

when subjects can use a general inhibitory mechanism to stop their responses. When selective inhibitory mechanisms are involved, the LRP threshold is not exceeded (5). This inconsistency between LRP and unit data may be the result of a difference in the way saccadic eye movements (as evaluated by Hanes and Schall) and hand movements (as evaluated by the Illinois group) are controlled.

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Hanes and Schall demonstrate a correlation between the neural activity in the frontal eye field (FEF) of monkeys and the commonly observed variability in the reaction times of visually evoked saccadic eve movements. However, the question remains as to whether this correlation represents a causal relationship, or whether it is only an epiphenomenon reflecting changes in neural activity in a number of related cortical eye fields. The authors appear to overemphasize the role of the FEF in initiating saccades. The FEF is one of at least five well-defined, highly interconnected areas in the cerebral cortex that each have credentials similar to those cited for the FEF. The down-played question is, What happens to the execution of saccadic eye movements when the FEF is completely destroyed? This experiment has been performed (1). After complete bilateral surgical removal of the FEF, monkeys were able to make accurate saccades with normal reaction times to visual targets as soon after surgery as they were able to be tested (4 days). Only when FEF lesions were combined with parietal eye field lesions were saccade deficits serious and long-lasting. This result demonstrates that the remainder of the cortical eye movement system is able to generate normal vision-guided saccades in the absence of the FEF. The neural activity within the FEF is therefore not the only neural activity in the cerebral cortex that can cause the execution of an accurate saccadic eye movement.

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Hanes and Schall address response time variance much more than its mean. This variance is called irreducible, and tentative explanations offered include unpreventable noise and desirable adaptation. Barinaga's Research News article refers



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to dice-throwing in the brain. However, the variability of response time can decrease with practice and other factors. A significant portion of the variance may be accounted for by considering that the cell examined has multiple inputs and that the circuits subserving these inputs have spontaneous activity that can leave them in different states on different trials. The brain can be involved in activities other than that specified by the researcher. One clue to the effect of the initial state on the rate of growth of neural activation is in the comparison of activation levels in panels C and D of figure 3 of Hanes's and Schall's report. The neural activations in slow and fast trials illustrated in the two graphs not only have different growth rates, they also appear to differ in prestimulus baseline firing rates. This does not alter the value of the data and conclusions, but points to network-related sources of variance as a major element in interpreting the results.

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Response: The work of the late Jean Requin should receive the appropriate recognition for bridging psychology and neuroscience. We were influenced by his approach. Experimental psychology and modern neuroscience research began together, but historical developments resulted in something of a divorce until fairly recently. Requin's work was one of the earliest manifestations of the new reunion of neuroscience and psychology that has become cognitive neuroscience.

In our report, we found that when monkeys inhibited eye movements, the activity of the cortical neurons did not reach the critical threshold that would have generated a movement. In contrast, Coles and his colleagues found that in some of their subjects, the magnitude of the movement-related scalp potential recorded when grasping movements were withheld exceeded the threshold observed before movements were executed. Thus, note 16 in our report misconstrues this element of their data, and a correction is in order. We agree that the basis for the difference between the brain systems responsible for eye movements and those responsible for hand movements deserves further attention.

Lynch contrasts our observation of a correlation between neural activity in the frontal eye field and eye movement initiation with the well-known fact that eye movements are produced by a distributed network of multiple cortical areas and subcortical structures. Our data and conclusions do not contradict this fact. Work in the oculomotor system, as well as in the skeletal motor system, shows clearly that many neurons contribute to the motor command. However, the precision of the relationship we observed suggests that the growth of activation of eye movement-related neural activity throughout the brain is coordinated in such a fashion that the instant that a movement is triggered is agreed on by the various brain regions. Consensus, rather than independence or competition, may be the rule of response preparation. Whether there is a coordinated rise of activation to a movement-triggering threshold, which may represent a motor system complement to the perceptual binding problem, can be evaluated experimentally by recording simultaneously from many movement-related neurons within and across saccade-related structures.

Richer and Achim make the astute observation that in figure 3 of our report, the level of activity preceding target presentation was higher in trials with shorter saccade latencies than it was in trials with longer latencies. However, in an analysis of all of our data, we found no evidence that the level of activity preceding target presentation accounted for a significant fraction of the variance of reaction time. Jeffrey D. Schall Doug P. Hanes Department of Psychology, Vanderbilt University, Nashville, TN 37240, USA E-mail: <u>schalljd@ctrvax.vanderbilt.edu</u>

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