

this anterior forebrain pathway generates or transmits signals about the match between the actual sound of the bird's voice and its intent will require testing by recording neural signals generated in this circuit by altered feedback.

Birdsong can also be used to address the more general question of how performance-based feedback is used to shape complex motor output. The anterior forebrain pathway is homologous to the mammalian basal ganglia, and experiments in this system support a role for the basal ganglia in evaluating sensory feedback during calibration of motor performance. Brainard and Doupe⁷ show that damage to these circuits may have little effect on previously learned behavior while conspicuously disrupting the capacity to modify that behavior adaptively. This offers a glimpse into how the basal ganglia may be

involved in motor learning, including speech learning. The present results on song modification in birds and the proposed role of cortical–basal ganglia circuitry suggest that it would be worthwhile to examine the function of the related human circuits during modification of speech by altering feedback, a manipulation already known to change the sounds people make¹². There is already evidence that the basal ganglia are more strongly activated when subjects speak a second language than when they speak in their native tongue¹³. Perhaps we, like songbirds, get strong signals from our basal ganglia when we vainly attempt to match our memory of high-school French to the sounds we are producing.

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Anterior cingulate and prefrontal cortex: who's in control?

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A sophisticated study of error-related brain potentials in patients with prefrontal lesions addresses how we monitor performance and adjust cognitive control based on task demands.

Cognitive control, the ability to guide thought and action in accord with internal intentions, lies at the heart of most higher mental faculties that make us human, such as planning, reasoning, problem solving and language. Prefrontal cortex (PFC) is critically involved in the control of behavior. What is less clear are the structures (possibly including PFC) that are involved in monitoring performance and deciding how and when to allocate control—that is, determining when a goal is not being satis-

factorily met and deciding to devote more effort to it. Such a function is needed because cognitive control has limited capacity, which is painfully obvious to anyone who has tried to talk on the phone while reading e-mail. In this situation, you soon lose track of the conversation, and once you recognize this (monitoring function), you give up on the computer and redirect attention to the phone (allocation function)—assuming you are sufficiently interested or polite! The existence of such processes has long been recognized in both empirical work¹ and cognitive theory², but little research has focused on the brain mechanisms involved.

In this issue, Gehring and Knight³ have addressed this question in patients with dorsolateral PFC damage by studying the error-related negativity (ERN)^{4,5}. This electrical scalp potential, the first identified neurobiological index of performance monitoring, is closely associ-

ated with commission of errors in cognitive tasks. The proposed localization of this signal to midline frontal structures, particularly anterior cingulate cortex (ACC), is supported by neuroimaging⁶. However, the precise monitoring function responsible for this signal is still unclear. Is it caused by error itself, or by processing conflicts that produce uncertainty and predispose to error? Nor is it clear how such monitoring functions relate to mechanisms for executing control (response correction or the response slowing that frequently follows errors).

Gehring and Knight's study³ is an admirable example of how sophisticated behavioral design combined with indices of brain function in brain-damaged patients can provide insights into the neural basis of cognitive function. In their task, subjects saw two letters, one red and one green. They were cued to respond only to one color (say, the red letter) and asked to decide whether this was an H or an S. They responded by squeezing one of two hand grips (one for H and another for S) that measured the response force and could also detect any effort to correct errors. Sometimes both letters were the same (red H, green H), so that the irrelevant letter did not offer any distraction, and sometimes they were different (red H, green S). On such 'incongruent' trials, subjects had to ignore the distracting letter while responding to the correct one. This task is similar in critical respects to more familiar response competition tasks (like

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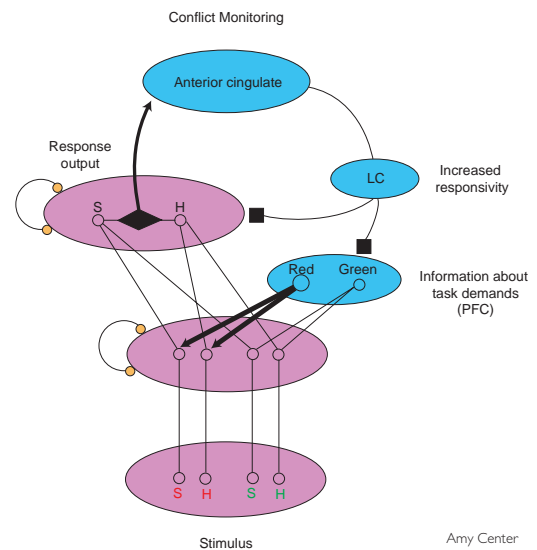
the Stroop task). All such tasks engage cognitive control by requiring selective attention to the relevant stimulus (or stimulus dimension—in this case, its color).

Such control is rarely perfect, so distracting stimuli usually influence processing. This is evidenced by an 'interference effect'. On incongruent trials, subjects tend to respond more slowly and/or make more errors (responding to irrelevant stimuli). They also make strategic adjustments based on their own behavior and on stimulus types. Subjects sometimes quickly correct an error. They also typically adopt a more conservative strategy to increase accuracy after an error by slowing their responses. Finally, they adjust to the occurrence of incongruent trials, whether they make an error or not, by increasing attentiveness on subsequent trials⁷ or during extended blocks of incongruent trials⁸, as shown by decreased interference effects under such conditions. Such adjustments indicate that subjects actively monitor their performance and use this information to adjust cognitive control allocated to the task.

To study how such monitoring functions (presumably reflected in the ERN) relate to the execution of control (indexed by behavior), Gehring and Knight recorded the ERN during task performance in patients with dorsolateral PFC damage. This provides a unique opportunity to determine which parameters are affected by such damage, thus permitting inferences about PFC's role in monitoring, the execution of control or both. The study of brain-damaged patients is particularly valuable, as it complements correlative neuroimaging methods with the opportunity to establish causal relationships. Such methodological advances are especially important in the study of performance monitoring, long the neglected stepchild of research on cognitive control.

Gehring and Knight found that patients committed approximately as many errors as control subjects, and the ERN associated with these responses was of comparable magnitude³. Thus the authors conclude appropriately that PFC does not itself generate the ERN. Frontally damaged patients also exhibited more forceful errors and diminished error correction, suggesting to the authors that monitoring capacity may be intact in patients, but their capacity for control is impaired. Nevertheless, Gehring and Knight conclude that simple models of

Fig. 1. Model of task used by Gehring and Knight. Components related to performance of the basic task are in purple, and components related to control are in blue. Stimuli are coded by feature-specific representations, and converge on an output layer with units corresponding to each response. The task demand layer has units corresponding to each of the two task-relevant stimulus features. Representations within layers are mutually inhibitory (that is, they compete, as designated by looped connections with filled circles). Note that this model is homologous to well-studied models of other response competition tasks, such as the Stroop task (see ref. 15), except that there is no asymmetry



between task-relevant and -irrelevant dimensions of processing. This model also adds a more fully specified control system, including a layer that responds to conflict (co-activation) of response units (ACC). This drives a neuromodulatory system (locus coeruleus, LC) that increases responsivity of processing units globally (for instance by a change in gain, designated by connections with squares). This modulates selective attention by its influence on representations of specific task demands (PFC) as well as motor preparation by its influence on response units.

interaction between PFC and ACC cannot explain their data. However, we support another interpretation, also considered by the authors: that PFC lesions allow multiple competing response representations to become active, and ACC detects the resulting response conflict. In this view, PFC exerts control by representing task demands (for example, target stimuli or relevant stimulus dimension). These representations favor the processing of task-relevant information, allowing it to competitively suppress irrelevant information activated by incongruent distractors, and thus reduce response conflict. Damage to PFC would weaken task representations, and thus increase conflict, even on correct trials. If the ERN reflects ACC response to such conflict, it should be present in correct trials for patients, as the authors observed. This interpretation is consistent with both fMRI results^{6,9,10} (A.W. MacDonald, J.D.C., V.A. Stenger & C.S.C., unpublished observations) and computational modeling of the control and monitoring mechanisms involved (Fig. 1).

One might ask, however, what leads Gehring and Knight to suggest that a more complex model is required. They argue that if information flows only from ACC to PFC, then PFC damage should not affect the ERN, contrary to their data. However, this is not neces-

sarily so. As just discussed, PFC damage could produce a larger ERN on correct trials by reducing control, and thus increasing conflict, on such trials. Another point, of greater concern, has to do with the pattern of corrective actions observed: increased force of error responses, reduction of error corrections, but intact post-error slowing. If PFC alone is responsible for control, then why does damage impair some forms of corrective action but not others? One answer may be that there are multiple forms of control, only one of which depends on PFC. Specifically, if error corrections and response force reflect one form of control (selective), whereas post-error slowing represents another (general preparatory), then PFC damage could impair the first type but not the second.

The form of control considered so far involves selective attention, which favors processing of a particular task-relevant stimulus type or dimension. Several theorists argue that such control is a cardinal function of PFC^{11,12}, and recent evidence from neuroimaging studies supports this view (for example, A.W. MacDonald, J.D.C., V.A. Stenger & C.S.C., unpublished observations; M. Banich *et al.*, *Soc. Neurosci. Abstr.* 25, 2167, 1999). Disturbing this form of PFC-dependent control can explain both increased error response force and diminished correc-

tions in frontally damaged patients. As noted above, the primary function of such control is to favor the processing of task-relevant information, and allow it to competitively suppress processing of distracting information. Thus, a disturbance in this mechanism permits stronger processing of distractors. Insofar as this is an important source of errors, these should be executed with greater force. The reduction in the number of error corrections can be explained similarly, as the likelihood for such correction is closely (and inversely) related to the strength of the competing process that led to the error. Note that a failure in PFC-dependent control should increase interference effects for incongruent stimuli, as has been observed in patients with frontal disturbances^{11,12}. Similarly, we would predict that these patients should fail to show sequential adjustment effects (that is, increases in selective attention) following incongruent trials. It would be interesting to know whether these effects occurred in the Gehring and Knight study³.

The preservation of post-error slowing in frontally damaged patients may be explained by assuming that it reflects the operation of a different form of control, independent of dorsolateral PFC. Although this view may seem unparsimonious, it is not *ad hoc*. Most observations of post-error slowing are reported in tasks that do not rely heavily on selective attention (that is, no distractors are presented, so increasing selective attention is not advantageous). Furthermore, the overall slowing of responses is not itself particularly selective. These considerations suggest that post-error slowing may be mediated by an effect on response preparation (for example, decreased response threshold) that does not depend on PFC, perhaps mediated by direct input from ACC to motor preparation areas. An intriguing alternative is that both PFC-mediated effects and response preparation effects may be mediated by a common brainstem neuromodulatory system that increases system responsivity globally (for instance, the gain of processing units in all parts of the network) as conflict in a task increases. Engaging this system would simultaneously heighten selective attention by augmenting activated representations in PFC, as well as motor preparation by doing so in response areas. We have proposed that the noradrenergic brainstem nucleus locus coeruleus (LC) may provide such a

mechanism¹³, a hypothesis with some empirical support¹⁴. Here we speculate that this LC system responds to ACC conflict detection (Fig. 1). This model is consistent with Gehring's and Knight's results, as PFC lesions would disrupt the system's ability to respond with adjustments in selective forms of control, but would preserve more general forms such as motor preparation (as evidenced by post-error slowing).

In our view, then, the same ACC response to conflict could drive two different but parallel pathways of control: one involving the PFC that increases selective attention (when this is useful), and another that is PFC independent and affects response preparation more directly. We have explored the plausibility of this hypothesis using neural network models of several cognitive tasks¹³ (M.M.B., T.S. Braver, C.S.C., D.M. Barch & J.D.C., unpublished observations).

However, Gehring and Knight's findings present an important challenge for this account. If response adjustments are driven by conflict detection, as indexed by the ERN, and in patients this is equivalent in magnitude for errors and correct trials, then why do they show adjustment effects selectively following errors? This is particularly puzzling in light of prior evidence linking performance slowing directly to the ERN⁴. There are several possible explanations of this finding. As the authors note, post-error slowing may result in part from the persistence of problems that caused the error. This may be a particularly problematic confound in patients.

Another important factor may be the heterogeneity of error trials, which are often composed of very fast responses and much slower ones. If this mix is different in patients—perhaps with a greater proportion of faster responses (consistent with the greater response force for errors), then the ERN (a response-averaged signal) to errors may have been artifactually blunted in contrast to correct trials. Selective averaging based on reaction time might reveal a larger ERN for error trials among patients than was reported, consistent with adjustments in performance on subsequent trials. In any event, it is clear that the relationship of the ERN to subsequent adjustments in performance warrants more detailed examination, both in empirical studies, and in computational models developed for normal function that can be used to explore the effects of damage.

In summary, the results of Gehring and Knight³ may be explained by the possibility of two pathways for control, both of which respond to ACC conflict detection and are mediated by neuromodulatory systems, but one of which is further mediated by PFC and has a more selective influence on task demands, while the other has a more general preparatory function. This account makes some interesting predictions (for instance, regarding effects of frontal damage on congruency effects, corresponding sequential adjustment effects and their relationship to the ERN). However, it also faces some interesting challenges that warrant further investigation. Whether this simple account of the division of labor between PFC and ACC prevails or, as suggested by Gehring and Knight, more complex interactions occur, the authors are to be commended for their elegant and provocative study of the neural mechanisms underlying monitoring and execution components of cognitive control. We laud Gehring and Knight for their work, and will continue to monitor with great interest the execution of their efforts in this exciting area of research that beckons for greater allocation of attention.

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