E-Letter responses to:

Ziad M. Hafed, Laurent Goffart, and Richard J. Krauzlis
A Neural Mechanism for Microsaccade Generation in the Primate Superior Colliculus
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A Neural Mechanism for Fixation Instability

Martin Rolfs

Laboratoire Psychologie de la Perception
Université Paris Descartes
45, rue des Saints-Pères
75006 Paris, France

Z. M. Hafed et al. show in their Report ("A neural mechanism for microsaccade generation in the primate superior colliculus," 13 February 2009, p. 940) how the brain generates the smallest rapid eye movements. Rostral neurons of the superior colliculus (SC) encode goal locations very close to the fovea and fire vigorously before microsaccades into their response field. The authors have made an important step in revealing the causal evidence for this mechanism, which had previously been explained in theory based principally on psychophysical evidence (1). Understanding the origin of fixation instability illuminates the basic limits of visual perception; in the absence of retinal image motion, perception fades (2, 3). However, fixation instability comprises both microsaccades and the slower, erratic drift movements (4), and both may work together to provide sufficient retinal image slip (5). I therefore highlight two important implications of this research for understanding how fixation instability is achieved to serve visual function.

First, if fixation instability serves to provide a necessary baseline of retinal motion, then a dynamic visual input may reduce the level required for the baseline. Indeed, one of the hallmarks of fixation behavior is a strong drop in microsaccade frequency in response to sensory events, an inhibition that may result from a strong decrease of rostral SC activity (1), which is compatible with Hafed et al.'s findings.

Second, the SC is involved in generating slow eye movements that pursue a moving target (6, 7). Therefore, I propose that if an imbalance of activity in the rostral SC does not generate a microsaccade, it biases slow drift movements, accounting for the cooperation of both in fixation control (8, 9). Examining the relation between spontaneous drift movements and rostral SC activity is certainly more of a challenge than examining the relation for microsaccades. However, this would provide the cornerstone for our comprehension of overall fixation control.

Martin Rolfs

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Reply to M. Rolfs' E-Letter

Ziad M. Hafed, Richard J. Krauzlis

Systems Neurobiology Laboratory
Salk Institute for Biological Studies
10010 North Torrey Pines Road
La Jolla, CA 92037, USA

Laurent Goffart

Institut de Neurosciences Cognitives de la Méditerranée
Equipe Dynamique de la Perception Visuelle et de l'Action, UMR 6193
CNRS–Aix-Marseille Universités
13402 Marseille, France.

We agree that the functional role of microsaccades and other fixational eye movements remains an interesting open question. We take issue, however, with M. Rolfs' use of the term "fixation instability." In our Report, we did not uncover a "neural mechanism for fixation instability," but rather a mechanism for generating a type of eye movement that is frequently observed during gaze fixation. There are several pathological conditions that involve problems with maintaining stable fixation, such as square-wave jerks, saccade oscillations, and ocular flutter (1). We suggest that the term "fixation instability" be reserved for these clinically identified oculomotor disturbances, and not for what is apparently a normal aspect of visuomotor function.

The questions of what triggers microsaccades, why their frequencies may be modulated by stimulus transients, and how visual functions are dependent on or utilize them are still unanswered. For example, with respect to the idea that microsaccades are important for vision because they prevent retinal image fading, many other contributors to retinal image motion do exist, including movements of the head, body, and objects in the world. In addition, high-acuity visual tasks that require focused and foveal attention, such as threading a needle (2), result in a reduction in microsaccade frequency and therefore a reduction in retinal image motion, not the opposite. Thus, it may be the case that the importance of fixational eye movements for visual perception lies more in how they refine the spatial localization of minute visual features or how they modulate the image statistics of natural scenes (3) than in how they refresh retinal images. These questions should continue to be a topic of active scientific inquiry.

We also agree that the neural mechanisms for slow drifts, and the functional role of these movements, deserve further investigation. Rolfs alluded to previous results relating the superior colliculus (SC) to smooth pursuit eye movements (4–7), and he suggested similar involvement in slow drifts. We should clarify here that these previous results have shown that SC activity is important for identifying the location of the goal (8) for pursuit, which is distinct from providing a premotor signal for the generation of slow eye movements. It is therefore possible that modulations of SC activity during slow drifts reflect the continuously shifting locus of the fixated target caused by these small eye movements. It is also possible that such modulations, if present, reflect ascending inputs (9) to the SC about eye position. In either case, the neural control of slow drifts is expected to arise from multiple distributed brain circuits, which may or may not include the SC.

There are also several other important questions that our study raises and are worthy of being mentioned here and investigated in the future, both at the behavioral and neurophysiological levels. Some of these questions are: Does the neural control of microsaccades originate in the SC, or are there inputs from the frontal eye fields (FEF) or other cortical areas that play a crucial role in how and when these movements are generated? Are there corollary discharge signals for microsaccades sent to cortex, as there are for larger voluntary saccades? Are microsaccades and

other fixational eye movements actively controlled by the brain to condition how visual signals are encoded? Answering these, and other questions about foveating mechanisms promises to be a very exciting challenge.

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