographic (EMG) responses were recorded in association with overt responses (see Coles, Gratton, Bashore, Eriksen, & Donchin, 1985; Eriksen, Coles, Morris, & O'Hara, 1985).

The Concept of Response Channels

The idea that there can be various levels of response-related activity in the nervous system can be conceptualized in terms of the notion of *response channel*. We use this term as a heuristic device to refer to that complex of structures whose activities are more or less directly related to the mechanical event that is defined as the overt response. A similar conception can be found in Gaillard (1978) and Näätänen and Merisalo (1977).

We consider the response channels to be distinct from the stimulus evaluation system, whose function is to analyze stimulus information, resulting in activation of the appropriate response channel. However, the level of activity of the response channel may be affected by other factors such as response priming or bias and response competition.

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Within this framework, different degrees of involvement of response structures are viewed as different degrees of response channel activation. Thus, response preparation can be represented as an increase in the level of activation of a response channel, whereas response competition is a decrease in the level of activation in one response channel as a result of the increase in the level of activation of another channel. The involvement of particular response structures can be represented by different thresholds of response channel activation. Thus, there is a threshold for the initiation of muscle activity, as revealed in the EMG, and a higher threshold for the initiation of an overt movement (see Coles et al., 1985).

Measuring the Activation of Response Channels

Information about the level of activation of the response channels can be gained by studying the activity of neural and muscular structures involved in the response, as well as by examining the overt response. In our previous work (Coles et al., 1985; Eriksen et al., 1985), we recorded both the EMG activity and the overt response (a squeeze of a dynamometer) to determine the time at which the level of response channel activation reached two particular thresholds (that for the EMG response and that for the squeeze response). Although this procedure allowed us to identify two points in the response channel activation function, it did not provide a continuous, analog description of the function.

In the present study, we used a component of the event-related brain potential (ERP), the readiness potential, as a measure of response channel activation. This component was first described by Kornhuber and Deecke (1965) in a study of self-paced movements. These authors recorded electrical activity from scalp electrodes and observed a negative, ramp-like potential that began some 800 ms prior to the execution of a movement and peaked just after the onset of the movement. Later research has shown that this potential is largest at scalp electrodes placed over the hemisphere contralateral to the hand responsible for the movement (e.g., Kutas & Donchin, 1974, 1977, 1980; Vaughan, Costa, & Ritter, 1972). Furthermore, several investigators (e.g., Kutas & Donchin, 1980; Rohrbaugh, Syndulko, & Lindsley, 1976) have described a similar lateralized potential that develops during the foreperiod of simple warned RT tasks. The peak of this lateralized potential, which occurs after the imperative stimulus, appears to be time-locked to the overt response. A similar lateralized potential develops *after* the imperative stimulus in choice RT tasks, when response choice depends on information provided by the imperative stimulus, but *before* the imperative stimulus, when the choice depends on information provided by the warning stimulus (Kutas & Donchin, 1980). Thus, the potential appears to become lateralized when a choice is made about the responding hand.

These observations led Kutas and Donchin to argue that the lateralization of the readiness potential can be viewed as an index of specific motor preparation.[1] Subsequent reports (e.g., Okada, Williamson, & Kaufman, 1982), which have used neuromagnetic techniques to relate this potential to motor cortex activity, also lend credibility to the suggestion that the readiness potential is a good candidate for a psychophysiological measure of response channel activation. Therefore, we propose to use the lateralization of the readiness potential as a measure of the relative activation, or priming, of response channels *prior* to stimulus presentation. Further variations in the lateralization of the readiness potential occurring *after* the presentation of the stimulus should reflect the relative activation of the response channels as a consequence of the processes of stimulus evaluation.

Prestimulus Activation

Prestimulus activation refers to activation of a response channel prior to the presentation of the imperative stimulus. When prestimulus activation occurs, the corresponding response should be facilitated, because the activation level at the time of stimulus presentation is closer to the overt response threshold. In extreme cases of prestimulus activation, small increases in activation that may occur as a consequence of mere stimulus presentation are sufficient to raise the activation level above the threshold and trigger the response. In this way, the probability of emitting a response in the absence of stimulus information will be enhanced. Responses of this type are commonly labeled *fast guesses*. According to this view, then, fast guesses occur when there is an increased level of response channel activation prior to the imperative stimulus. Such a hypothesis is compatible with explanations of the effects of instructional sets, priming or warning stimuli, and manipulations of response probability on the accuracy and latency of responses (see, for instance, Posner, 1978). More generally, this hypothesis—the variable baseline hypothesis—interprets variability in response bias in terms of variability in the baseline level of response channel activation.

This hypothesis differs from that proposed by Grice, Nullmeyer, and Spiker (1982), who argued that fast guesses are a consequence of variations in the setting of the response criterion. According to this "variable criterion hypothesis," fast guesses occur when the response criterion is set to such a low level that small fluctuations in the activation of the response channel (occurring after the stimulus) are sufficient to cross the criterion, or threshold, and trigger the response.

We propose that measures of the lateralized readiness potential can be used to evaluate these two hypotheses. In particular, if trials on which a fast guess occurs are associated with a greater prestimulus activation than slow response trials, then we should observe a larger lateralized readiness potential

[1] Another negative component, labeled *contingent negative variation* (CNV; Walter, Cooper, Aldridge, McCallum, & Winter, 1964) can be observed in the foreperiod of warned RT tasks (see, for instance, Coles et al., 1985). The distinction between the readiness potential and the CNV has recently been debated (see Rohrbaugh & Gaillard, 1983). In particular, the possibility of a CNV in the absence of an overt response has been discussed. The arguments related to this debate are beyond the scope of this article. We note that, in general, the literature about CNV has focused on its relation to "attentional" or "alertness" states, while that on the readiness potential has focused on its relation with motor responses. Because we will consider the scalp negativity preceding the stimulus in terms of its relation with the motor response, we will use the label *readiness potential* to refer to it.

during the foreperiod of fast guess trials. On the other hand, if fast guesses occur when the response criterion is set to a lower level than for slow response trials, there should be less lateralization of the readiness potential at the time the overt response is initiated on fast guess trials.

Poststimulus Activation

Poststimulus activation refers to the activation of the response channels that occurs as a consequence of the processing of the imperative stimulus. In our previous work (Coles et al., 1985; Eriksen et al., 1985), we have found evidence to suggest that responses can be activated in a parallel and continuous fashion as stimulus evaluation proceeds. We used a choice RT paradigm in which the stimulus was made up of a central target letter flanked by noise letters (cf. B. Eriksen & C. Eriksen, 1974). The noise letters could call either for the same response as the target (compatible noise) or for the opposite response (incompatible noise). Analysis of EMG and overt response measures revealed that on some trials both responses were initiated (Coles et al., 1985; Eriksen et al., 1985). These double responses occurred more often when incompatible noise was presented, suggesting parallel processing of target and noise information. Furthermore, conditional accuracy functions (i.e., accuracy conditional on latency) revealed that when subjects responded quickly to incompatible noise stimuli, they tended to make errors. Indeed, they erred more than would be predicted on the basis of chance responding (Coles et al., 1985). On the other hand, slow responses to incompatible noise stimuli were associated with high accuracy. The accuracy of responses on compatible noise trials was always greater than chance. These data suggest that responses are sometimes given on the basis of a preliminary analysis of stimulus information, whose output is dominated by the noise letters. In the case of incompatible noise, the output of this preliminary analysis leads to the incorrect response, because it is dominated by the noise letters.

These data support the thesis that response channels can be activated by partial analysis of stimulus information. Therefore, the lateralization of the readiness potential in the period following the presentation of the imperative stimulus, and preceding the overt response, should provide evidence of the influence of this partial analysis. In particular, if the imperative stimulus contains incompatible noise, we should observe activation of the incorrect response.

The Present Experiment

We have proposed that several factors can affect the activation of response channels. These include pre- and poststimulus response activation. Furthermore, we have argued that the lateralized readiness potential can be used as a measure of the relative activation of response channels.

These ideas were explored in the present experiment. In particular, we used electrophysiological measures to investigate the mechanisms responsible for fast guesses and to provide a description of the influence of evaluation processes on the response system. To this end, subjects performed a version of the noise/compatibility task (cf. Coles et al., 1985). They were instructed to respond quickly, and a large number of trials were collected so as to provide a reasonable sample of fast guesses. Thus, in the present experiment we replicated the basic design of the Coles et al. (1985) study. However, rather than focusing on the temporal relation between two measures of peripheral response activation (EMG and overt movement), we concentrated on a more central measure of response activation. In particular, we recorded the readiness potential from lateral scalp electrodes to derive an analog measure of the relative activation of response channels.

Method

Subjects

Six graduate students (2 females) at the University of Illinois were paid $3.50 an hour, plus bonuses, for participation. The subjects (between 23 and 25 years old) had normal or corrected-to-normal vision and hearing. The subjects were not informed of the precise purposes of the experiment.

Stimuli

Each trial was initiated by a warning tone (1000 Hz, 50-ms duration, 65-dB amplitude), generated by a Schlumberger sine-square audio generator (Model SG-18A) and administered binaurally through headphones. One thousand ms after the warning tone, a five-letter array (HHHHH, SSHSS, SSSSS, or HHSHH) was presented on a DEC VT-11 CRT display for 100 ms. The interval between two consecutive trials varied randomly between 3,500 and 5,500 ms. The subject sat facing the screen at a distance of 1 m in such a way that the angle subtended by each letter was approximately 0.5°. Thus, the angle subtended by the whole array was approximately 2.5°. A fixation dot, placed 0.1° above the location of the target letter, remained visible throughout the experiment.

Procedure

Each subject took part in one practice and three experimental sessions. The subjects received 17 blocks of trials during each of the four sessions. In the first 15 blocks (made up of 80 trials each), one of the four arrays was presented on each trial. In the last 2 blocks (made up of 40 trials each), one of only two arrays (either HHHHH and SSHSS or SSSSS and HHSHH) was presented on each single trial. Thus, in the last two blocks the central stimulus was always the same, making these blocks equivalent to a "simple RT" task rather than the "choice RT" task used in the first 15 blocks.[2] In each case, the stimulus arrays were equiprobable (.25 for each stimulus array in the first 15 blocks, .50 in the last two blocks), and their presentation order was randomized. Each subject was instructed to respond to one of the two target letters (*H* or *S*) with one hand (left or right) and to the other target letter with the other hand by squeezing a zero-displacement dynamometer. The association between target letter and responding hand was consistent for each subject and counterbalanced across subjects. Speed was emphasized over accuracy, and subjects were trained, by means of verbal feedback, to respond at a speed that produced error rates ranging between .10 and .20.

[2] The simple RT condition was used to determine whether in fact a scalp lateralization of the readiness potential could be observed under conditions of extreme response bias. Because such a result was in fact obtained, we will not discuss this condition further.

Overt Response Measurement

When subjects squeezed the zero-displacement dynamometers (Daytronic Linear Velocity Force Transducers, Model 152A, with conditioner amplifiers, Model 830A; see Kutas & Donchin, 1977), a voltage was generated which was proportional to the force applied to the transducer. This signal was digitized at 100 Hz for 1,000 ms following array presentation, giving a continuous recording of the force output of both hands following each stimulus. A Schmitt-trigger could be set to any preselected force level. When the force reached a prescribed criterion, the system recorded the occurrence of an overt "criterion" response. Before the practice session, the value of each subject's maximum squeeze force was determined for each hand separately. Criterion values for each subject were set at 25% of the maximum force applied by that subject for that hand. During the practice sessions only, a click was presented over a loudspeaker whenever the force exerted on the transducer crossed the criterion.

Psychophysiological Recording

The electroencephalogram (EEG) was recorded from Fz, Cz, Pz, (according to the 10/20 system, Jasper, 1958), C3′ (4 cm to the left of Cz), and C4′ (4 cm to the right of Cz) referenced to linked mastoids, using Burden Ag/AgCl electrodes. Vertical and horizontal electro-oculographic activity (EOG) was recorded by using Beckman biopotential Ag/AgCl electrodes placed above and below the right eye and at 2 cm external to the outer canthus of each eye. Ground electrodes were placed on the forehead. The EMG was recorded by attaching pairs of Beckman electrodes on both right and left forearms, using standard forearm flexor placements (Lippold, 1967). For EEG and EOG electrodes the impedance was less than 5 Kohm, for EMG, below 20 Kohm. The EEG and EOG signals were amplified by Grass amplifiers (model 7P122) and filtered on-line by using a high-frequency cut-off point at 35 Hz and a time constant of 8 s for the high-pass filter. The EMG signals were conditioned by using a Grass Model 7P3B preamplifier and integrator combination. The preamplifier had a half-amplitude low-frequency cut-off at 0.3 Hz, while the output of the integrator (full wave rectification) was passed through a filter with time constant of 0.05 s. For each psychophysiological measure (EEG, EOG, and EMG) and each trial, the derived voltages were digitized at 100 Hz for 2,100 ms, starting 100 ms prior to the presentation of the warning tone and ending 1,000 ms after the presentation of the array.

Data Reduction

Motor responses. As we noted above, the subjects were required to squeeze the dynamometers to a criterion of at least 25% of maximum force to register a "criterion response." This response criterion was used for on-line feedback, and RT was defined as the latency at which this criterion was crossed. However, a different response classification system was used for most of the analyses. In particular, a measure based on the *onset latency of the EMG response* was used.

The onset latency of the EMG response was evaluated on each trial according to the following procedure (cf. Coles et al., 1985). First, a minimum criterion value was established for each subject to discriminate the EMG response from random variations in background EMG. Because the background EMG was typically characterized by very small amplitude activity, the EMG response was readily identifiable. When the integrated EMG exceeded the minimum criterion, an EMG response was defined as having started, and the latency of this activity was noted. EMG responses in both arms could be observed on the same trial. In the present article, we will focus on analyses based on the onset latency of the first EMG response.

ERP measures. For each single trial, the EEG data were corrected for both vertical and horizontal ocular movement artifacts by using a modification of the procedure described in Gratton, Coles, and Donchin (1983). The corrected single trial data were then stored for further analysis, including computation of averages and analysis of ERP component parameters on each single trial. For the single trial analysis, the data from the five scalp electrodes (Fz, Cz, Pz, C3′, and C4′) were smoothed by using a low-pass digital filter (high-frequency cut-off point at 3.14 Hz, two iterations), and the baseline level was subtracted by averaging the first 10 points of the epoch (corresponding to 100 ms).

The *readiness potential* was assessed by using the ERP waveforms recorded at C3′ and C4′. These electrodes were placed on scalp regions close to brain motor areas. Previous research has shown that the amplitude of the readiness potential is maximum at these locations when squeeze responses are required (Kutas & Donchin, 1977; Bashore, McCarthy, Heffley, Clapman, & Donchin, 1982).

Results

The results section will be divided into several parts. First, we describe the basic conditional accuracy functions for the two noise/compatibility conditions. Second, we present data concerning the influence of prestimulus response activation mechanisms on the latency and accuracy of overt responses. Third, we analyze the mechanisms of poststimulus activation by studying the influence of stimulus evaluation on the relative activation of the two responses. Fourth, we consider the possibility that responses are released when relative response activation reaches a fixed criterion. Finally, we describe an analysis of the absolute, rather than relative, activation of each response.[3]

Conditional Accuracy Functions

Conditional accuracy functions were derived for compatible and incompatible conditions and for each of the 6 subjects by computing response accuracy for each of seven 50-ms response latency bins ranging from 100 to 449 ms (cf. Lappin & Disch, 1972a, 1972b; Pachella, 1974). The accuracy and latency of each "response" were defined either in terms of the first criterion squeeze (Figure 1, left panels) or onset of the first EMG response (Figure 1, right panels). Average functions are shown in the upper panels of Figure 1, while the proportions of responses for each latency bin are shown in the lower

[3] In our previous study (Coles et al., 1985), we analyzed both EMG and squeeze onset latencies. Squeeze and EMG latency data from the present study replicated those obtained previously for the "random-warned" condition. In particular, for incompatible arrays relative to compatible arrays, we observed (a) longer RTs (by 41 ms), $F(1, 5) = 59.16$, $p < .001$; (b) longer intervals between EMG and squeeze onsets (by 6 ms); $F(1, 5) = 7.66$, $p < .05$; and (c) more trials for which both squeeze responses were initiated, $F(3, 15) = 24.30$, $p < .001$. Furthermore, on trials where both squeeze responses were initiated, the EMG to squeeze interval was prolonged by 18 ms, $F(2, 10) = 32.52$, $p < .001$. Thus, data from the present experiment confirm that response competition is a contributing factor to the differences in mean RTs between compatible and incompatible noise stimuli.

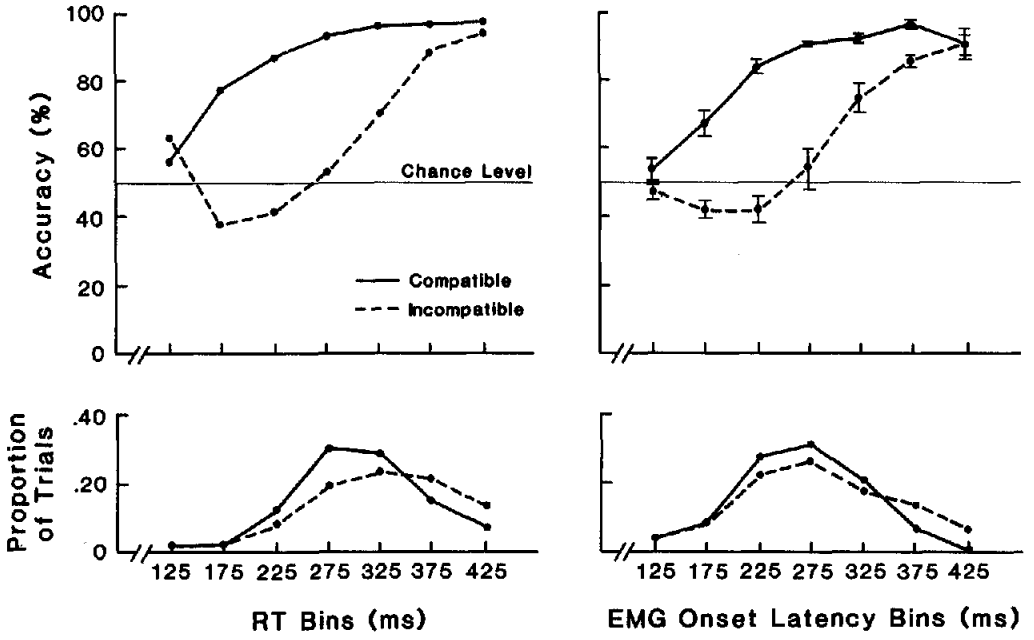

Figure 1. Conditional accuracy functions (upper panels) and proportion of trials for each response latency bin (lower panels) for compatible (solid) and incompatible (dashed) trials. (Values on the ordinate correspond to the midpoint of each response latency bin [reaction time, RT, in milliseconds]. Data for trials classified on the basis of criterion squeeze response are shown in the left panels, and for trials classified on the basis of the electromyographic activity [EMG] onset in the right panels. For the conditional accuracy functions based on EMG onset latency, standard errors are indicated by the vertical bars. For criterion squeeze response functions, only 3 subjects had trials in the 100–149 ms latency bin.)

panels. The conditional accuracy functions based on the two response definitions are qualitatively similar, suggesting that both measures are manifestations of the activity of the same underlying system. However, there are many fewer criterion squeeze responses than EMG responses with a latency shorter than 200 ms. (In fact, only 3 subjects had criterion squeeze responses with a latency of less than 150 ms.) This supports the view that EMG responses represent a more sensitive measure of response activation than the squeeze response (see also Coles et al., 1985; Eriksen et al., 1985). Furthermore, EMG responses are less affected by response competition than are squeeze responses (see Coles et al., 1985, and footnote 3). These two characteristics of the EMG onset measure make it more suitable than the criterion squeeze response as an index of peripheral response activation. Therefore, we used the EMG measure in the remainder of this article as the basis for response classification.

Analysis of the conditional accuracy functions based on EMG onset revealed that the proportion of correct responses increased with increasing response latency, $F(6, 30) = 59.78$, $p < .001$, and that subjects were more often correct on compatible than incompatible trials, $F(1, 5) = 144.40$, $p < .001$. The most interesting aspect of these functions, however, was that they differed in shape, as reflected in the significant interaction between compatibility and response latency, $F(6, 30) = 9.96$, $p < .001$. As can be seen in Figure 1, the most noticeable difference between the functions occurred at response latencies of between 150 and 249 ms. For responses in the 150–199 and 200–249 latency bins, accuracy was significantly greater than chance (.50) for compatible arrays, $t(5) = 4.25$ and 15.72 (one-tailed), but significantly below chance for incompatible arrays, $t(5) = -3.47$ and -2.34 (one-tailed). This differential pattern of response accuracy, which was evident in all subjects, suggests that for these response latencies the noise letters are controlling response accuracy (cf. Coles et al., 1985). Because the noise letter in incompatible arrays calls for the incorrect response, responses for these arrays tended to be less accurate than chance. On the other hand, very fast responses (with a latency of less than 150 ms) tended to have a chance level of accuracy regardless of the compatibility of the array. For compatible and incompatible arrays, response accuracy was not significantly different from chance (50%), $t(5) = 0.80$ and -1.22, respectively. Because the accuracy of these responses was unaffected by stimulus information, they could be classified as "fast guesses." Conversely, slow responses, with a latency greater than 300 ms, tended to be correct regardless of the compatibility of the array. As we have argued previously, the accuracy of these slower responses was controlled by analysis of target letter information (cf. Coles et al., 1985).

Prestimulus Activation

In this section, measures of the lateralized readiness potential are used to describe the pattern of response activation processes that occurs during the foreperiod (between warning

tone and array presentation). In particular, we focus on the question of whether the accuracy of fast-guess responses is under the control of an activation process that occurs before array presentation. Such an analysis should reveal whether these fast responses are associated with a higher baseline level of response activation, as predicted by the variable baseline hypothesis.

To examine the activation process, we computed the difference in voltage between the two laterally placed electrodes (C3′ and C4′) for every time point on each trial beginning 100 ms before tone onset and extending through the time of array presentation. As we noted earlier, the potential at these electrode sites is more negative on the side contralateral to the responding hand, when subjects are informed which hand to use to respond (see Kutas & Donchin, 1980). Thus, we predicted that the direction and degree of laterality observed during the foreperiod would be related to the nature of the response to the array. The values of lateralization were computed on the basis of which hand appeared to be correct on a particular trial, so that negative values (upward deflections in Figures 2, 4, and 5) always indicate more negativity at the electrode site contralateral to the correct response.

To compare lateralization waveforms preceding fast and slow responses, we sorted trials according to EMG onset latency (using 50-ms bins ranging from 100 to 399 ms), response accuracy (correct or incorrect response) and noise/compatibility (compatible or incompatible noise). To obtain stable estimates of the lateralized brain potential, a data base of at least 50 trials must be used to derive average waveforms. In fact, the shortest EMG onset latency bin (100–149 ms) for all types of trial and later EMG onset latency bins for incorrect compatible trials did not match the 50-trial criterion. For this reason we had to restrict our analysis to the remaining cells (EMG onset latency bins between 150 and 399 ms for correct compatible trials and for correct and incorrect incompatible trials). Note that the shortest EMG onset latency bin for which average lateralization waveforms were computed was 150–199 ms. Although the conditional accuracy data indicated that fast guess trials were most evident in the 100–149-ms bin, a substantial number of the trials in the 150–199-ms bin are also fast guesses, because accuracy is still quite close to chance for this bin.

Figure 2 shows average laterality values from incompatible trials during the foreperiod for correct and incorrect response trials with different response latencies (150–199 ms in the lower panel and 300–349 ms in the upper panel). As with the conditional accuracy functions, we used the EMG response to define both response accuracy and response latency.

Inspection of Figure 2 suggests that the readiness potential preceding fast responses becomes lateralized during the foreperiod and that the direction of the laterality is related to the correctness of the subsequent response. This inference is supported by several analyses. First, when laterality is defined in terms of the mean absolute value of the C3′–C4′ difference in the last 100 ms of the foreperiod, fast responses are associated with a greater degree of laterality than slow responses (the analysis was restricted to correct responses and to latency bins ranging between 150 and 399 ms because of unstable ERP data on other bins), $F(4, 20) = 3.31$, $p < .05$. Further analysis revealed a significant linear trend in lateralization as a function of EMG onset latency bin, $F(1, 20) = 10.45$, $p < .01$. Second, the direction of the laterality is related to response accuracy. Fast correct responses are associated with laterality in the direction of the correct response (greater negativity at the electrode location contralateral to the correct response), while fast incorrect responses are associated with laterality in the direction of the incorrect response (greater negativity at the electrode location ipsilateral to the correct response), $F(1, 5) = 19.54$, $p < .01$. (This analysis was restricted to incompatible trials because there were not enough incorrect responses to compatible arrays to yield stable averages.)

Finally, we classified each individual trial into one of three categories: trials for which the readiness potential was larger over the motor cortex contralateral to the correct response ("contralateral" trials); trials for which the readiness potential was larger over the motor cortex ipsilateral to the correct response ("ipsilateral" trials); and trials for which there was no significant difference between the two sides ("nonlateralized" trials).[4] We computed the conditional accuracy functions separately for each of these categories of trials. The average conditional accuracy functions for compatible and incompatible trials computed in this way are shown in Figure 3.

We predicted that the accuracy of fast guesses would be greater than chance when the correct response was prepared in advance of stimulus presentation—that is, for contralateral trials—and below chance when the incorrect response was prepared in advance of stimulus presentation—that is, for ipsilateral trials. This prediction was confirmed. An analysis of variance conducted on the conditional accuracy functions (for which accuracy was used as a dependent variable and noise compatibility, response latency bin, and trial category were used as factors) indicated a significant main effect of trial category, $F(2, 10) = 5.66$, $p < .05$, as well as a significant two-way interaction between trial category and response latency bin, $F(10, 50) = 2.88$, $p < .01$. Subsequent analyses revealed, for the main effect of trial category, a significant linear trend with decreases in accuracy from contralateral to nonlateralized to ipsilateral trials, and, for the interaction, a significant linear trend only for the first response latency bin (between 100 and 149 ms).

Taken together, these data suggest that subjects sometimes chose and activated a particular response during the foreperiod before the array was presented, although, objectively, each

[4] Estimates of the magnitude of the lateralized readiness potential on each single trial were obtained at each electrode by computing the average EEG activity of the 100 ms preceding the appearance of the stimulus array. Single trials were then classified into one of three categories (ipsilateral, contralateral, and nonlateralized) by comparing the readiness potential at C3′ and C4′. The voltage difference between these two electrodes was transformed into standard scores (for each subject). If the standardized difference value between the two electrodes was between +0.5 and −0.5, the trial was said to be "nonlateralized." If the difference was larger than this criterion, the trial was said to be "ipsilateral" if there was more negativity at the electrode ipsilateral to the hand corresponding to the correct response, and "contralateral" if there was more negativity at the electrode contralateral to the hand corresponding to the correct response.

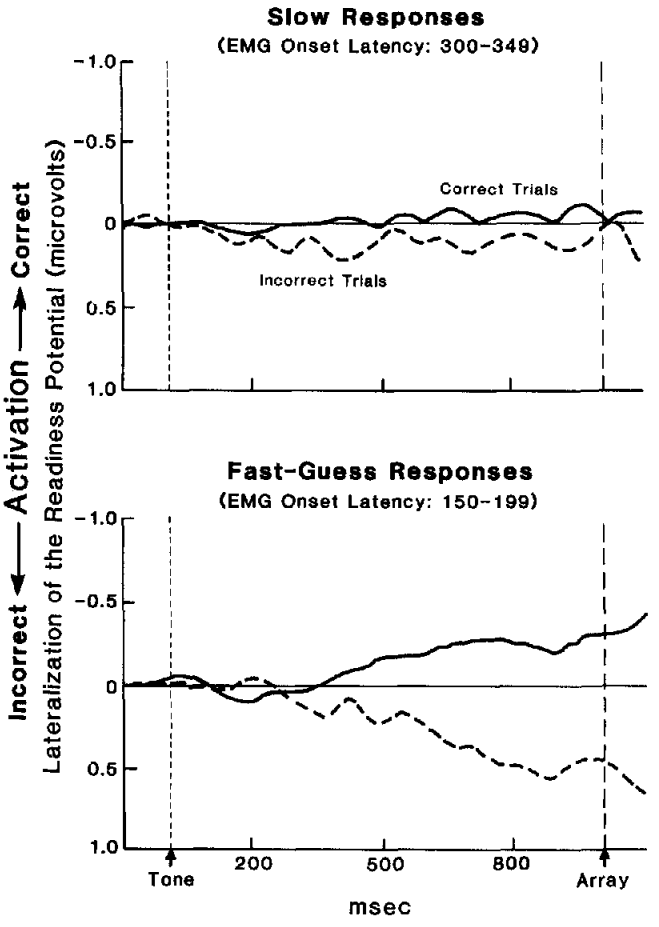

Figure 2. Grand-average event-related brain potential waveforms depicting the voltage difference between the scalp electrodes contralateral and ipsilateral to the correct response for correct (solid) and incorrect (dashed) response trials, from the incompatible condition. (The waveforms in the upper panel are averages for trials with electromyographic [EMG] response latencies between 300 and 349 ms; the waveforms in the lower panel refer to trials with response latencies between 150 and 199 ms. Upward deflections indicate lateralization [larger negativity] toward the scalp electrode contralateral to the correct response; downward deflections indicate lateralization toward the scalp electrode ipsilateral to the correct response [and contralateral to the incorrect response].)

response was equiprobable. When a fast response was emitted on these trials, its accuracy depended on whether the subject happened to have chosen the correct response. This is consistent with our interpretation that these trials are fast guesses. However, the data presented so far are not sufficient to conclude that fast guesses occur when there is a high level of prestimulus response activation. Data bearing upon this issue will be presented later, when we consider a measure of individual response channel activation (the single-sided readiness potential). The presence of variable levels of response activation during the foreperiod and their relation to response accuracy is consistent with the variable baseline hypothesis of response activation. In fact, it appears that the relative activation of response channels in the foreperiod is related to the accuracy of a fast guess.

Poststimulus Activation

In this section, we show how measures of the readiness potential are sensitive to the modulation of response channel activation by stimulus evaluation processes. In particular, these measures support the view that stimulus-related response activation can occur prior to complete stimulus evaluation.

As described above, we computed the difference between the electrical potential recorded from the two lateral electrodes (C3′ and C4′) on each trial. However, we now focus on the poststimulus period from array presentation until the end of the recording epoch 1,000 ms later. Trials were sorted as a function of array compatibility, and average lateralization values were computed. The resulting average waveforms are shown in the middle panel of Figure 4. Note that these waveforms include all trials regardless of response accuracy or latency.

If the response channels are continually influenced by information coming from stimulus evaluation processes, then we would expect that postarray values of laterality would reveal this influence. We argued previously on the basis of the conditional accuracy functions (reproduced in the upper panel of Figure 4) that responses with a latency between 150 and 249 ms appeared to be affected more by the noise letters than by the target letter, suggesting an influence of preliminary phases of the stimulus evaluation process on response channels. In fact, the average lateralization waveform for incompatible noise exhibits a "dip" toward incorrect response activation between 150 and 250 ms poststimulus (see middle panel of Figure 4). This dip corresponds to greater negativity at the scalp site contralateral to the incorrect response. Note

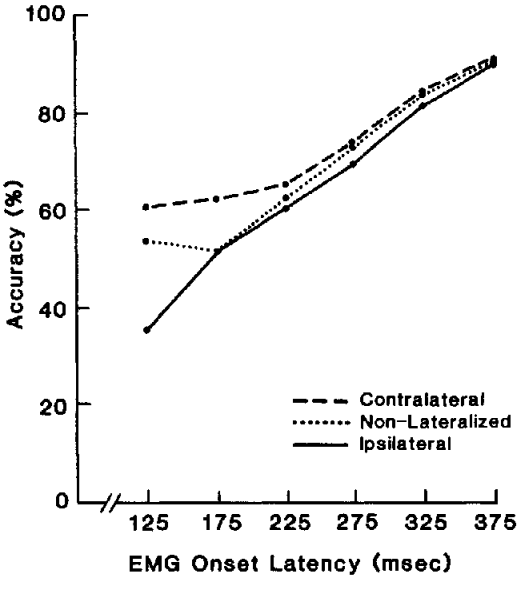

Figure 3. Conditional accuracy functions, averaged over compatible and incompatible conditions, as a function of the lateralization of the readiness potential during the last 100 ms of the foreperiod. (Values of electromyographic [EMG] response latency are shown on the abscissa.)

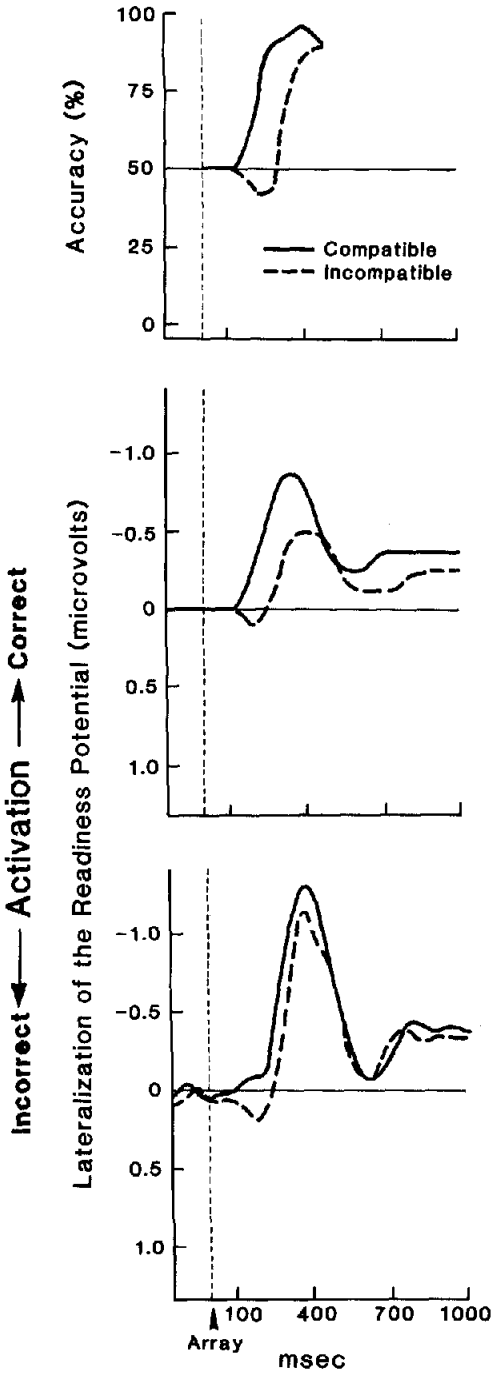

Figure 4. Upper panel: conditional accuracy functions for compatible and incompatible trials. Middle panel: event-related brain potential (ERP) waveforms of the lateralized readiness potential following array presentation, averaged separately for all compatible and for all incompatible trials. Lower panel: ERP waveforms of the lateralized readiness potential following array presentation, for compatible and incompatible correct trials with a response latency between 300 and 349 ms.

that the waveforms shown in the middle panel of Figure 4 correspond very closely to the conditional accuracy functions shown in the upper panel. The correlations computed between the average laterality (sampled at the beginning of each response latency bin) and conditional accuracy values for each of the seven bins were .93 for compatible and .94 for incompatible arrays ($p < .01$). When these correlations were computed on an individual subject basis, the mean value for compatible arrays was .87 (.05 confidence interval = .79–.92) while that for incompatible arrays was .89 (.05 confidence interval = .82–.94).

These data point to a remarkable convergence between two quite different procedures used to gain insights into the influence of the evaluation process on the response channels. One procedure is based on measures of scalp activity and the other on the latency and accuracy of responses.

Although both procedures converge in their description of the time-course of evaluation processes, the accuracy of this description is compromised by the fact that both procedures are based on data aggregated over trials. Because of this, it is not clear whether the functions depicted in the upper and middle panels of Figure 4 represent the nature and time course of the evaluation process on *individual* trials. The figures suggest that information about the array is accumulated gradually and that this information is continuously available to response systems. However, the gradually increasing continuous function that characterizes conditional accuracy curves can be generated by aggregating trials for which the output of stimulus evaluation is, in fact, discrete but variable in latency over trials. Similarly, the description provided by the aggregated laterality data could be the result of averaging trials for which a discrete change in laterality occurred (in the direction of the response that was executed) but for which the change varied in latency. To demonstrate that indeed response activation occurs as stimulus evaluation proceeds, we need to demonstrate the influence of different phases of the stimulus evaluation process on the response system *within* the same trial.

To this end, we partitioned the laterality data as a function of response latency and accuracy. Because we were particularly concerned with finding evidence for an influence of preliminary evaluation processes on the response systems, we focused on the dip in laterality seen in the waveform for incompatible arrays in the middle panel of Figure 4. We argued above that this dip represents early activation of the incorrect response due to the influence of the incompatible noise letters. We reasoned that if we could find evidence for this dip on trials in which the first peripheral response given was correct, then we could infer that, indeed, on these trials at least, preliminary evaluation processes could result in preliminary response activation. Furthermore, the evidence for an influence of evaluation processes on response systems should be most evident for relatively slow trials, because fast trials are influenced by prestimulus activation effects, as we saw earlier. Thus, we focused our analysis on relatively slow, correct response trials only (with a response latency of 300–349 ms). On these trials, by definition, the EMG activity was first observed on the correct side. The lower panel of Figure 4 gives the laterality values for these trials.

The dip can be clearly seen in the waveform for the incompatible arrays while a simultaneous increase in laterality toward the correct response can be seen in the waveform for the compatible arrays. The difference between compatible and incompatible arrays in the area of the waveform between 170 and 210 ms after the array presentation was significant, $t(5) = 2.67$, $p < .05$ (one-tailed), as was the difference between the same area measure for incompatible arrays and zero, $t(5) = -2.78$, $p < .05$ (one-tailed).

Thus, the measure of the lateralized readiness potential suggests that processing of the noise letters can result in preliminary incorrect response activation, even though the correct overt response is ultimately given, presumably as a result of a later occurring analysis of the target letter. This influence is evident for long latency responses for which prestimulus activation effects are minimal.

Response Execution

Grice's variable criterion hypothesis (Grice et al., 1982) leads to the prediction that the level of activation of the response channels at the moment of the response should be lower for fast than slow responses, whereas a fixed criterion hypothesis predicts a constant level, regardless of response latency. Figure 5 shows the average laterality waveforms from 100 ms before the warning stimulus until the time of the response. Waveforms for the compatible incorrect trials are not included because of insufficient data (see above).

To evaluate the merit of the variable criterion hypothesis, we focus on the laterality value at the time an EMG response was observed (indicated by vertical lines in Figure 5).[5] An analysis of variance of these data revealed no significant effects of compatibility, $F(1, 5) = 3.02$, $p > .05$, or response latency (both the main effect of response latency bin and the interaction of response latency bin by response accuracy were not significant, $F[3, 15] = 0.13$, and $F[3, 15] = 0.46$, respectively), but a significant effect of accuracy, $F(1, 5) = 133.39$, $p < .001$. In fact, the mean laterality values were −0.71 and +0.65 microvolts for correct and incorrect responses, respectively. These data indicate, then, that the absolute laterality value at the moment the EMG response is triggered is constant regardless of response latency and that, as expected, the direction of laterality is related to response accuracy.

Individual Response Channel Activation

The measure of the lateralization of the readiness potential we have used so far provides only an index of the *relative* activation of the response channels. This relative measure cannot reveal generalized facilitation of both responses, nor can it distinguish cases in which one response is activated from cases in which the other response is inhibited. A related problem is that, conceptually, it is reasonable to expect the occurrence of a fast guess to be dependent on the absolute level of activation of a response channel. Similarly, the emission of an EMG response may depend on the absolute level of response channel activation.

For these reasons, it would be useful to obtain a *separate* measure of the activation of each response channel. To derive

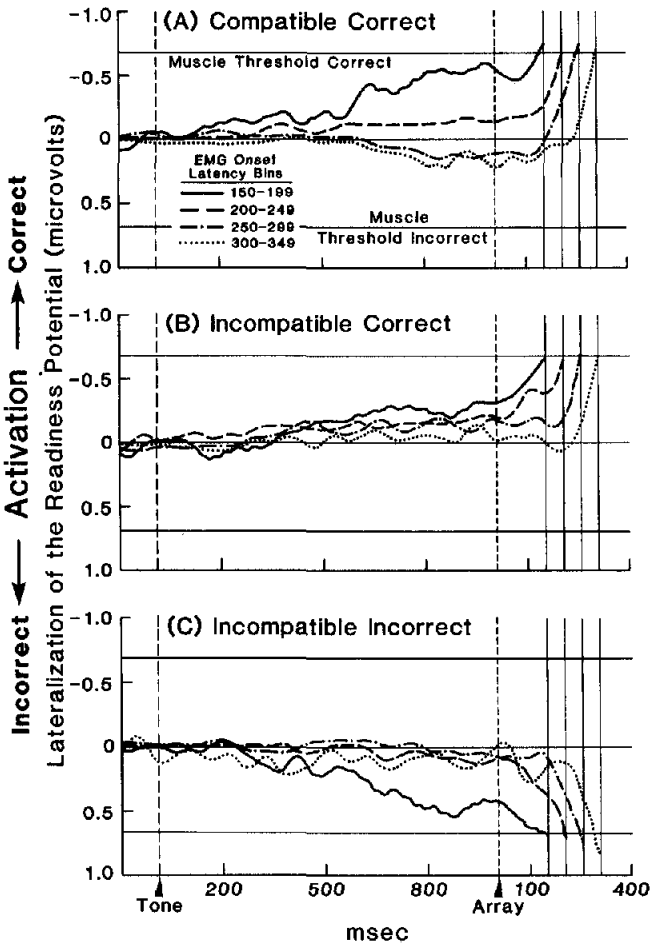

Figure 5. Event-related brain potential waveforms of the lateralized readiness potential for the period beginning 100 ms before the warning tone and ending at the time of the electromyographic (EMG) response to the array. (Separate waveforms are shown for compatible correct [upper panel], incompatible correct [middle panel], and incompatible incorrect trials [lower panel], for four response latency bins. The solid vertical lines in each panel indicate the EMG response latency for each bin. The upper and lower horizontal lines in each panel indicate the inferred thresholds for correct and incorrect muscle response emission.)

such a measure we have to solve the methodological problem of isolating ERP components. In fact, the potential observed at each of the lateral scalp electrodes (C3′ and C4′) is given by the sum of potentials generated locally (i.e., the single-side readiness potential) and of other potentials generated in other regions of the brain and propagated by volume conduction. Therefore, to obtain "pure" measures of the potential associated with the activation of just one response channel, we need to remove the influence of the other potentials. Note that when we measure the lateralization of the readiness potential (i.e., the difference between C3′ and C4′), the influence of

[5] To account for the transmission delay between the cortex and the muscle, we actually sampled the value of the lateralized readiness potential at the *beginning* of each EMG onset latency bin—that is, on the average, 25 ms before the onset of EMG activity.

other brain potentials, not associated with a specific motor response, is eliminated by the subtraction procedure.

We approached the problem of isolating the single-sided readiness potential by using the vector filter procedure (Gratton, Coles, & Donchin, 1987). A description of the procedure and its derivation is given in the Appendix.

Examples of the waveforms obtained with the procedure are shown in Figure 6. The waveforms represent the estimated activation of correct and incorrect response channels throughout the experimental epoch for four response latency bins. Average waveforms were computed separately for each compatibility condition and for correct and incorrect trials. The 8 waveforms for the compatible correct condition shown in Figure 6 are representative of the 32 waveforms available.

Several aspects of this figure are noteworthy. First, during the foreperiod, there is greater activation of the correct side for fast than for slow trials. To determine whether the absolute amplitude of the readiness potential predicts if a fast guess will occur, we computed the probability that a fast response (i.e., a response with a latency shorter than 200 ms) would be emitted as a function of the level of negativity prior to the array at the electrode contralateral to the responding hand. Then we assessed the level of negativity prior to the array by computing the average single-sided readiness potential in the last 100 ms of the foreperiod at the electrode site contralateral to the correct response (when the probability of emitting a correct response was concerned) and at the electrode contralateral to the incorrect response (when the probability of emitting an incorrect response was concerned). We sorted the trials according to their prior negativity into five categories, ordered from a large to a low level of negativity prior to the array. The prediction that a fast response was more likely to occur on trials with a larger negativity prior to the array was confirmed, $F(4, 20) = 2.96$, $p < .05$. The probability ranged from .144 to .108 from the high to the low level of negativity. A post hoc analysis revealed a significant linear trend in probability as a function of prior negativity, $F(1, 20) = 7.36$, $p < .02$. Thus, the probability of a fast response was a function of the level of negativity prior to the array at the electrode contralateral to the responding hand.

A second interesting aspect of Figure 6 is that there appears to be a fixed level of activation of the response channels at the moment the EMG is triggered. Thus, the picture of response activation processes provided by Figure 6 (and by comparable waveforms for the other conditions) confirms the results reported in the previous sections (see Figure 5).

The derivation of single-sided readiness potential measures enabled us to focus on one additional question, that is, the effect of mere stimulus presentation on response channels. One striking common aspect of all the individual response channel functions (those shown in Figure 6 and 24 other comparable waveforms from incompatible and incorrect trials) is that both correct and incorrect response channels show an increase in activation between 50 and 150 ms after the array. In the case of very fast response trials, when the level of activation of a response channel is close to the criterion due to priming in the foreperiod, this increase in activation appears to raise the function above the threshold for the emission of an EMG response.

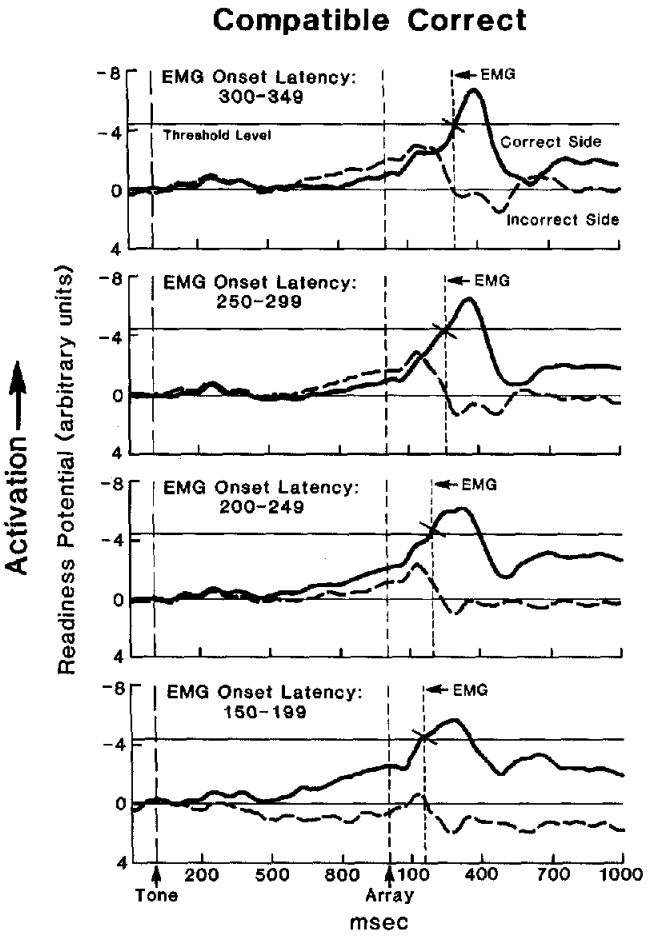

Figure 6. Event-related brain potential waveforms representing the correct (solid) and incorrect (dashed) single-side readiness potentials for compatible correct trials. (Separate average waveforms are shown for four electromyographic [EMG] response latency bins. The dashed vertical lines after array presentation indicate the EMG response latency for each bin. The upper horizontal line in each panel indicate the inferred threshold for muscle response emission.)

Discussion

In this article, we have presented an analysis of response channel activation in a warned, choice RT paradigm, in which the stimulus information was sometimes conflicting. We proposed that activation can be modulated by several mechanisms including those related to prestimulus activation or bias and those related to stimulus evaluation processes.

Measures of the lateralized readiness potential indicate that on some trials, subjects appeared to select and activate particular responses during the foreperiod before the imperative stimulus (array) was presented. In fact, fast responses (with a latency of less than 200 ms) were associated with large and significant lateralization of the readiness potential during the foreperiod. If the lateralization was in the direction associated with the correct response, the response tended to be accurate. If it was in the direction associated with the incorrect response, the response tended to be inaccurate. For these fast responses,

the lateralization of the readiness potential was similar to that observed in simple RT conditions when the subject knows in advance the hand to be used to make a response (cf. Kutas & Donchin, 1980). Thus, we infer that a fast guess was preceded by selection and activation of a response in advance of stimulus presentation. Furthermore, when prestimulus selection and activation occurred, the mere presentation of the stimulus appeared to trigger a response. This "mere presentation" effect may be due to a "response energizing" process, such as that proposed by Grice et al. (1982) and Sanders (1981). Preliminary evidence for such a process is provided by the analysis of single-sided readiness potential measures, which suggests that, regardless of response latency, both correct and incorrect response channels exhibited increased activation following stimulus presentation. However, this increased activation might also be attributed to some stimulus-processing activity, not completely eliminated by our procedure for isolating the single-sided readiness potential (see Appendix).

After stimulus presentation, responses appear to be activated as information about the stimulus becomes available—unless, of course, a fast guess is made. In particular, conditional accuracy functions and lateralized readiness potential values suggest a tendency for incorrect response activation between 150 and 250 ms after the presentation of an incompatible array. Because we were able to demonstrate the presence of this incorrect response activation for correct trials of relatively constant latency (300–349 ms), we inferred that, at least on some trials, stimulus evaluation processes may affect the response systems before the evaluation processes are complete.

Finally, we have shown that responses (EMG activity) occur when response activation achieves a particular fixed level. Analyses of both the lateralized and single-sided readiness potential at the time of response onset indicated that the amplitude of the readiness potential was fixed for all response latencies.

These results have implications for various issues in the study of human information processing. First, they provide support for a "variable-baseline/fixed-criterion" hypothesis of response activation, in contrast to a "fixed-baseline/variable-criterion" hypothesis. Specifically, we have demonstrated that a measure that is intimately related to response processes (the readiness potential) varies in the foreperiod and that this variability is related to the speed and accuracy of responses. We have also shown that the same measure appears to have a fixed value at the time of the response, regardless of response latency. Although the latter observation suggests that a *fixed* level of activation of some central structure determines whether a peripheral response will be activated, it is, of course, possible that *variable* criteria operate at earlier phases of response activation, although our measures of activation provide no evidence about the operation of variable criteria. It should be noted that the portion of ERP waveform that is associated with the moment at which the EMG response is triggered may reflect the execution of the response (a fixed phenomenon) rather than the preparation of the response (a variable phenomenon). However, recent neurophysiological data question the distinction between response execution and preparation. In fact, Requin (1985) showed that there are three classes of neurons in the motor cortex that fire during a warned RT trial. Some fire only during the foreperiod, others fire only during the response, and still others fire during both the foreperiod and the response. Although our scalp recordings cannot distinguish among the activity of these three classes of neurons, we should note that Requin (1985) proposes that the three classes operate as an integrated system. We consider this neurophysiological system as constituting a central portion of the response channel. Thus, response preparation and response execution can be represented by different aspects of the response channel activity.

A second issue addressed by the present experiment concerns the nature of the stimulus evaluation process. The interference determined by irrelevant noise presented visually in close proximity to target information has been studied extensively (for a review, see Johnston & Dark, 1986). In the present study, conditional accuracy and lateralization data converge in suggesting that there are at least two phases in stimulus evaluation. During the first phase (between 150 and 250 ms) the correct response tends to be activated when the noise letters are compatible, and the incorrect response tends to be activated when the noise letters are incompatible. Later the correct response is activated regardless of the nature of the noise letters. As we have noted previously (Coles et al., 1985), the first phase appears to correspond to an analysis of all the letters or features in the array without regard to their location, while the second phase may be related to the association of the letters with their location. This two-phase conception of stimulus evaluation processes is consistent with the feature integration theory of Treisman and Gelade (1980; see also Treisman, Sykes, & Gelade, 1977) and with the "zoom-lens" analogy proposed by Eriksen and Yeh (1985, see also Näätänen, 1982). Our data appear to be consistent with the interpretation that location plays an important role in visual attention (Johnston & Dark, 1986). However, because we did not manipulate information about location, our data cannot serve to determine whether this role is qualitatively different from that of other stimulus dimensions (as proposed by the zoom-lens analogy) or similar to that of other stimulus dimensions (as proposed by the feature-integration theory).

The conditional accuracy functions also reveal that accuracy is between .90 and .95 for incompatible arrays at long response latencies, when accuracy might be expected to approximate unity. This suggests that the stimulus evaluation system sometimes fails to correctly identify the target letter, perhaps because its operation terminates when some degree of certainty about the stimulus is reached or because its performance is sometimes data limited.

A third issue addressed by the present experiment concerns the possibility that stimulus information can affect response-related processes before the evaluation process is completed. Our measure of preliminary response activation (at a central level) showed that incorrect responses can be partially activated on the same trials for which a correct overt response is given later. These data are inconsistent with a single-stage decision model that places the decision stage at an earlier level than that manifested by the readiness potential measures. Furthermore, our previous findings (Coles et al., 1985) are inconsistent with a model that places a single-decision stage

at an earlier level than the peripheral response (EMG or squeeze). In the earlier study, we found that both correct and incorrect peripheral responses could be activated on the same trial. Taken together, these data argue against single-decision stage models (e.g., Sternberg, 1969) because information is apparently transmitted from stimulus evaluation to response systems at at least two moments in time. This is consistent with continuous flow (Eriksen & Schultz, 1979; Grice et al., 1982) or multiple-decision-stage models (Miller, 1982).

In conclusion, we have illustrated how the concept of response channels and the measurement of their activation can provide insights into the mechanisms involved in a warned, choice RT task. Our data suggest that response channels are continuously active and that when this activity crosses a threshold, a peripheral response is emitted. Reaction times measure the latency at which the threshold is crossed and the overt response is emitted. However, they provide little information about the behavior of the response channel activation function in the period preceding the moment at which the overt response is emitted. In our previous work, we used measures of the EMG to provide a second point in the description of the response channel activation function (Coles et al., 1985; Eriksen et al., 1985). In the present experiment, we extended our measurement repertoire to include a *continuous* measure (the lateralization of the readiness potential), and we have illustrated its power in providing insights into various aspects of the human information processing system.

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Appendix

Deviation of Single-Sided Readiness Potential Measures

The potential recorded at each of the two lateral electrodes (C3′ and C4′) can be considered as being given by the sum of the readiness potential generated locally (i.e., in the region of the brain close to the electrode) and of scaled fractions of potentials generated in other parts of the brain and propagated by volume conduction. The other potentials may include the readiness potential generated underneath the contralateral electrode, as well as other ERP components. This can be expressed by the following formulas:

$$V(\mathrm{C3}') = a1 * V(\mathrm{C3}'\mathrm{t}) + a2 * V(\mathrm{C4}'\mathrm{t}) + b1 * \mathrm{V(ERP1)} + \ldots + b\mathrm{n} * V(\mathrm{ERPn}) \quad \text{(A1a)}$$

$$V(\mathrm{C4}') = a1 * V(\mathrm{C4}'\mathrm{t}) + a2 * V(\mathrm{C3}'\mathrm{t}) + b1 * \mathrm{V(ERP1)} + \ldots + b\mathrm{n} * \mathrm{V(ERPn)}, \quad \text{(A1b)}$$

where $V(\mathrm{C3}')$ and $V(\mathrm{C4}')$ are the potentials recorded at C3′ and C4′; $V(\mathrm{C3}'\mathrm{t})$ and $V(\mathrm{C4}'\mathrm{t})$ are the "true" readiness potentials generated underneath C3′ and C4′; V(ERP) are potentials associated with other ERP components that may affect the recordings; and $a1$, $a2$, $b1$, and bn, are scaling factors.

By subtracting A1b from A1a we get A2:

$$V(\mathrm{C3}') - V(\mathrm{C4}') = (a1 - a2) * [V(\mathrm{C3}'\mathrm{t}) - V(\mathrm{C4}'\mathrm{t})]. \quad \text{(A2)}$$

Thus, apart from a scaling factor, the difference between the potentials observed at the lateral electrodes is equal to the difference between the "true" readiness potentials.

Although the derivation of the differential measure is quite straightforward, the separate measurement of the readiness potential on each side is more problematic. In fact, Formulas A1a and A1b can be transformed into A3a and A3b:

$$a1 * V(\mathrm{C3}'\mathrm{t}) = V(\mathrm{C3}') - a2 * V(\mathrm{C4}'\mathrm{t}) - b1 * \mathrm{V(ERP1)} - \ldots - b\mathrm{n} * V(\mathrm{ERPn}) \quad \text{(A3a)}$$

$$a1 * V(\mathrm{C4}'\mathrm{t}) = V(\mathrm{C4}') - a2 * V(\mathrm{C3}'\mathrm{t}) - b1 * \mathrm{V(ERP1)} - \ldots - b\mathrm{n} * V(\mathrm{ERPn}). \quad \text{(A3b)}$$

From A3a and A3b it is evident that in order to obtain the value of the readiness potential on each side, the contribution of components with a significant influence on C3′ and C4′ must be estimated and subtracted from the voltages recorded at C3′ and C4′. Gratton, Coles, and Donchin (1987) have developed a procedure (vector filter) that allows the investigator to separate the contribution of ERP components characterized by different distribution over the scalp electrodes. This technique is based on the development of a series of "spatial filters," each specific for a different component. These filters are given by a series of weights, one for each electrode. The filtered data are represented by linear combinations of the values observed at each electrode. If sets of weights are chosen to be orthogonal, the corresponding filtered components will be orthogonal (although not necessarily uncorrelated). The vector filter procedure can result in a reduction of the contribution of overlapping components, although it cannot guarantee their complete elimination. Examples of the gains in component identification and measurement obtained by applying the vector filter procedure are given in Gratton, Kramer, Coles, and Donchin (1987) and in Fabiani, Gratton, Karis, and Donchin (1987).

In the analysis of the data of the present experiment, we chose sets of weights according to the following criteria. First, they should suppress the contamination due to the contralateral readiness potential. For this reason, the two sets of weights used for estimating independently the readiness potential on each side were selected so as to be orthogonal (i.e., their cross-product was equal to 0). Second, they should be such as to suppress the influence of other ERP components. In particular, we filtered out the contribution of components with an exclusively midline distribution. This was accomplished by giving equal weights to the midline electrodes (Fz, Cz, and Pz). Third, to eliminate general components (with an equivalent influence at all sites), the sum of each set of weights was equal to 0.

The vector filters used were the following:

$$\mathrm{C3}'\mathrm{t} = 0.888 * \mathrm{C3}' - 0.114 * \mathrm{C4}' - 0.258 * \mathrm{Fz} - 0.258 * \mathrm{Cz} - 0.258 * \mathrm{Pz} \quad \text{(A4a)}$$

$$C4't = 0.888 * C4' - 0.114 * C3' - 0.258 * Fz - 0.258 * Cz - 0.258 * Pz. \quad (A4b)$$

Note that the sum of squares of each set of weights is equal to 1, so that the vector filter amounts to a rotation of axes in the space defined by the electrode locations.

To provide some indication of the validity of this procedure, we computed the average correlation across trials, over the 6 subjects, between C3′ and C4′ (in the last 100 ms before array presentation) before and after applying vector filter. As can be seen by comparing Equations A3a and A3b, the "undesired" components (i.e., the contralateral readiness potential, as well as the other ERP components) will produce similar effects on C3′ and C4′ and will therefore tend to increase the correlation between the potential recorded at these two electrodes. The average correlation was .83 ± .03 before correction and .00 ± .08 after correction. Note that the latter correlation could have been different from 0 even though orthogonal sets of weights were used. This would occur if the procedure under- or overestimated the effects of volume conduction or if there was some "structural" or "functional" association between the brain electrical activity generated beneath the two lateral electrodes. The latter might occur under conditions of "response competition" or "response energizing."

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