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A brain potential manifestation of error-related processing*

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Many theories of human cognition assume that mechanisms exist to oversee the operation of the information processing system. The concept of an information processing overseer is central to theories of executive or supervisory cognitive control systems (e.g. Logan 1985; Stuss and Benson 1986; Shallice 1988). In particular, many theories incorporate the notion that systems exist that are dedicated to monitoring action and compensating for errors, including theories of action (MacKay 1987), learning (Adams 1971; Rumelhart et al. 1986), speaking (Levelt 1989), and consciousness (Kosslyn and Koenig 1992). Indeed, the fallibility of the information processing system (see Norman 1981, 1988; Reason 1984, 1987, 1990) suggests that error-monitoring and compensation mechanisms must be integral parts of human thought and action. Nonetheless, although the existence of error-monitoring systems has been inferred from their presumed behavioral consequences, very few direct observations have revealed the activity of neural systems that appear specifically activated during the commission of errors; whether the brain actually implements these overseeing functions therefore remains open to question.

The existence of error-related processing may be established either by (1) demonstrating the existence of processes that operate only in response to errors, or (2) demonstrating the existence of processes that, although they operate all the time, produce a representation or action on error trials that is different from the representation or action produced on correct trials. In the experimental psychology and motor control literatures, studies of error detection and compensation[1] have proceeded on the assumption that behavior that minimizes or corrects for errors arises from error-related processing. For example, a variety of studies in the choice reaction time (RT) literature (e.g. Rabbitt 1966, 1967, 1968; Rabbitt, Cumming, and Vyas 1978; Rabbitt and Vyas 1981) examined the characteristics of the correct responses that often follow errors assuming that the responses were corrections resulting from processing of the error. It is clear, however, that this need not be the case: for example, few papers consider the possibility that these 'error-correcting responses' are the result of normal processing activity that occurs in parallel with error-producing activity but that culminates more slowly in an overt response. Another set of studies arising from the motor control literature has made a similar assumption regarding corrections of directional errors in step-tracking performance (cf.

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Gibbs 1965; Angel and Higgins 1969; Higgins and Angel 1970; Megaw 1972; Angel 1976; Cooke and Diggles 1984), and similar criticisms can be leveled against these studies.

Other forms of putative compensatory behavior need not arise from processing that is specifically error-related. For example, erroneous responses that are less forceful than correct responses and thus appear to be inhibited (Rabbitt 1978) might simply be less activated, perhaps because less stimulus information gives rise to them than to the correct responses. Moreover, normal information processing may have properties that minimize erroneous response tendencies (cf. Coles et al. 1985; Dell 1986). That is, producing the correct response might itself be sufficient to minimize the erroneous response tendency through response competition (Coles et al. 1985), spreading activation within a representational network (Dell 1986; Cohen et al. 1992) or some other means. Such mechanisms could explain 'partial' errors (e.g. Coles et al. 1985), observed when electromyographic (EMG) or Event-Related Brain Potential (ERP) evidence suggests erroneous response activity even when no overt error is observed. Finally, the slowing observed on trials after an error trial (Rabbitt 1966; Laming 1968) could result from a continuance of the processing difficulty that caused the error, rather than from a strategic slowing to avoid additional errors.

As a result of this ambiguity, few studies have offered direct evidence for error processing. One exception is the EMG data of Gordon and Ghez (1987), which showed increased antagonist muscle activity in association with erroneous agonist activity, but some uncertainty remains regarding the origin of the increased activity, in that it could have been pre-programmed in order to perform the task used in that paper. Another exception is the observation that the P300 elicited in response to errors predicts the likelihood of errors on a subsequent trial of the same type in a choice RT experiment (Donchin et al. 1988): larger error-trial P300s were associated with subsequent correct responses than with subsequent errors. Thus, in each case, the investigators demonstrated an *increase* in the activity of some physiological variable uniquely associated with the modulation of the erroneous behavior.

In this chapter, we present data supporting the existence of brain mechanisms involved in detecting and compensating for errors. We (Gehring et al. 1990; Gehring et al. 1993) and Falkenstein's group at Dortmund (Falkenstein et al. 1990, Falkenstein et al., this volume) have independently described a component of the human ERP that appears on those trials in choice RT tasks on which subjects execute the wrong response. These error trials are characterized by a relatively sharp ERP component occurring at about the moment the error is executed. In this chapter, we present data from several experiments that point to the ubiquity of this phenomenon and suggest a possible functional role for the processing it represents. We explore the possibility that the ERN described by Gehring et al. (1990) manifests the activity of a neural system involved in monitoring responses and compensating for errors when they occur.

Appearance of the ERN in existing choice-RT data sets

In this section we present our initial findings regarding the component and some analyses of existing data sets that elucidate some of the characteristics of the component. The data are drawn from experiments conducted at the Cognitive Psychophysiology Laboratory at the University of Illinois.

Sentence-verification task

We first observed the component in a study in which 16 subjects performed a categorization task (Gehring et al. in preparation). On each trial in this task, 2 words appeared on a visual display, one above the other. Subjects responded 'yes' or 'no' by squeezing 1 of 2 zero-displacement dynamometers (see Kutas and Donchin 1977) to indicate whether the upper word on the display referred to an exemplar of the category defined by the lower word. Thus, if 'robin' appeared above 'bird,' the subject responded 'yes' with one hand; if 'spoon' appeared above 'bird' the subject responded 'no' with the other hand. Figure 1 shows the ERPs elicited in this task. The *response-locked* ERPs (panel b) are synchronized according to the re-

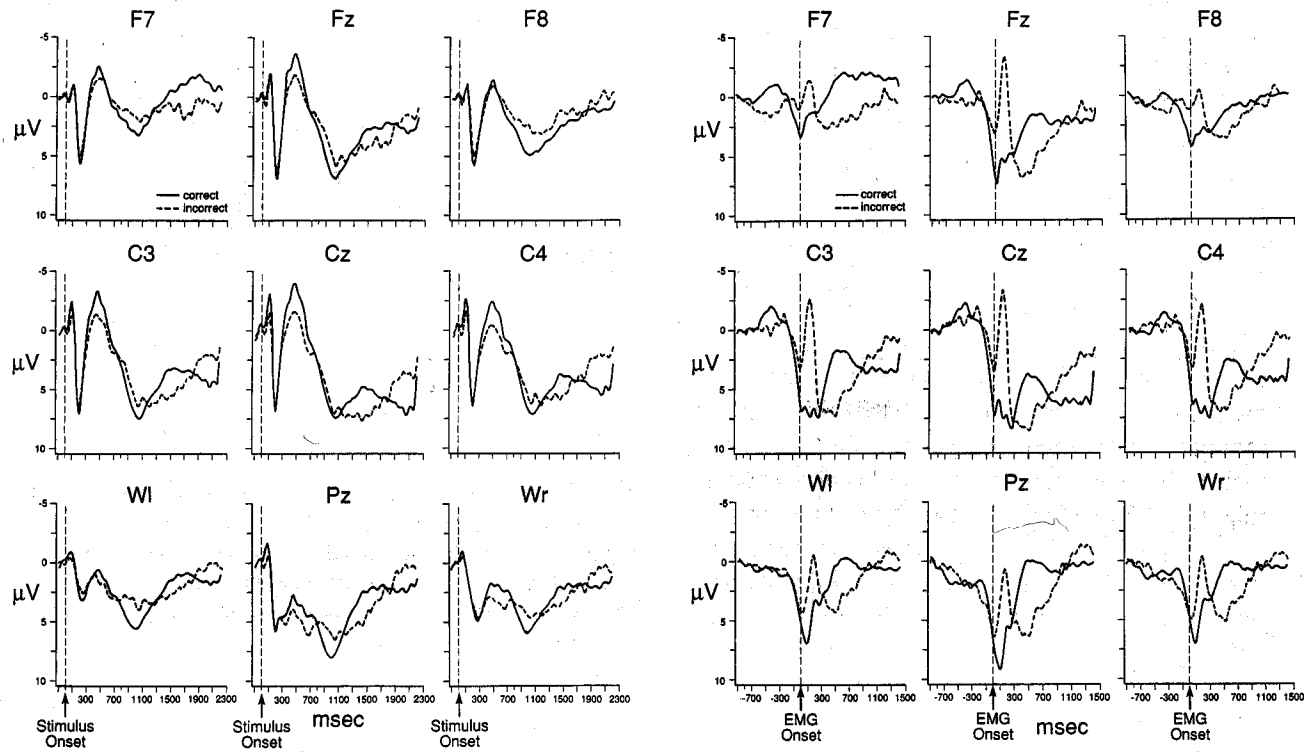

Fig. 1. Grand average ERP waveforms from a category verification task (Gehring et al. in preparation). The solid line represents trials on which the response was correct; dashed lines represent error trials. In *A*), the waveforms are time-locked to the stimulus. In *B*), the waveforms are time-locked to the response, in this case, the onset of the squeeze response.

sponse on each trial, in this case according to first detectable squeeze activity. The component begins approximately at the moment of the onset of squeeze activity and peaks approximately 100 msec later.[2]

We submitted the data from the categorization task to a 9 (electrode site) × 2 (response accuracy) repeated-measures analysis of variance. An electrode site × accuracy interaction ($F(8, 120) = 24.08$, $p < .0001$, $MSe = 6129.6$) and subsequent comparisons revealed that the ERP waveforms for correct and incorrect trials differed at all electrode sites except F7 and F8 (e.g. Fz: $F(1, 15) = 17.23$, $p < .001$, $MSe = 1160.7$. Cz: $F(1, 15) = 39.87$, $p < .0005$, $MSe = 1180.63$. Pz: $F(1, 15) = 51.97$, $p < .0005$, $MSe = 809.28$). *Post hoc* comparisons revealed that the effect of accuracy was larger at the Cz than at the Fz electrode, $F(1, 15) = 14.45$, $p < .0025$, $MSe = 197.5$, but did not differ significantly at the Fz and Pz electrodes, $F(1, 15) = 6.0$, $p < .05$, $MSe = 337.9$ (with Bonferroni correction).

As a first step in characterizing this ERP component, we sought to determine whether it is larger at scalp sites contralateral either to the incorrect response or to the correct response. Such a scalp distribution might indicate that the component is motoric, possibly related to producing or inhibiting one of the responses. Figure 2 shows the component elicited in the same set of subjects during a task that required them to respond according to the central letter of a 5-letter array (see the discussion of Gehring et al. 1992, below, for a description of this task). The data from the letter discrimination task were collected using 2 lateral scalp electrodes. We classified the lateral electrodes (C3' and C4', 4 cm to the left and right of Cz) according to their relationship to the hand of response (contralateral or ipsilateral). Figure 3 shows the averages (from error trials only) computed according to this electrode classification. Note the difference between the waveforms prior to the response, corresponding to the lateralized readiness

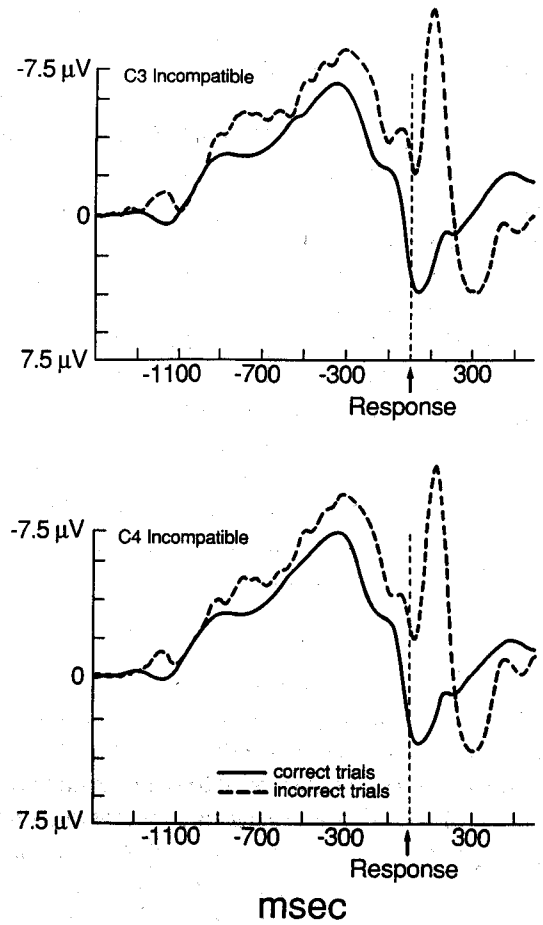

Fig. 2. Grand average response-locked ERP waveforms from a letter discrimination task performed by the same subjects as in Fig. 1. The waveforms are from trials in which flanking noise letters corresponded to the erroneous response. Solid lines represent correct response trials; dashed lines represent error trials.

potential (cf. Coles 1989): the electrode contralateral to the error response shows greater readiness potential activity than the other electrode. We submitted the data from the letter discrimination task to a 2 (electrode site: contralateral *vs.* ipsilateral) × 2 (response accuracy) repeated measures ANOVA. A main effect of accuracy ($F(1, 15) = 11.87, p < .005, MSe = 8313.91$) again confirmed the existence of the component, but the lack of a significant site × accuracy interaction ($F(1, 15) = .75, p > .10, MSe = 352.61$) indicates that the difference between correct and incorrect trials was the same for both electrode sites.[3]

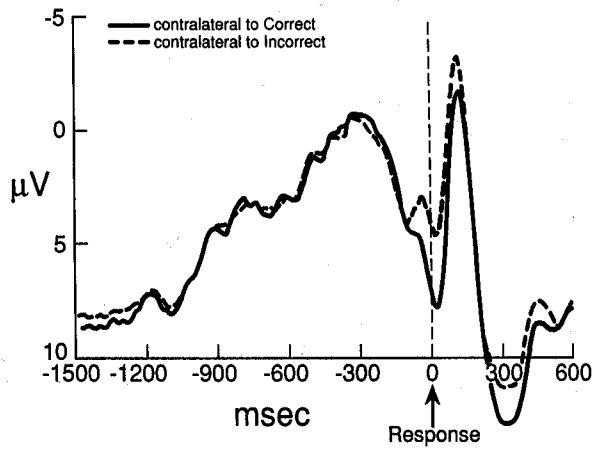

Fig. 3. Grand average response-locked ERP waveforms from error trials, recorded from C3' and C4', but averaged according to the relationship between the electrode and the responding hand. The solid line represents the electrode contralateral to the correct response. The dashed line represents the activity at the electrode contralateral to the error response.

Sternberg memory search task

To confirm the existence of the component and to rule out possible artifacts, we turned to data from a group of different subjects who had performed a Sternberg memory search task, using a button press response (Strayer et al. in preparation). Seven young (mean age = 20.6) and 7 elderly (mean age = 71.7) subjects performed this task under consistent and varied mapping conditions (cf. Schneider and Shiffrin 1977). To collect the data, different amplifiers and software were used. Figure 4 shows the average ERP from the consistent-mapping condition with a memory-set size of 2 letters for the young and elderly subjects. As in the study discussed above, the component appears in response-locked averages at about the moment of the overt response, and peaks approximately 100 msec later.

We performed three separate analyses:

1. Given the report of Gemba et al. (1986) that error-related cingulate cortex activity in monkeys appeared only in intermediate phases of learning, one might expect the amplitude of the component to vary with practice on the task. First, for the young subjects only, we submitted the ERP component measure[4] taken at the Cz electrode to a 2 (before and after practice) × 2 (memory-set size 2 or 4) × 2 (consistent or varied mapping) × 2 (target or non-

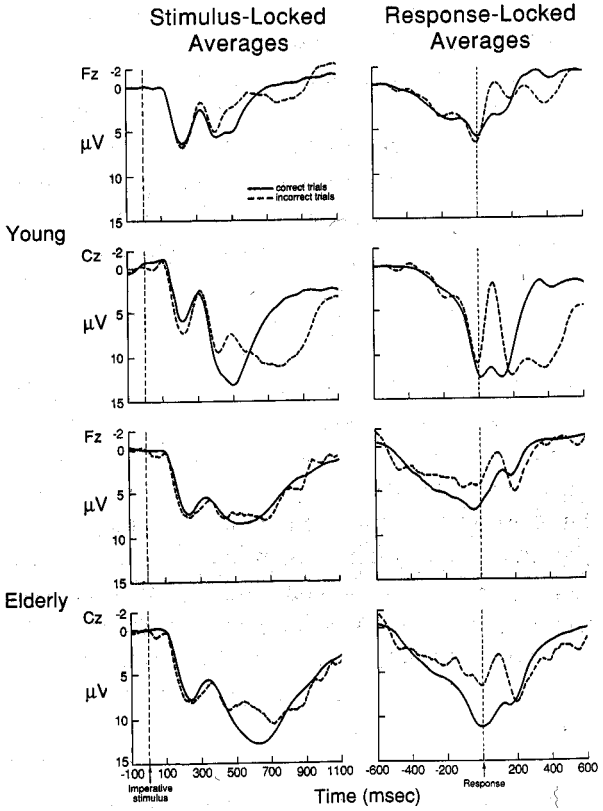

Fig. 4. Grand average ERP waveforms from a Sternberg memory search task.

target trial) × 2 (response accuracy) repeated-measures ANOVA. A significant main effect of response accuracy ($F(1, 6) = 10.61, p < .025, MSe = 39998.1$) confirmed the effect observed in the other study. However, the effect of response accuracy did not interact with practice or any of the other independent variables.

2. One might also expect error-related activity to vary with the latency of the error response, with slow errors possibly arising for different reasons than fast errors (cf. Yellott 1971). Restricting the analysis to young subjects, we divided the error RT distribution for each subject into quartiles, and computed separate ERP averages for each quartile. Figure 5 shows these average ERPs. We submitted measures of error-related negativity (ERN) amplitude to a 4 (RT quartile) repeated-measures ANOVA. A significant main effect ($F(3, 18) = 4.08$, $p < .05$, $MSe = 2340.9$) and subsequent *post hoc* comparison indicated that the slowest responses were associated with a smaller ERN than the 3 faster quartiles, $F(1, 6) = 19.29$, $p < .005$, $MSe = 5825.91$. Although it is difficult to determine the extent to which variability in the latency of the component on these trials contributed to the effect, the analysis suggests that the slower errors evoked less error-related processing than the fast errors.

3. Finally, we sought to explore the effects of aging on the ERN. We restricted the analysis to session 1, consistent mapping set size 2 target trials. We submitted measures of ERN amplitude to a 2 (group: young or elderly) × 3 (electrode site: Fz, Cz, or Pz) × 2 (response accuracy) repeated measures ANOVA. A significant group × accuracy interaction ($F(1, 12) = 5.17$, $p < .05$, $MSe = 6770.6$) and subsequent comparisons revealed that the difference between correct and incorrect trials was larger for the young than for the elderly subjects.

Eriksen noise-compatibility task

Thus far, the data suggest the existence of a component that is concurrent with error responses in choice RT tasks. We turn now to data (from Gehring et al. 1992) that allow us to examine the relationship between the amplitude of this component and the force with which the error was executed.

In Gehring et al. (1992), subjects were required to indicate by squeezing 1 of 2 dynamometers whether the central letter of a 5-letter stimulus

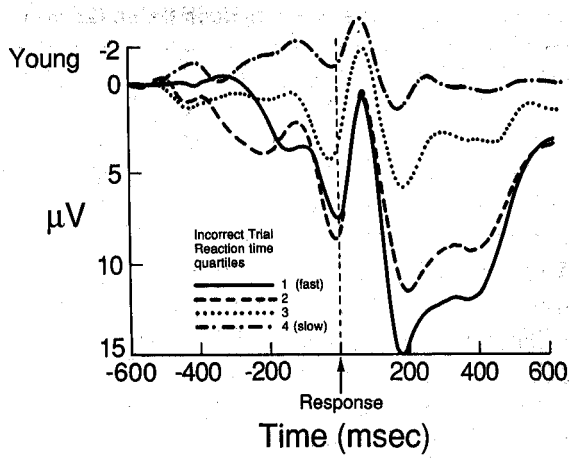

Fig. 5. Grand average ERP waveforms at Cz from error trials from the task in Fig. 4. The 4 lines represent error trials drawn from 4 different quartiles of the error RT distribution (fast to slow).

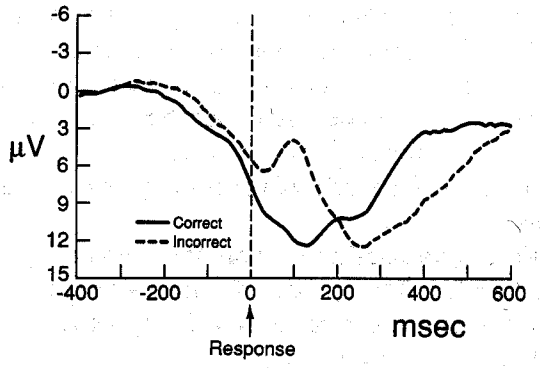

Fig. 6. Grand average ERP waveforms recorded at the C3 electrode in the letter discrimination task (from Gehring et al. 1992).

array was an H or an S. Four noise letters (H or S) surrounded the central letter. Four stimulus arrays were thus possible: HHHHH, SSSSS, HHSHH, SSHSS (cf. Eriksen and Eriksen 1974). In some conditions, a warning stimulus 1 sec before the array indicated with probability .8 what the central letter would be. In other conditions, a neutral, non-informative warning stimulus appeared. (For other details, see Gehring et al. 1992).

Figure 6 compares correct and incorrect trial ERP waveforms from this experiment. Although the ERN does appear in this task, it is smaller than in the other tasks above. We submitted the ERN amplitude measures to a 2 (neutral *vs.* informative precue) × 3 (electrode site) × 2 (accuracy) repeated-measures ANOVA. A main effect of accuracy ($F(1, 7) = 22.79$, $p < .005$, $MSe = 19006.0$), a precue × accuracy interaction ($F(1, 7) = 17.27$, $p < .005$, $MSe = 287.61$), and subsequent comparisons suggested that the component did indeed appear in this experiment, and that the difference between correct and incorrect trials was larger in the neutral precue conditions ($F(1, 7) = 17.27$, $p < .005$, $MSe = 287.61$ – not visible in the figure).

The squeeze responses in this task allowed us to analyze the relationship between the amplitude of the component and the *extent* of error activity. Here, we defined the accuracy of responses according to the first detectable EMG activity. We defined large errors to be those on which the amplitude of the erroneous *squeeze* response fell above the median error squeeze amplitude for that subject. Small errors were those below the median. Figure 7 shows the average waveforms at the Cz electrode site for small and large error responses. Measures of ERN amplitude at the Cz electrode were submitted to a 2 (neutral *vs.* informative precue) × 2 (small *vs.* large squeeze) repeated-measures ANOVA. A significant main effect of squeeze amplitude ($F(1, 7) = 14.87$, $p < .01$, $MSe = 207.1$) suggests that the component was larger on trials where the error activity was small than where the error was large.

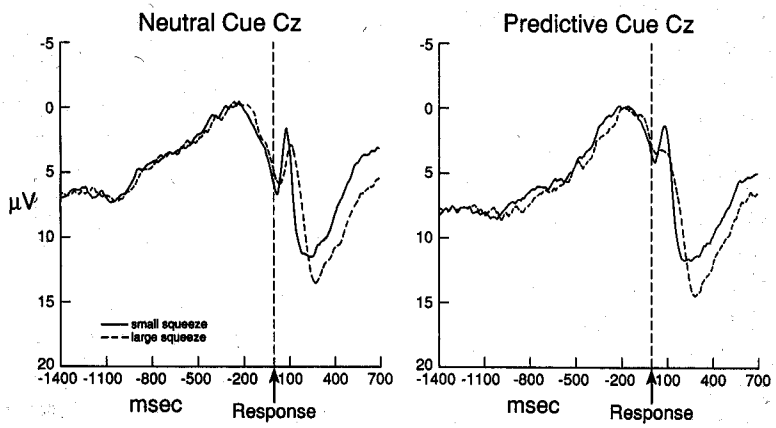

Fig. 7. Grand average response-locked error trial ERP waveforms from Cz from the Gehring et al. (1992) data. The solid line represents errors in which the squeeze fell below the median squeeze force level ('small squeezes'), whereas the dashed line represents errors falling above that median ('large squeezes'). 'Neutral' indicates trials in which the precue was non-informative, 'Predictive' indicates trials in which a precue predicted which imperative stimulus would appear on the trial (see Gehring et al. 1992, for details).

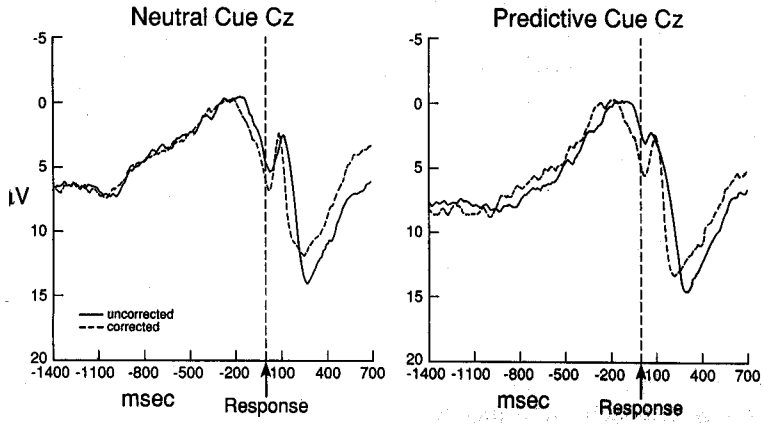

Fig. 8. Grand average response-locked error trial ERP waveforms from Cz from the Gehring et al. (1992) data. The solid line represents errors that were not corrected, the dashed line represents errors that were corrected. 'Neutral' and 'Predictive' retain the same meaning as in Fig. 7.

Next, we attempted to determine whether the component is associated with error-correction activity. We defined corrected errors to be those on which the initial incorrect EMG activity was followed by a correct EMG response. Figure 8 compares the waveforms on corrected and uncorrected trials. We submitted the ERN amplitude measures to a 2 (neutral *vs.* informative precue) × 2 (uncorrected *vs.* corrected) repeated-measures ANOVA. The negativity was larger on trials where the error was corrected than where the error was not corrected, $F(1, 7) = 10.38, p < .05, MSe = 291.0$.[5]

Summary

The analyses reviewed thus far suggest that a negative-going ERP component is evident at about the moment of the response on error trials or shortly thereafter. In these experiments, the component appears to be:

1. Largest at central and frontal electrode sites.
2. Not lateralized according to the side of the error response.
3. Smaller for slow errors.
4. Smaller in elderly subjects.
5. Larger when error responses are small.
6. Larger when errors are corrected.

In addition, across experiments, the component appears to be larger when subjects are performing slowly and more accurately. In particular, the data of Fig. 2 are from the same task as those in Fig. 6, yet the component in Fig. 2 is much larger. The only difference between these studies (except for the subjects involved) was that the subjects performed much more accurately (error rates less than .05) in Fig. 2 than in Fig. 6 (error rates greater than .20).

The speed–accuracy experiment

The data reviewed thus far suggest that the component behaves much as one would expect a manifestation of error-related processing to behave: it occurs at about the same time as the error, it is related to measures of error compensation, and it appears to be larger in experiments where accuracy levels were high. With these data in mind, we undertook an additional experiment.

In this experiment (reported in Gehring et al. 1993), subjects performed the Eriksen noise-compatibility task with a non-informative warning stimulus similar to the one used by Gehring et al. (1992). Three different conditions were used that varied in the degree to which fast or accurate performance was encouraged. In one condition, a bonus system induced subjects to respond quickly, with relatively little regard for accuracy. In another condition, the payoffs induced subjects to respond relatively slowly and accurately. A third condition was intermediate in accuracy emphasis. We manipulated speed–accuracy emphasis based on the assumption that the information-processing system would be more likely to invoke error-related processing, or that error-related processing would be

enhanced, if there were a demand for high accuracy. Within each accuracy condition, stimuli were presented that contained noise letters whose identity sometimes called for the erroneous response.

In addition to evaluating the effects of these manipulations, we performed several analyses to investigate the possible detection of and compensation for errors and the relationship of such behavior to the brain activity manifested by the ERN. Subjects responded using dynamometers, so that we could compare the relative force of errors and correct responses. In addition, we measured the EMG from the muscle that executed the squeeze, in order to detect errors that are initiated but not completed (cf. Coles et al. 1985). We predicted that, if the ERN indeed manifests the activity of an error-detection and compensation system, then its amplitude should be greater when error-detection and compensation occurs. (For additional details, see Gehring et al. 1993).

The performance data indicated that subjects responded more slowly and accurately when accuracy was emphasized: Correct EMG onset latency and accuracy (percent correct) values for the 3 conditions were: Speed: 136.9 msec, 67.6%; Neutral, 271.8 msec, 78.8%; Accuracy: 304.4 msec, 89.4% (see Gehring et al. in press). Because of the performance differences between the 3 speed conditions, we restricted the analyses to all trials with responses falling within a single 50 msec epoch. We chose the 50 msec bin for each subject that yielded the most responses across the 3 conditions. This restricted analysis ensured that the effects on the various measures do not result simply from different RTs contributing to the mean values observed for the measure. Note, however, that it is not possible to equate both the absolute RT epoch (e.g. 250–300 msec) and the part of the distribution in which the RTs fall (e.g. the first quartile) between the 3 conditions.

The ERP data confirmed the existence of the ERN in this task. Figure 9 shows the effect of the speed–accuracy manipulation on ERN amplitude. The amplitude of the ERN increases with increasing emphasis on response accuracy. We derived a measure of ERN amplitude based on stepwise discriminant analysis, using the posterior probability that a trial was classified as an error trial as the

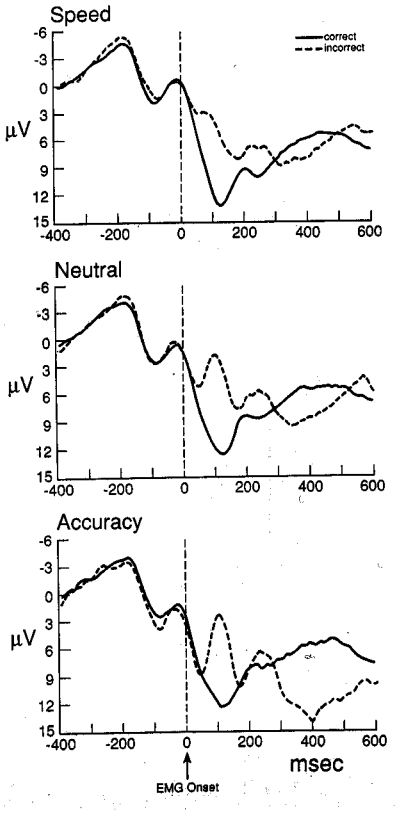

Fig. 9. The effects of different speed-accuracy instructions on the ERN recorded at Cz. For each of the 3 conditions, trials with the same RT were used. (From Gehring et al. 1993, reprinted with the permission of Cambridge University Press.)

measure of ERN amplitude (see Gehring et al. 1993 for further details). We partitioned the data according to 4 quartiles of ERN amplitude from that analysis. Figure 10 (left panel) shows the ERPs at Cz averaged from each of those quartiles; it suggests that the measure based on discriminant function analysis is sensitive to the characteristic morphology evident in the figures from the preceding sections. We found that the amplitude of the ERN measured in this manner was related to 3 different measures of error compensation activity: First, the larger the ERN on a trial the smaller the associated error response (Fig. 10, right panel, top). Second, larger ERNs were associated with a greater probability that the error would be corrected (Fig. 10, right panel, middle). Finally, when the ERN was large, correct responses on the trial following the error trial were slower than when the ERN was small (Fig. 10, right panel, bottom).

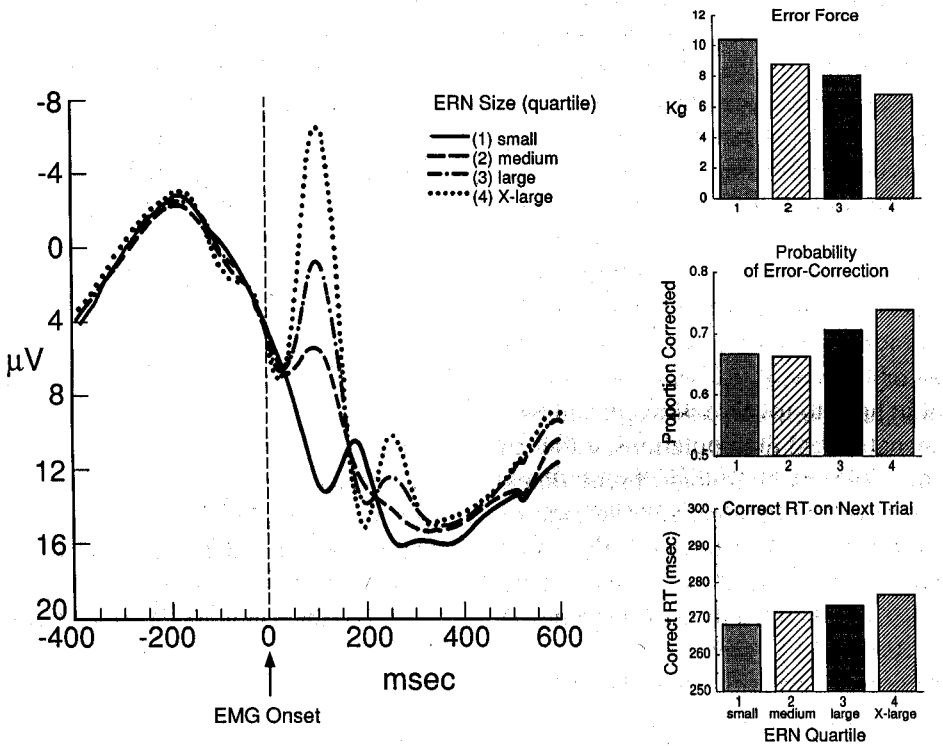

Fig. 10. Left panel: Average ERPs at the Cz electrode as a function of the 4 levels of the posterior probability measure of ERN amplitude. Right panel, top: Error squeeze force in Kg as a function of the 4 levels of ERN. Right panel, middle: Probability of error correction as a function of the 4 levels of ERN. Right panel, bottom: Correct RT on the trial following an error as a function of the 4 levels of ERN. (From Gehring et al. 1993, reprinted with the permission of Cambridge University Press.)

Taken together, this pattern of data suggests that the ERN behaves much as one would expect if it were a manifestation of some part of a system involved in error detection and compensation.

Discussion

The data presented in this paper demonstrate the existence of a processing entity in the brain whose activity is greater when erroneous response activity is present than when it is not. This activity appears at about the same time as the error itself, beginning at about the moment of error response onset and peaking 100 msec later. Error-related processing appears to be consistently activated in choice RT tasks, being evident in a variety of different paradigms.

Other aspects of the data reviewed above point to a functional role for the error-related processing. The relationship between the ERN and the relative emphasis on accuracy *vs.* speed (both between and within a single task) suggests that the process manifested by the ERN is either more active or more likely to be active when accuracy is important to the subject. Other findings point to a relationship between the ERN and compensatory behavior. Specifically, the relationship between the amplitude of the ERN and the magnitude of the error response (in the data from Gehring et al. 1992, and Gehring et al. 1993) point to a possible relationship between the process manifested by the ERN and inhibition of the error response: large amplitude ERNs would be associated with inhibited error responses under this scenario. Similarly, the relationship between the ERN and the correction of the error (on the error trial) and slowing down (on subsequent trials) might point to a role for the process manifested by the ERN in producing these compensatory behaviors. Of course, the

process manifested by the ERN might be shared by systems involved in producing these separate compensatory behaviors, or the process might simply share common inputs, outputs, or some other resource with these compensatory systems. In any case, the observed relationships suggest that the ERN taps into the activity of the system at some level.

A number of issues remain unresolved, however. One is whether there is a causal relationship between the process manifested by the ERN and the compensatory behavior. Research addressing this issue would have to involve neurophysiological and neuroanatomical manipulations, either in animals or through human neurological syndromes. A second unresolved issue concerns the type of input information used by the process manifested by the ERN: it is not clear what sort of error would fail to produce an ERN, and thus no constraints on its production are evident. Another unresolved issue concerns the precise computational role of the process: the data discussed in this paper do not specify which computations related to detecting, inhibiting, or correcting the error are manifested by the ERN. A closely related question concerns the generality of the process. Most accounts of error detection and compensation have concentrated on a single domain (e.g. choice RT or speech production), but it is possible that the ERN manifests the operation of a more general system that operates when errors occur in a variety of response modalities.

A number of neurophysiological findings suggest loci within the brain that might have a role in producing the ERN and provide a starting point for those wishing to investigate the neural origins of the ERN. Brooks and his colleagues (Brooks 1986; Gemba et al. 1986), examining transcortical field potentials recorded from the brains of behaving monkeys implanted with chronic electrodes, reported a phenomenon that may provide an animal model of the ERN discussed in the present paper. The task required monkeys to extend their wrist while a lamp was lit (for 900 msec). The monkeys received no reward if the response occurred after the 900 msec epoch; errors were responses falling outside the 900 msec window. Gemba et al. (1986) examined the transcortical field potentials recorded from the anterior cingulate cortex (area 24) of monkeys performing this task. The recordings involved 2 electrodes, 1 situated on the mesial cingulate surface and the other 2 mm deeper into the cingulate tissue. For early training sessions, no observable difference between correct and incorrect trials appeared in the post response epoch. Once the monkeys began to respond appropriately 45% of the time, however, a potential appeared on error trials as a positive deflection in the surface-depth difference waveform, beginning about 50 msec following the erroneous response. Thus, the polarity of the potential was such that the deflection appeared more positive at the cortical surface electrode than deeper in the cortex. By the session in which 70% of the responses were appropriate, the deflection disappeared from the waveform.[6]

Sasaki and Gemba (1986) reported the existence of field potentials arising in the prefrontal cortex of monkeys on No-go trials in a Go/No-go RT task. These potentials were not evident on corresponding Go trials. In a later study (Sasaki et al. 1989), the prefrontal areas producing the No-go potential were electrically stimulated on Go trials. Stimulation suppressed or delayed the Go movement. ERP recordings from humans performing a similar task (Gemba and Sasaki 1989) showed a negative potential occurring on No-go trials that was maximal at Fz and Cz. Indeed, its morphology appears similar to the ERN, although the authors did not examine its timing to erroneous responses (No-go errors). Thus, to the extent that processing a No-go stimulus shares characteristics with the processing of an erroneous response, the No-go potential can be considered related to the ERN.

Taken together, these field potential recordings provide evidence for neurophysiological mechanisms whose activity is analogous to the activity exhibited by the ERN: the relationship of the ERN to the presence of erroneous activity is similar to the anterior cingulate activity observed by Gemba et al. (1986), and the possible relationship of the ERN to response suppression is like the activity of the No-go potential of Sasaki and Gemba (1986). The presence of frontal lobe structures involved in activity related to error detection and compen-

sation would be consistent with current theories of frontal lobe function (e.g. Goldberg 1985; Stuss and Benson 1986; Shallice 1988), particularly with regard to the functions of the prefrontal cortex. These theories – indeed, most theories – of frontal lobe function ascribe 'executive' functions to the frontal lobes, that is, functions involved in preparing and regulating information processing and action. A fundamental part of this activity is error detection and compensation (cf. Luria 1966).

Notes

1. Here we use the term 'compensation' to include processes involved in inhibiting the error, processes involved in correcting the error by immediately executing the designated correct response, and processes that operate to minimize the likelihood of future errors, e.g. by correcting faulty biases and strategies.
2. To quantify the component in these and the other tasks discussed in this article, the data were digitally filtered with a 59-point, equal-ripple, zero-phase-shift, optimal finite impulse response low pass filter with a passband cutoff frequency of 8 Hz and a stopband cutoff frequency of 10 Hz (see Farwell et al. 1993). Then we computed the mean amplitude of the ERP waveform from 80 to 120 msec following the response, subtracting a pre-response baseline of 50 msec.

   The Greenhouse-Geisser correction was applied where appropriate to correct for possible violations of the analysis of variance assumption of sphericity. The text lists corrected p-values.
3. It is difficult, of course, to tease apart the lateralization in these waveforms that results from the lateralized readiness potential, and the possible lateralization of the error-related negativity. Nonetheless, the measure we used for this analysis, with its pre-response baseline, should be sensitive to differences that emerge after the response. Differences emerging before the response, such as the lateralized readiness potential, should not affect the measure. Furthermore, visual inspection failed to reveal a point of inflection in the lateralized readiness potential waveforms corresponding to the peak of the error-related negativity. Such an inflection point would have suggested that error-related negativity activity contributed to the difference between the contralateral and ipsilateral waveforms.
4. Because the length of the recording epoch was too short for the 59 point filter used on the other data discussed in this paper, these data were filtered using a low-pass digital filter (boxcar method) with a high frequency cut-off point of 6.29 Hz.
5. The difference is visually most apparent as a 'peak-to-peak' difference. Our measure of error-related negativity amplitude, with its pre-response baseline, is sensitive to such differences, whereas other measures using a pre-stimulus baseline might not be.
6. Note that Niki and Watanabe (1979) found single units in cingulate cortex that responded selectively to errors in a task involving differential reinforcement of long latencies, a task which is less similar to choice reaction time than the task used by Gemba et al. (1986).

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