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Review

Neuronal mechanisms for visual stability: progress and problems

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How our vision remains stable in spite of the interruptions produced by saccadic eye movements has been a repeatedly revisited perceptual puzzle. The major hypothesis is that a corollary discharge (CD) or efference copy signal provides information that the eye has moved, and this information is used to compensate for the motion. There has been progress in the search for neuronal correlates of such a CD in the monkey brain, the best animal model of the human visual system. In this article, we briefly summarize the evidence for a CD pathway to frontal cortex, and then consider four questions on the relation of neuronal mechanisms in the monkey brain to stable visual perception. First, how can we determine whether the neuronal activity is related to stable visual perception? Second, is the activity a possible neuronal correlate of the proposed transsaccadic memory hypothesis of visual stability? Third, are the neuronal mechanisms modified by visual attention and does our perceived visual stability actually result from neuronal mechanisms related primarily to the central visual field? Fourth, does the pathway from superior colliculus through the pulvinar nucleus to visual cortex contribute to visual stability through suppression of the visual blur produced by saccades?

Keywords: corollary discharge; efference copy; visual stability; superior colliculus; medial dorsal nucleus; frontal eye field

1. INTRODUCTION

Our vision remains stable despite the interruptions produced by rapid saccadic eye movements that shift the image on the retina several times per second. These saccades are the key to our remarkable visual abilities because they move our high-resolution fovea rapidly from one part of the visual scene to another in order to direct this high resolution towards successive regions of the visual field. Saccades also present two major problems that the brain must solve. The first problem is to construct a stable picture of the visual world from the successive 'snapshots' obtained as we foveate objects of interest between eye movements. With each new snapshot, the retinal image is displaced, and objects of interest are likewise displaced. The visual system must piece these snapshots together accurately to produce a stable percept. The second problem is that each eye movement causes the visual scene to be swept rapidly across the retina, but this powerful visual motion stimulus is simply an artefact of our own movement and must be ignored. How we get the benefits of high-resolution perception without the disruptions inherent in the eye movements that facilitate it has been a repeatedly revisited perceptual puzzle.

The major hypothesis used to explain this stability of perception is that there is a non-visual signal providing information that the eye has moved, and this information in some way is used to compensate for the motion. Three sources of non-visual signals are available to the brain. The first two are both sources of inflow information: proprioception from the eye muscles and visual information from the retina itself. The third source is referred to as outflow information: signals within the brain that indicate that the eye is about to move. The inflow signals for saccades are likely to be of minimal importance. Experiments on proprioception have indicated that it contributes little information on each saccade, and the visual input during the saccade is little more than a blurred image (see the more extensive discussion in [1]). In contrast, the signals within the brain have been considered critical for producing visual stability. These internal brain signals have been referred to as a 'sense of will' by von Helmholtz [2] in the nineteenth century and as a 'corollary discharge (CD)' by Sperry [3] and as an 'efference copy' by von Holst & Mittelstaedt [4] in the twentieth century.

Progress in the search for neuronal mechanisms underlying a CD or efference copy recently has been made in the best animal model of the human visual system, the Old World monkey [5–8]. This progress has been at higher levels of the pathway for the control of movement that are remote from the final efferent pathway. Accordingly, the concept of a corollary rather than an efference copy seems more appropriate, and here we will therefore use the term CD, but the two terms are essentially interchangeable. This work centres on the identification of a pathway to frontal cortex and the recognition that the activity in the

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One contribution of 11 to a Theme Issue 'Visual stability'.

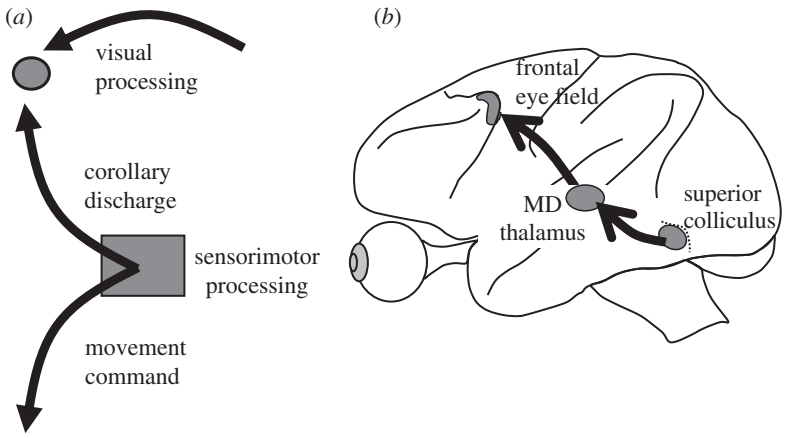

Figure 1. A corollary discharge (CD) in the monkey brain. (*a*) The CD originates from the same sensorimotor processing area as does the motor command to produce the saccadic eye movement. The CD projects to other regions of the brain including those devoted to visual processing. (*b*) A pathway conveying a CD extends from the intermediate layers of the superior colliculus (SC), through the medial dorsal (MD) nucleus of the thalamus, to the frontal eye field (FEF) in the frontal cortex.

pathway has the characteristics expected of a CD associated with saccadic eye movements. This CD in turn acts on the neurons in frontal cortex thought to have a relation to stable visual perception.

In this article, we will first briefly summarize the progress made in exploring this pathway and the associated frontal cortex neurons. More detailed information on this pathway is available in recent reviews [1,9,10]. We then describe several ongoing questions in relating the neuronal activity already identified in the monkey brain to issues of visual stability in humans. We consider four questions. First, how the neuronal activity can be related to the monkey's visual perception: Is the identified activity can be related at all to the neuronal mechanisms for stable perception? Second, we explore whether the neuronal activity underlies the mechanisms for stability proposed on the basis of a specific set of psychophysical experiments: Does the neuronal activity underlie the proposed transsaccadic memory hypothesis of visual stability? Third, we begin to consider the interaction of visual stability and visual attention: Are the neuronal mechanisms studied so far modified by visual attention? Finally, we consider what contribution pathways other than those to frontal cortex might make to visual perception: Do subcortical paths through the pulvinar nucleus of the thalamus contribute to the stability of visual perception?

2. THE RELATION OF COROLLARY DISCHARGE TO SHIFTING RECEPTIVE FIELDS IN FRONTAL CORTEX

The logic of a CD is relatively simple: the same signal sent from a sensorimotor area in the brain to generate a movement is also sent to other regions of the brain to inform those regions of the impending movement (figure 1*a*). This copy sent to other regions is the CD. Areas thought to receive such information include areas where sensory processing occurs or specifically visual processing in the case of the saccadic system.

In the monkey brain a putative CD pathway for saccades extends from the superior colliculus (SC) on the roof of the midbrain to frontal cortex. This pathway extends from neurons in the SC intermediate layers through the medial dorsal (MD) nucleus of the thalamus to the cortical frontal eye field (FEF, figure 1*b*). The characteristics of the neuronal activity travelling in this anatomically identified pathway [11] meet the four criteria that seem to be necessary for identifying the activity as a CD based on previous research in simpler animals [5–7,10]. First, the neurons originate in a sensorimotor structure that is clearly related to the generation of movement, the intermediate layers of the SC, where neurons increase their discharge before saccades to the opposite visual hemifield. Second, neurons in MD, which have been identified as relay neurons, carry a movement related signal; they increase their activity before saccades to the contralateral visual field. Third, these MD neurons are not in the pathway that is necessary to generate saccades because inactivation of the relay neurons did not disrupt either visually or memory guided saccades. Fourth, and most important, inactivation of MD did disrupt the monkey's ability to do a double-step saccade task that requires a CD for its performance [12]. These characteristics were deemed to be substantial enough to conclude that this anatomically identified pathway from SC to the FEF carries a CD for saccadic eye movements.

The next question is whether this CD contributes to the stability of perception. The specific contribution is that it might help to resolve the problem of displacement; with each saccade, areas of interest are displaced on the retina. The proposed connection between CD and perceptual stability is based on the landmark experiment of Duhamel *et al.* [13], who demonstrated that neurons in the parietal cortex had the remarkable attribute that their visual sensitivity shifted in anticipation of the upcoming saccade. This change in sensitivity has been referred to as remapping or spatial updating (which emphasizes the conceptual significance) or as shifting receptive fields (RFs; which simply describes the neuronal activity), and the term shifting RFs will be used here.

How might the problem of displacement be resolved at a neuronal level? Suppose a monkey looks at one point (figure 2*a* left, fixation point) and we

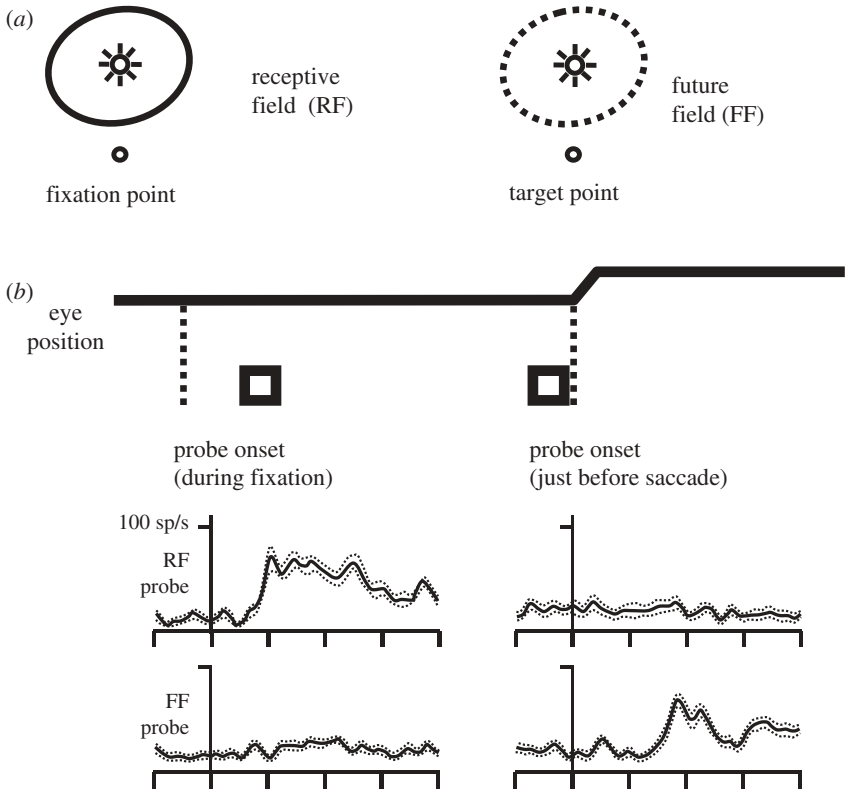

Figure 2. Neurons with shifting receptive fields (RFs) in frontal and parietal cortex. (*a*) The defining characteristic of a neuron showing a shifting RF is that just before the onset of a saccade, a region of the visual field becomes sensitive to visual stimulation. This region has the same spatial relation to the target of the saccade as the RF has to the present fixation point. The region is referred to as the future field (FF) of the neuron. (*b*) An example of an FEF neuron with a shifting RF. The left column shows the response in a spike density plot over multiple trials to a 50 ms probe stimulus in the RF of the neuron (upper record) and the lack of response at that time in the FF (lower record). The right column shows the increase in activity to the probe stimulus in the FF flashed just before the onset of the saccade and the lack of response in the RF field at that time. Adapted from Sommer & Wurtz [8].

record from a neuron in its brain. The neuron will be maximally sensitive to visual stimuli falling in one part of the visual field, the RF of the neuron (figure 2*a*, RF with a stimulus in its centre). This is simply the conventional RF seen throughout the visual system. Some cortical neurons, however, have an added property. As the monkey prepares to make a saccade to a target point, the sensitivity of these neurons shifts to the location the RF will occupy after the saccade. This location is referred to as the future field (FF) of the neuron (figure 2*a* right). For a time before the saccade some neurons are activated by a stimulus in both their current RF and FF. After the saccade, the FF of the neuron becomes the RF. The whole sequence then begins again with the preparation of the next saccade.

The relation of this FF activity to visual stability is based on the idea that it is the increased sensitivity at the FF before the saccade that highlights the same object before and after the saccade [13]. If the saccade produced the visual displacement, what fell on a given location of the retina before the saccade should fall on that same part after the saccade. Comparing the activity in the RF after the saccade to the activity in the FF before the saccade is a potential neuronal mechanism that might underlie the perception of visual stability.

Shifting RFs were first identified in the lateral intraparietal area (LIP) of parietal cortex [13] and have subsequently been studied in the FEF area of frontal cortex [8,14,15]. Subsequent studies have extended the initial findings on shifting RFs in LIP [16–19]. Comparison of shifting RFs between areas found that they are progressively less robust in earlier extrastriate visual areas [20]. In humans, evidence consistent with RF shifts has been provided by studies using functional magnetic resonance imaging [21–23] and transcranial magnetic stimulation [24].

To study neurons with shifting RFs, the usual strategy is to probe the sensitivity of the RF and FF locations at varying times before and after the saccade with 50 ms light flashes. Figure 2*b* illustrates the activity in the RF and in the FF during fixation and just before a saccade for an example FEF neuron. Long before the saccade, the neuron responded to probes flashed in the RF but not in the FF (figure 2*b* left). In contrast, when the probe was flashed just before the saccade, the activity in the FF increased (figure 2*b* right). The increased sensitivity at the FF was not due just to the preparation to make the saccade because when there was no probe, there was no increase in activity (not shown); the change in sensitivity in the FF was evident only when probed with a stimulus. In some cases the RF activity declined at the same time when the FF activity increased (as in figure 2*b*) and in other cases the RF activity remained. When one declines and the other increases, the time course of the changes is close to symmetrical [19].

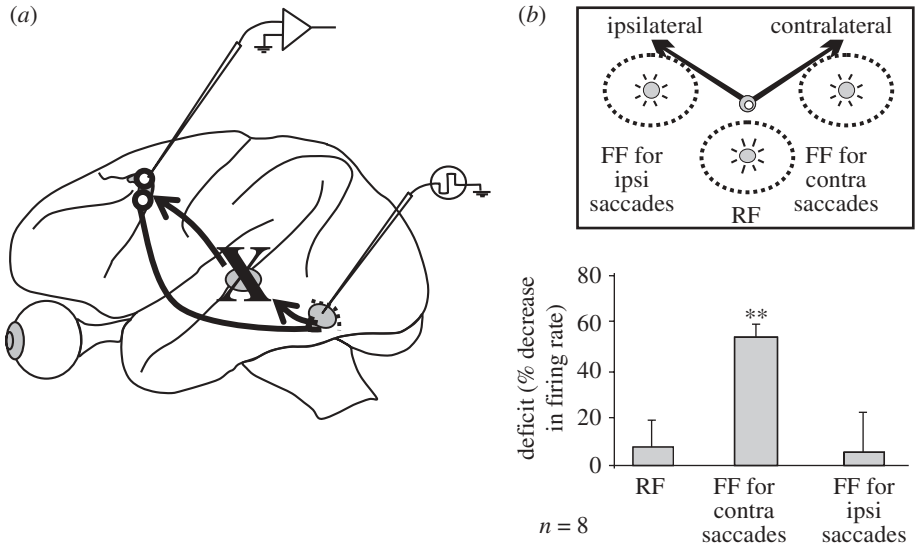

Figure 3. Dependence of FEF shifting RFs on the CD input from MD thalamus. (*a*) FEF neurons were identified as receiving from or projecting to the SC by using stimulation of the SC. Those FEF neurons with both visual RFs and shifting RFs were studied before and during inactivation of the relay region of MD. (*b*) The per cent decrease (mean and s.e.m.) in the activity of eight FEF neurons with shifting RFs during MD inactivation. Bar graph shows (i) the lack of decrease in the RF response, (ii) over 50% decrease in the FF with saccades directed to the visual field contralateral to the brain that was inactivated, (iii) the lack of decrease in the FF with saccades to the ipsilateral visual field. Asterisks indicate a significant difference with $p < 0.0001$, *t*-test. Adapted from Sommer & Wurtz [8].

In order for the increased sensitivity of the FF to be located where the RF will be after a saccade, these neurons with shifting RFs must have information about the direction and amplitude of the impending saccade, and they must have it prior to saccade execution. This is exactly the information that could be provided by a CD of the impending saccade: the CD has these metrics of the saccade and it has them before the saccade is executed. The obvious next question is whether the CD that ascends from the SC to the FEF provides the CD that drives the shifting RFs. This question was answered by first verifying that the FF activity had the temporal and spatial characteristics expected if the activity resulted from input from the SC–MD–FEF pathway carrying the CD. Both spatial and temporal characteristics of the shifts were consistent with the input of such a corollary, as summarized elsewhere [8]. The acid test of the contribution of this identified CD to the FF activity is whether the shift of activity in the FEF neurons depends upon the input from the CD pathway. Because the pathway passes through an identified region of MD thalamus, it was possible to test this dependence by inactivating that region while measuring the shift of activity in an FEF neuron. To do this, neurons in the FEF were studied that both had clear shifting RFs and had established connections to SC as indicated by orthodromic or antidromic stimulation (figure 3*a*). Next, the region of the relay in MD thalamus was verified and then inactivated with the gamma-aminobutyric acid agonist muscimol. The amplitude of the RF and FF activity was then measured before and after the MD inactivation [8]. The graph in figure 3*b* shows that there was a significant decrease in the amplitude of the FF response across the eight experiments that were completed. The shift was reduced by about 50 per cent. This was true for the saccades to the contralateral visual field that should receive input related to that field from the SC through the inactivated MD. For saccades to the ipsilateral visual field, which should not receive such input, there was no such deficit.

Further control experiments showed the specificity of the deficit. The RF response of the neurons was not significantly changed, which indicates that the MD inactivation did not block the input of the visual input to the FEF neurons. The monkey also could still make saccades to the target, confirming that the MD inactivation did not block saccade generation. Thus, neither the visual nor the saccade-related activity was altered by MD inactivation. It was only the magnitude of the shifting RF activity that was altered, which is dependent upon the interaction of visual and CD input. These findings provide strong evidence that the CD transmitted through MD is a major source of the input that drives the shifting RFs.

3. DO COROLLARY DISCHARGE DRIVEN SHIFTING RECEPTIVE FIELDS CONTRIBUTE TO STABLE VISUAL PERCEPTION?

At this point, we can be reasonably certain about three facts on the relation between the neuronal activity we discussed and stable visual perception. First, we have an identified CD pathway that carries information about saccadic eye movements to the frontal cortex from the SC. Second, we know that neurons in FEF, LIP and other areas have shifting RFs. This anticipatory activity in the FF of the neuron predicts the response to the visual stimulus that will fall on the RF of the neuron after the saccade. Third, the CD required to determine the location of the FF is at least in part provided by the CD reaching the FEF from SC. So the general nature of the shifting RFs and the source of the required CD has been sketched out.

What is missing in this sketch is the demonstration that the shifting RFs materially contribute to visual stability and, farther down the road, the discovery of the exact mechanisms by which they do so. At this point, the most parsimonious interpretation is probably that they contribute to the control of movements that depend upon a CD, such as the second saccade in the double-step task that is altered after disruption of the CD [5,7]. A critical test for relating the shifting RFs to stable perception is a demonstration that without them stable perception is impaired. More specifically, the test would be an inactivation that reduces the shifting RFs with subsequent testing for a deficit in stable perception, just as interruption of the CD disrupts the CD-dependent saccades in a double-step task.

The critical test has yet to be performed, but one of the established observations now makes the test possible. Namely, the shifting RFs now have been shown to be dependent upon the input of the CD emanating from the SC, and this CD can be interrupted by inactivation of MD. Prior to this observation, the only test possible was inactivation of cortical areas with neuronal shifting RFs, which would disrupt not only the shifting fields but also any visual processing in the area. MD inactivation makes it possible to inactivate the CD without such general visual disruption (as in figure 3 for testing the dependence of shifting RFs on the CD). Thus, inactivation of MD offers the possibility of reducing the CD reaching cortex, reducing the FEF shift, and if the shift is in fact related to perception, reducing stable perception.

The next experimental step is to devise a way for the monkey to inform the experimenter that there is a reduction in perception. This will be challenging but several approaches are already available. The required task is one that shows that a monkey with reduced shifts has a reduced ability to distinguish stimulus displacement owing to its own movement from genuine shifts in an external physical stimulus. One approach, demonstrated recently by Sommer & Crapse [25], directly measured a monkey's ability to detect the movement of a stimulus during an eye movement using psychophysical procedures. Another approach might be to train monkeys on the task devised by Deubel *et al.* [26] for testing the human's ability to detect shifts in the saccade target when it is displaced during a saccade. While these experiments are demanding psychophysically, they indicate that establishing the relation of the shifting RFs to perception might turn out to be a tractable one. They do, however, have two major limitations. The first is if there is no deficit: this provides very limited information because the inactivation alters only one of what might be many CD pathways in the brain. The second is if there is a clear deficit, it might have resulted from blocking other mechanisms that depend on the integrity of MD rather than just acting on the CD. But even with this caveat, the experiment would establish an MD pathway as critical for behaviourally determined visual stability.

4. ARE SHIFTING RECEPTIVE FIELDS RELATED TO TRANSSACCADIC MEMORY?

How might neuronal phenomena relate to hypotheses about visual stability that emerge from psychophysical experiments? Here, we consider the possibility that the neuronal activity seen in frontal and parietal cortex might be related to a specific hypothesis about visual stability referred to as the transsaccadic memory hypothesis. The hypothesis grew out of a series of psychophysical experiments by Deubel and his collaborators [26–28]. We focus on this transsaccadic hypothesis because the observations underlying it are relatively simple and easy to compare with the neuronal activity already observed. Transsaccadic hypotheses are more general, however, having been proposed as a result of a number of types of experiments including those by McConkie & Currie [29]. In addition, recent experiments that show stimulus adaptation acting across saccades provides further evidence that post-saccadic perception takes into account visual input before the saccade [30].

The basic premise of the transsaccadic memory hypothesis is that visual stability is assumed unless there is specific evidence that the assumption can be rejected. In simplified outline, the transsaccadic memory proposal comprises three main stages. First, the features of the saccadic target and of objects immediately surrounding it are stored in a transsaccadic memory. Second, after the saccade, this memory of the target and surrounding area is compared with what is now at and around the new fixation point. Finally, the outcome is evaluated. If the before-saccade and after-saccade features are similar, the assumption of a stable visual world is met. If not, the target must have moved, and the assumption of stability fails.

The key observation underlying this transsaccadic hypothesis is that the visual presence of the saccadic target immediately after the saccade is essential to perceptual stability. This has been tested by displacing the saccade target while the saccade is in flight and asking the subject whether the displacement was detectable. The important finding is that even relatively large displacements of the saccade target are not detected unless the target is absent immediately after the end of the saccade. The insensitivity to large displacements had been reported by Bridgeman *et al.* [31]. Deubel *et al.* [32,33] went on to show that this failure to detect displacement can be reversed by blanking out the target for at least 50 ms after the end of the saccade. The point is that there seemed to be something special about the presence of the target immediately after the saccade, a point that had been emphasized previously [34]. In the transsaccadic memory hypothesis, if the target has not changed by the time it becomes the fixation point after the saccade, stability is assumed and the displacement that occurred is ignored. But if the fixation point after the saccade is different from the saccade target, the assumption of stability is rejected and the displacement is recognized as real. The fundamental assumption is that if the target remains in the same position before and after the saccade, it and the visual world are stable. The total absence of the target right after the saccade is the largest change possible in the target and should

therefore produce the clearest recognition of a change with a saccade.

This default assumption of stability has its roots in the ideas about the role of CD posited by MacKay [35] a number of years ago. He argued that a CD is best regarded as a question asked at the start of a saccade, with the answer provided by the visual input resulting from the saccade. In the views he put forward, the world is assumed to be stable unless the post-movement answer to the CD question provides evidence to the contrary. The optimal inference approach, which is based on Bayes' theorem and has been used to explain visual perception at the time of saccades [36,37], has striking similarities to Mackay's ideas. Prophetically, MacKay also expected that the stability assumption would be tested at points in the brain well beyond the initial stages of visual processing, probably where both visual and saccadic activity were represented.

This brings us back to the implementation of transsaccadic memory by neurons with shifting RFs in frontal and parietal cortex. The basic mechanism of the transsaccadic memory hypothesis is that an object at or near the target of the impending saccade is compared with the object at or near the fixation point after the saccade. The shifting RF neuronal activity has a parallel to this comparison: activity before the saccade to a stimulus in the FF can be compared with after the saccade to the response to the same stimulus in the RF. The initial experiments on shifting RFs were performed before the generation of the transsaccadic hypothesis, but the potential parallel between this psychophysically-based hypothesis and the neuronal observations makes it worth exploring the similarities and differences between them.

A significant problem in relating the transsaccadic hypothesis to the shifting RFs is spatial resolution. If a comparison is made between the activity related to the visual stimulus present in the FF before the saccade and the RF after the saccade, this comparison will be made between two stimuli falling on regions of the retina with substantially different spatial resolutions. In the case of a neuron with a RF 3° to the right of the current fixation point and an impending 20° horizontal saccade, this would mean that the visual stimulus for the FF will have the lower resolution of 23° eccentricity rather than 3°. The comparison pre- and post-saccade would be of necessity at substantially different resolutions. A possible solution to this problem would be for the comparison to be done at a low spatial resolution regardless of the retinal resolution of the specific stimulus. If the comparison were made in the primary visual area (V1), the difference of 20° in eccentricity would make the comparison difficult. In contrast, if the comparison were done at higher levels in the visual system with relatively lower spatial frequency resolution, the problem would be substantially reduced. Then only the lower spatial frequencies of the stimulus would be processed and the difference between the central visual field and the periphery would be less severe. FEF and LIP might be ideal areas for the FF to RF comparison. Our knowledge of the spatial frequency sensitivity of neurons in these areas is unfortunately limited, but their relative lack of stimulus specificity (compared with V1) would be consistent with relatively low spatial frequency responses. Perhaps the declining prominence of shifting RFs as one moves backward in the dorsal visual pathway from LIP, to V3A, V2 and V1 [20] is related to an increasing resolution at the earlier stages of the visual pathway. If FEF and LIP do indeed have such a lower spatial frequency, this property would seem to make them ideal candidates for such a comparison of features in the pre-saccadic and post-saccadic space. We also lack detailed knowledge of the FF size when compared with the RF field size, and this could also influence the pre- and post-saccadic comparison.

A closely related issue in comparing the transsaccadic memory hypothesis to the shifting RFs is that the hypothesis entails a pre-saccadic to post-saccadic comparison between objects in and around the saccade target. This would seem to require neuronal mechanisms for object recognition that are unlikely to exist in FEF or LIP. There are two factors, however, that may mitigate this problem. First, psychophysical experiments have not yet specified the degree of precision for the 'match' between the pre-saccadic and post-saccadic objects. Experiments showed that the reference objects need not be at the saccade target and that they need not be exactly the same before and after the saccade [26]. The issue is the range of variation that is tolerated and whether that range would fit within the range of discrimination of neurons in FEF and LIP. The presumed low spatial frequency sensitivity of neurons in these areas might fit a relative lack of specificity observed in the transsaccadic memory experiments [26]. The second mitigating factor is that LIP, and possibly also FEF, may have more feature selectivity than is typically appreciated [38].

The final problem that must be at least acknowledged is that a comparator of some type would be needed, and the nature of such a neuronal comparator remains unknown. This is hardly surprising given that whether the comparison is even made is not established. A comparator might in fact not be needed if an alternative interpretation of the FF activity is considered: the FF activity acts just as an indicator that a saccade has occurred. As a consequence of the indicator, the transient with the saccade is ignored as an invitation to attend to a new object just as occurs in change blindness (M. Shadlen 2009, personal communication). Essentially, this indicator logic also relies on an assumption about stability, but now if there is an indicator, a saccade is assumed to occur and the disruption from the saccade is ignored. There is no need for a comparator, and spatial resolution is irrelevant. There remains, however, the issue of what 'reads' the indicator and the mechanism by which this reading produces the visual stability.

5. THE ROLE OF ATTENTION: MUST THE WHOLE VISUAL FIELD BE STABILIZED?

Saccades generate the problem of perceptual stability by displacing the visual scene with each eye movement. Attention drives saccades; they are usually made to a part of the visual field to which attention is directed.

Thus, visual stability and attention are intertwined at a behavioural level. At a neuronal level, the mechanisms related to saccade preparation might underlie the shifts of attention as well [39–41]. It is therefore worth beginning to consider the possible relationships between visual attention and visual stability. Two behavioural demonstrations emphasize this point.

First, as we have already noted from the discussion of transsaccadic memory, the objects compared before and after a saccade have to be at least near the saccade target. There can be little doubt that top-down attention or goal-directed attention accompanies these saccades. A number of experiments have shown increased efficiency of visual processing of stimuli located around the target of the upcoming saccade [42–44]. One example is the improvement in letter discrimination when one letter among a cluster of letters is the target of the saccade [42]. The other aspect of these attention experiments is that it is apparently not possible to direct attention to one location and make a saccade to an adjacent location, an indication of the strength of the coupling between spatial attention and saccades [42]. The point of these experiments for the present discussion is that attention is primarily (though not exclusively) directed to the future central visual field and not the field as a whole.

A second line of experiments on the phenomenon of change blindness (for summaries see [45–47]) emphasizes the importance of attention. The key point of these experiments is that even a substantial change in the visual scene is not seen if the transient associated with the change is eliminated. In contrast, if attention is directed to the visual field area where the change occurs, the change is readily apparent. The phenomenon has also been demonstrated in monkeys [48]. The relevance of this to the current discussion is that visual changes occurring during saccadic eye movements are also missed [49]. These experiments indicate that we probably do not maintain a perceptual image of the entire visual scene but only that part to which we pay attention.

One inference we can draw from these two lines of behavioural experiments is that stability might be only required in regions of the visual field where attention is directed. A further inference is that, because attention is usually directed to the target of an impending saccade, any mechanism of stabilization might be concentrated on (but not limited to) the visual field around the saccade target. This inference has substantial implications for investigating the role of attention on the possible neuronal mechanisms of stability including those related to shifting RFs.

There are two issues. First, in the experiments done so far, neurons in which shifting RFs have been investigated have been in regions beyond the fovea, principally in the regions beyond 5°–10° from the fixation point. The frequency and magnitude of shifts for neurons with RFs at or near the fovea have simply not been investigated. So the visual field of greatest interest when we consider the role of attention has not been investigated. Second, when we consider attention, we have to distinguish types of attention, one in the behavioural analysis and another in the neuronal experiments. In the behavioural analysis, we are considering the effect of top-down or goal-directed attention that is directed towards the location of the next fixation. In the neuronal experiments, this top-down attention is directed to an arbitrary saccade target not to the stimulus in the FF of the neurons. In fact, in order to make sure the monkey is not planning a saccade to the FF stimulus, a number of experiments have required a second saccade to a target away from the FF stimulus [5,14], which should direct both the saccade and any goal-directed attention away from the FF stimulus. Instead of being modulated by goal-directed attention, the activity at the FF in the shifting RF experiments is more likely to be modified by the shift of attention generated by the stimulus onset. This bottom-up attention may be a major factor because the experiments are done with flashing lights on a blank screen in a darkened room, conditions that should maximize onset attention. Such an onset attention effect has been illustrated in LIP by a reduction of the FF activity when there is no onset attention (see fig. 1 in Gottlieb *et al.* [50]).

The effect of onset attention in the FEF has been investigated in a recent study by Joiner *et al.* [51], who found a significant reduction in the amplitude of FF activity in FEF neurons under conditions in which we would expect the onset attention effect to be reduced. In these experiments the location and size of the RF of an FEF neuron were determined along with the magnitude of the response to a 50 ms flash at a point in the centre of the RF. The next step was to test the effect of adding distractors in the visual field because adding distractors in the visual field should reduce the strength of onset attention [52]. In order to be certain that these distractors were remote from the RF, so that they did not visually alter the response of the FEF neuron, the RF edge of the neurons was estimated and the distractors were then placed at least 10° beyond that point (figure 4*a*). These added stimuli came on in the distractor trials at the same time as the RF stimulus. The addition of the distractors did not significantly alter the response to the flash in the centre of the RF for this example neuron ($p = 0.11$, one-tailed t-test, figure 4*b*, grey line), although for other neurons it did do so. The next question was whether the FF showed any modulation with the addition of distractors (figure 4*c*). We tested this by flashing a spot of light in the centre of the FF just before the onset of the saccade and then testing the effect of adding distractors. The distractors were flashed at the same time as the FF stimulus and again outside of the FF, assuming that the FF is about the same size as the RF. The result for this neuron was that with the distractors, the FF activity of the neuron was significantly reduced ($p < 0.001$, one-tailed t-test) from that observed when the FF stimulus was presented alone (figure 4*d*). Thus, insofar as the distractors act to reduce the onset attention effect of the single visual stimulus, this experiment demonstrates that the amplitude of the typical FF activity benefits from an onset attention effect.

Two additional points need to be made about this experiment. First, the reduction in the amplitude of

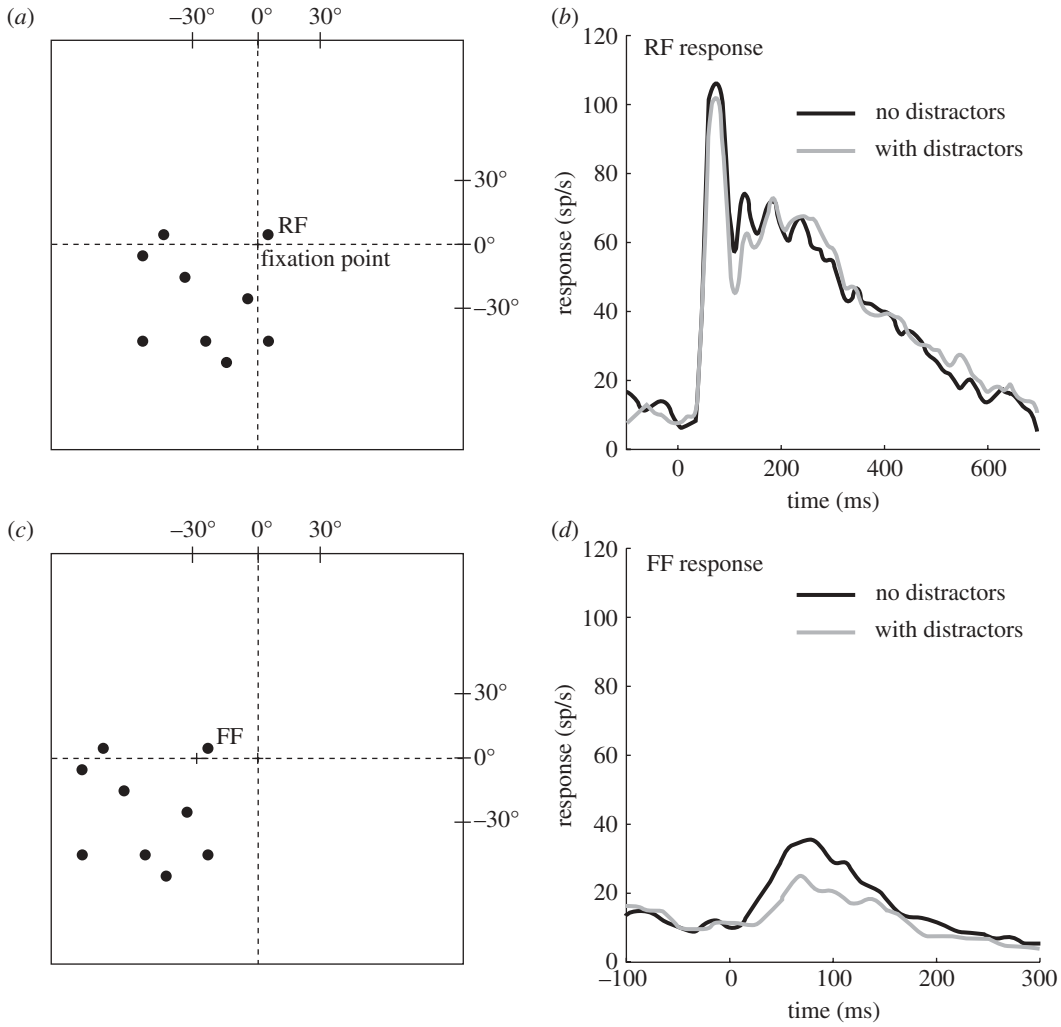

Figure 4. Reduction of FF activity when onset attention is reduced by the presence of distractors. (*a*) The location of the flashed stimulus falling in the RF of an FEF neuron (RF) and the configuration of eight flashed distractor stimuli outside the RF. (*b*) Response to the RF stimulus and to the RF stimulus with distractors. (*c*) Location of the flashed FF stimulus flashed just before the saccade and the eight distractors flashed at the same time. (*d*) Activity following the FF stimulus and following the FF stimulus with distractors.

the FF activity with the addition of distractors might be a minimal estimate of the effect of reducing onset attention. Because we tried to keep the distractor stimuli outside of the RF of the neuron, we placed them at a substantial distance from the FF stimulus, and we have probably reduced their effectiveness as distractors. Second, the reduction in FF visual activity with distractors was essentially measured as the difference between the change in the RF and the FF with the addition of distractors. Therefore, if the distractor stimuli invaded the RF of the neuron in spite of our efforts to minimize this possibility, this invasion would be present in both the RF and the FF visual activity.

This demonstration of the contribution of onset attention only indicates that this bottom-up modulation probably enhances the amplitude of the FF response in the shifting RF experiments. This observation becomes important, however, because it might mask the relative amplitude of the shifting RF response for neurons at different eccentricities; the amplitude might be determined by the amplitude of the onset attention rather than that of the shift itself. This in turn relates to the questions on the possible essential role of attention considered above, namely that the shift effect should be largest at the centre of the visual field if it underlies visual stability implemented by a transsaccadic memory. The next step then is to determine the frequency and amplitude of the shift effect with the onset attention effect reduced. This, of course, is only the first step in understanding the role of attention in the shifting RFs and ultimately their contribution to visual stability.

6. DO THE ASCENDING COROLLARY DISCHARGE PATHS THROUGH THE PULVINAR CONTRIBUTE TO VISUAL STABILITY?

We have concentrated on the pathway from SC through MD thalamus to the FEF, but it represents only one of several subcortical paths to cortex that may participate in creating the percept of stability. Other pathways to frontal cortex may also carry such a CD to wide regions of the frontal cortex, and still others carry information to parietal and occipital cortex. A major pathway linking brainstem to cortical visual areas emanates in the superficial layers of the SC and travels through the pulvinar nucleus of the thalamus (figure 5*a*). This pulvinar

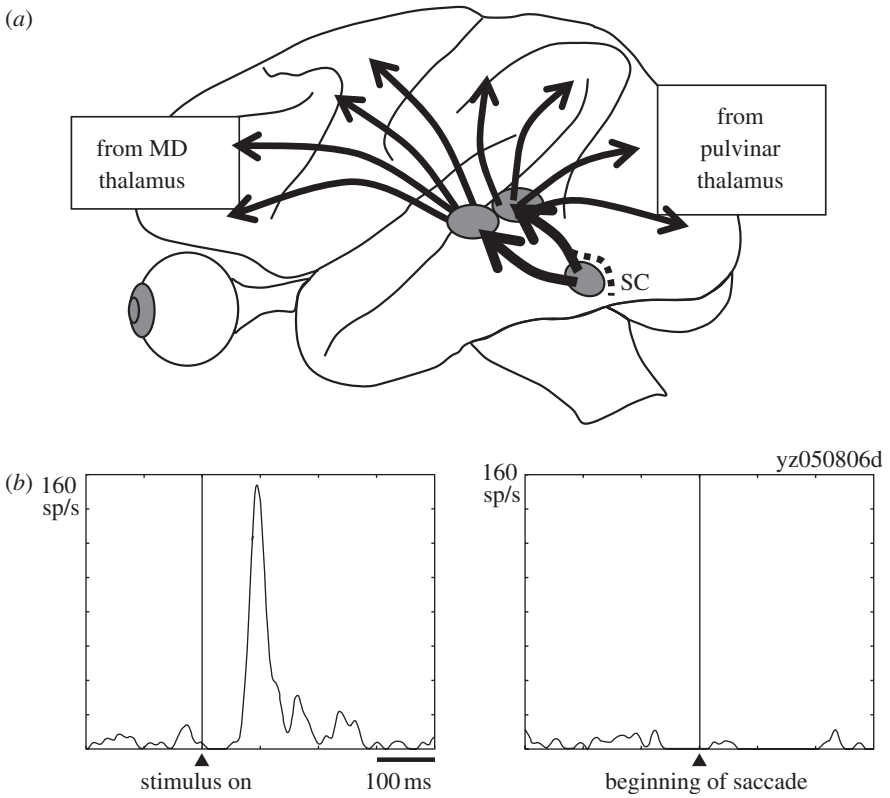

Figure 5. Contribution of the SC—pulvinar—cortical pathway to the visual suppression with saccades. (*a*) Symmetry of projections from MD to frontal cortex and from pulvinar to parietal and occipital cortex. (*b*) Increased activity of a pulvinar neuron in the path from SC to MT to a visual stimulus (left), and decrease in activity of that neuron following a saccade (right). Adapted from Berman & Wurtz [53].

pathway also may convey the effect of a CD on visual processing. While we have so far considered the issue of visual stability related to the displacement of the image during saccades, we will conclude the discussion of stability by including some recent experiments directed at the other compensation for the visual disruption resulting from saccades, the suppression of the blur or smear resulting from visual stimulation during saccades.

How does the visual system effectively ignore the visual blur caused by saccades? The CD is one available signal that the visual system could use to distinguish visual stimulation owing to self-generated eye movements (for the interaction of CD and visual masking see [1]). The effect of a CD on visual processing was first demonstrated for the visual neurons in the superficial layers of the SC [54–56]. Neurons in the superficial SC are visual in nature, and respond strongly to large-field motion stimuli [56]. One might think, then, that these neurons might also respond to the visual stimulation brought about by saccades, which similarly cause a rapid sweep of the entire visual field across the retina. Instead, the activity of many of these superficial layer neurons is suppressed after a saccade. Furthermore, the suppression occurs at about the time that the visual input generated by the sweep of the retinal image would reach the SC. This suppression is of both the visual response to stimuli that swept across the retina during the saccade and of any background activity present. Experiments by Richmond & Wurtz [55] demonstrated that the saccadic suppression was triggered by a CD and did not depend on a proprioceptive signal from the eye muscles. To do this, the eye muscles were inactivated (via a retrobulbar bloc), but the time of the attempted saccade was still known by recording from neurons in the extraocular muscle motor nuclei. When the burst of these motor neurons indicated an attempted saccade, there was still a suppression of the background activity of the SC visual neurons, despite the inactivation of the eye muscles. Therefore, the suppression was not generated by proprioceptive signals from the muscles but by an internal signal, the CD of the motor command.

Suppression has been observed in visual areas other than the superficial SC, and the comparison of suppression in several areas may hold clues to how the signals are conveyed. For example, the suppression seen in the SC visual neurons is more prominent than that seen in the lateral geniculate nucleus (LGN) or V1 cortex (see review by Wurtz [1]). Suppression has also been observed in the extrastriate visual motion areas of the middle temporal (MT) and medial superior temporal (MST) cortex, originally by Thiele *et al.* [57] and more recently by Ibbotson and colleagues [58–60]. Recent experiments by Bremmer *et al.* [61] have included the ventral intraparietal and lateral intraparietal areas as well as MT and MST, and have found saccadic suppression that is qualitatively different in each of these areas. They conclude that this variation does not support the view that suppression observed in cortex is just the consequence of changes in the visual input arriving at cortex, presumably through the LGN. This raises the intriguing possibility that these cortical areas do

not inherit the suppression from the geniculocortical visual pathway but from the CD suppression in the superficial SC.

The pathway from SC to visual cortex is through the pulvinar nucleus of the thalamus but the exact pathway is unknown and in some dispute. Finding the pathway would open the possibility of interrupting any signal relaying suppression or other CD signals to cortex without damaging the sources of the signal in the SC or the target in visual cortex. The interruption in the pulvinar would be analogous to the inactivation of MD we have described in studying the contribution of CD to frontal cortex.

At least one leg of this pathway from SC superficial layer visual neurons to area MT has recently been identified [53] using the same techniques that were successful in identifying the pathway from SC to FEF. In these experiments, neurons in the likely relay pathway [62–64] were identified as relay neurons if they were antidromically activated by stimulation of MT and orthodromically activated by stimulation in SC visual layers. This technique not only identified relay neurons, but also defined the areas within pulvinar where neurons were related either to MT or to SC. Furthermore, subsequent histological staining allowed for the identification of subregions of the inferior pulvinar in which these neurons were located (using the nomenclature of Stepniewska & Kaas for pulvinar subregions [65,66]). The relay neurons were centred on the medial region of the inferior pulvinar, consistent with anatomical studies showing a strong projection from this region to MT [62,64].

In considering the relevance of this pathway for visual stability, a key question is whether the pulvinar neurons convey the suppression signal found in the SC. Previous studies in the pulvinar have found neurons with visual responses and with suppression after saccades [67,68]. Their properties were similar to those seen in the SC, but their connectivity was unknown. In the current study, neurons were identified as having input from SC and/or projections to MT, and a number have been found to show this suppression. Figure 5*b* shows one of these neurons having a response to a visual stimulus as well as a suppression of spontaneous activity after saccades [53]. This observation confirms the presence of the suppression signal in pulvinar neurons. Some of these neurons showing suppression with saccades had been shown to be relays between SC and MT, which indicates that suppression is conveyed to MT from SC through the pulvinar.

The next question is whether the neurons in this pulvinar path to area MT make a causal contribution to the neurons in MT that are suppressed by saccades. The contribution of the identified pulvinar path should now be testable by inactivation of the relay region in the pulvinar using the same techniques used to inactivate the MD nucleus.

7. CONCLUSION

Moving from a behavioural problem, particularly one related to higher order visual perception, to possible underlying neuronal mechanisms risks overstating the affinity between the two levels of analysis and the causal relation between them. It is unlikely that we have avoided these hazards, but our hope is that by considering several questions about the relation of possible neuronal mechanisms to stable visual perception, we might direct future experiments towards answering these questions.

At this point, we have neuronal activity in parietal and frontal cortex that anticipates before the saccade what will occur after the saccade, and at least for frontal cortex, the CD needed to produce this anticipation. We also have a growing number of hypotheses about how such activity might be related to visual stability, but at a neuronal level all are sketches of possibilities and not models with testable predictions.

What is obvious is that the question of visual stability and its underlying neuronal mechanisms is such a central issue in perception that it would be remarkable if it did not depend on multiple factors in behaviour, including memory and attention, and on multiple brain circuits, including those to parietal and occipital cortex in addition to the recently studied pathways to frontal cortex. Neuronal mechanisms that have tended to be regarded traditionally as separate entities might instead be regarded as intertwined in producing visual stability.

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