Miniature eye movements jitter the retinal image unceasingly, raising the question of how perceptual continuity is achieved during visual fixation. Recent work discovered suppression of visual bursts in the superior colliculus around the time of microsaccades, tiny jerks of the eyes that support visual perception while gaze is fixed. This finding suggests that corollary discharge, supporting visual stability when rapid eye movements drastically shift the retinal image, may also exist for the smallest saccades.

Eye movements are the visual system’s most powerful tool to gather relevant information distributed across the visual scene. In particular, saccades rapidly swing the eyes into new positions, bringing potentially interesting objects closer to the fovea and increasing the available visual resolution. However, the clear benefits of these rapid eye movements come at a cost. Because of the high speeds of the eyes during the movement, the image is smeared across the retina, degrading target details during and around the time of a saccade; however, the retinal smear is never perceived and we can easily keep track of relevant items in the scene as the eyes move about (e.g., Cavanagh et al. 2010; Wurtz 2008). This perceptual continuity (also referred to as visual stability) is probably achieved by a combination of several mechanisms, including 1) predictive remapping of behaviorally relevant locations, anticipating the consequences of saccades (e.g., Wurtz 2008; Rolfs et al. 2011); 2) active suppression of visual information during saccades (Diament et al. 2000); and 3) strong backward masking by the clear and stable retinal image after the saccade (Castet et al. 2002).

Although the latter is obviously based on retinal information, predictive remapping and saccadic suppression require an extraretinal corollary-discharge signal accompanying the saccade command. One such signal originates in the superior colliculus (SC), making this subcortical key node of eye movement control a central player in visual stability (Wurtz 2008).

As long as the problem of perceptual continuity in the presence of eye movements has been discussed it has largely been kept in the realm of large eye movements, although the eyes continue to move even while resting at an object in the visual scene, jittering about a constrained spatial region. These miniature fixational eye movements include irregular ocular drifts as well as microsaccades, often traversing less than a quarter of a degree of visual angle. Although fixational eye movements are small, they produce shifts of the retinal image that would easily be seen if they were caused by motion in the world—however, we do not see the world bathed in jitter. On the contrary, microsaccades and other fixational eye movements serve vision in many ways (Rolfs 2009).

What are the mechanisms enabling this perceptual absence of perpetual motion? In general terms, fixational eye movements can be estimated and discarded based on the consistent image motion they generate across the whole retina (Murakami and Cavanagh 1998). Microsaccades, however, share their generative mechanisms with large saccades, including a causal role of the SC (Hafed et al. 2009). This opens the possibility that corollary-discharge–based mechanisms also exist for microsaccades. In a recent issue of The Journal of Neuroscience, Hafed and Krauzlis (2010) provided evidence for such an extraretinal mechanism with a potential source in the SC whose anatomical features (Lee et al. 2007) and central role in both eye movement control and corollary discharge (for a review, see Wurtz 2008) made it a primary target to study sensory suppression at the time of microsaccades.

Hafed and Krauzlis triggered bursts of neural activity in caudal SC cells by flashing bright stimuli in the visual periphery of monkeys fixating a small spot. Carefully monitoring the monkey’s eye position, Hafed and Krauzlis observed that the neurons responded with vigorous bursts when microsaccades were rare and more weakly when the stimulus flashed around the time of a microsaccade. In their short report, the authors did not explore whether this effect was instantiated either by inverse changes of oculomotor behavior and neural responses across experimental sessions (e.g., more microsaccades and less responsive neurons by the end of the day) or by instant changes in neural responsiveness at the time of microsaccades. Clearly, however, microsaccadic events correlated with a strong reduction of the magnitude of stimulus-induced bursts in the SC. This visual suppression started around 70 ms prior to microsaccade onset and lasted until about 70 ms after the end of the microsaccade, a time course largely compatible with that observed for the elevation of visual thresholds around the time of microsaccades (Zuber and Stark 1966).

Having revealed a neural correlate of microsaccadic suppression in the SC, Hafed and Krauzlis then established a correlation between this neural effect and the monkeys’ behavior. In a separate set of trials, the authors collected saccadic response time data when the fixation target stepped into the visual periphery, triggering a reactive saccade. As in humans (Rolfs et al. 2006), response times increased when a microsaccade occurred at the time of stimulus onset (see Fig. 1A). The time course of this behavioral effect was remarkably similar to the time course of neural suppression of visual signals, giving rise to a sensory interpretation of lengthened saccadic latencies after microsaccades. Mediated by inhibitory connections of the rostral SC (generating microsaccades; Hafed et al. 2009) to upstream sensory areas (e.g., the lateral geniculate nucleus [LGN]), sensory suppression could weaken neural responses of SC cells coding the target location, thereby delaying saccade
execution (Fig. 1B, Mechanism I). This intriguing account of the behavioral data suggests that microsaccades delay subsequent behavior on the basis of their consequences for sensory processing. It seems unlikely, however, that sensory suppression fully captures these interactions between microsaccades and saccades because the original studies involved both memory-guided and visually guided saccades and found microsaccade-induced prolongation of saccade latencies even if the target stimulus was long gone when the saccade was due.

Still, the observed correlation between neural activity and behavior is stunning and calls for future investigation to unravel the contributions of microsaccade-related sensory and motor processes to the generation of intended action. One alternative explanation for the correlation of microsaccade-related visual responses in the SC and saccadic response times is based on competing motor plans. Those neurons in the SC map that generate microsaccades might inhibit distant sites in the SC map directly via long-range, lateral interactions, instantiating a competition between active visual fixation (including microsaccades) and saccade generation (Fig. 1B, Mechanism II). In this view, inhibition of neurons in the caudal SC results in a reduced impact of visual signals impinging on their receptive fields and thus in a delayed generation of a response saccade. Based on the present set of data, however, this proposal (as the one put forward by the authors) is beyond proper evaluation at this time. It should be the goal of future studies to turn strong correlations into causal evidence for either hypothesis.

What is the functional value of the sensory suppression that Hafed and Krauzlis found in the SC? Our answer is twofold. Many scholars, including Hafed and Krauzlis (2010), imply that one purpose for saccadic suppression of sensory signals is the reduction of blurry visual input created by the rapid shift of
the retinal image. However, the necessity of an active suppression mechanism to counteract a disruption of perceptual stability by self-generated visual signals has been strongly contested. The stable retinal image at the onset of fixation (after a saccade) by itself provides an efficient mask of the perisaccadic blur, rendering it invisible (Castet et al. 2002). Therefore we argue here, and pointed out earlier (Rolfs 2009), that microsaccadic suppression plays a more indirect role in perceptual continuity, by emphasizing the reliable information coming in after the microsaccade. Visual suppression at the time of microsaccades effectively adds net power to the instant visual input following microsaccades, producing recurrent synchronized stimulation and advancing the processing of dependable visual information during fixation.

Sensory suppression at the time of microsaccades may also serve a second purpose: to largely preserve the distribution of activity in the topographic map in the SC when a microsaccade causes the image to sweep across the retina. This may be crucial for two reasons. As we have pointed out earlier, the SC is a key structure in the generation of saccadic eye movements. Its intermediate layers span a map of relevant locations. Each location in the map, when stimulated sufficiently, triggers a saccadic eye movement with a stereotypical amplitude and direction (see Fig. 1B for an illustration). Visual stimulation at a particular location in the visual field causes local activation in this map and thus contributes to the generation of a saccadic eye movement. When a microsaccade sweeps the visual field across the retina, transient visual activation may impinge anywhere on the map. Reduced responsiveness of SC neurons around the time of microsaccades may therefore prevent the spurious triggering of large saccades to random locations. In fact, these inhibitory effects may linger on during the subsequent fixation, to result in the prolongation of saccade latencies that we discussed earlier. Second, we now know that the SC is also necessary for the control of covert spatial attention. The most striking evidence for this is that reversible inactivation of a location in the SC map impairs the covert selection of signals for perceptual judgments at the corresponding location in the visual field (Lovejoy and Krauzlis 2010). Thus activation in the SC map indexes relevant targets in the visual scene, i.e., effectively represents attentional pointers to locations in space (Cavanagh et al. 2010). This crucial location information might be lost if microsaccade-induced visual transients had a large impact on the distribution of SC activity and inhibition of visual input at the time of microsaccades may prevent this from happening.

The study by Hafed and Krauzlis takes an important step toward the understanding of visual stability in the face of continuous eye movements during fixation. Their work points to a potential origin of microsaccadic suppression and, more generally, to a corollary-discharge signal for these fixational eye movements. It provides a starting point for a new line of studies aimed at uncovering extraretinal mechanisms accompanying microsaccades. We expect this field to flourish in the years to come. Besides the closer scrutiny of the origins, consequences, and functions of microsaccadic suppression, it will also include the study of spatial updating across microsaccades as well as many other aspects of what might be called transmicrosaccadic perception.

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