

THE ROLE OF FIXATIONAL EYE MOVEMENTS IN VISUAL PERCEPTION

*Susana Martinez-Conde**, *Stephen L. Macknik** and *David H. Hubel[‡]*

Our eyes continually move even while we fix our gaze on an object. Although these fixational eye movements have a magnitude that should make them visible to us, we are unaware of them. If fixational eye movements are counteracted, our visual perception fades completely as a result of neural adaptation. So, our visual system has a built-in paradox — we must fix our gaze to inspect the minute details of our world, but if we were to fixate perfectly, the entire world would fade from view. Owing to their role in counteracting adaptation, fixational eye movements have been studied to elucidate how the brain makes our environment visible. Moreover, because we are not aware of these eye movements, they have been studied to understand the underpinnings of visual awareness. Recent studies of fixational eye movements have focused on determining how visible perception is encoded by neurons in various visual areas of the brain.

We live in a constantly changing world, so nervous systems have evolved to detect changes in our environment. Motion in our visual field might indicate that a predator is approaching or that prey is escaping. Stationary objects generally pose less of a threat and so tend to be studiously ignored owing to neuronal adaptation mechanisms. Some nervous systems are specialized to detect only motion signals; for example, a frog might not see a resting fly, but will react rapidly as soon as the fly takes off. In a sense, this ability to see only moving objects might be true of all visual systems. Even our own visual system can detect stationary objects only because the images projected onto our retinas are never stationary for long (FIG. 1). Involuntary fixational eye movements prevent the adaptation of our neurons to an unchanging environment. The movement of the retinal images is what keeps stationary objects of interest from fading perceptually.

The development of methods to counteract eye movements and thereby cause visual fading^{1,2} led to a large amount of research, mostly during the 1950s and 1960s, which aimed to characterize the eye movements that occur during fixation. But in the late 1970s, the field of fixational eye movements seemed to arrive at an impasse. Interest in fixational eye movements began to wane because of difficulties in data collection,

discrepancies between results from different laboratories, and disagreements over interpretation of the available data. A revival of interest in the late 1990s was ushered in by the development of accurate methods for the measurement of eye movements³, in addition to the advent of single-unit recording techniques in alert monkeys. For the first time, it was possible to investigate the types of neuronal response (if any) that are generated by eye movements during visual fixation. By correlating neural activity with fixational eye movements (which are themselves correlated with the maintenance of visibility), these methods began to address how visibility itself is encoded in the brain. Here, we discuss recent discoveries concerning the neural correlates of visibility and our understanding of how our perception of a stable visual world is achieved during fixation.

Fixational eye movements and adaptation
Our visual system is governed by neural adaptation — steady illumination produces weak neural responses, whereas abrupt changes in illumination across space and time generate strong responses^{4–6}. In this sense, neural adaptation is the cornerstone of all visual processing. The cost of such a system is that unchanging features of the scene fade from view. Eye movements

**Barrow Neurological Institute, St. Joseph's Hospital, 350 W. Thomas Road, Phoenix, Arizona 85013, USA.*
[‡]Department of Neurobiology, Harvard Medical School, 220 Longwood Avenue, Boston, Massachusetts 02115, USA.
Correspondence to S.M.-C.
e-mail: smart@neuralcorrelate.com
doi:10.1038/nrn1348

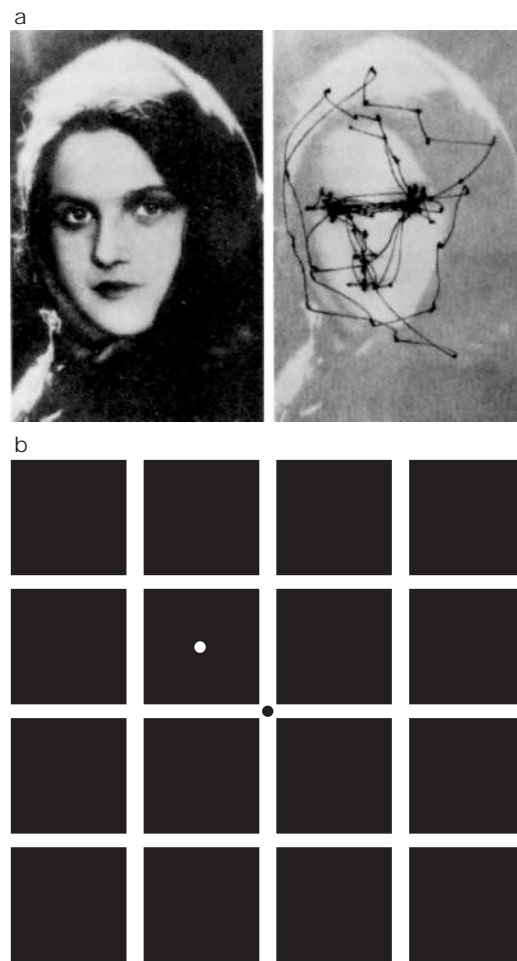


Figure 1 | **Eye movements during visual fixation.** **a** | An observer views a picture (left) while eye positions are monitored (right). The eyes jump, seem to fixate or rest momentarily, producing a small dot on the trace, then jump to a new region of interest. However, even during these fixation, or 'rest' times, the eyes are never still, but continuously produce fixational eye movements — drifts, tremor and microsaccades. Reproduced, with permission, from REF. 9 © (1967) Plenum. **b** | Pattern for showing fixational eye movements. To experience it, look at the central black dot for about a minute, then look at the white dot in the adjacent dark square. The dark after-image of the white line pattern should be seen in constant motion owing to fixational eye movements. Reproduced, with permission, from REF. 115 © (1961) Taylor & Francis.

during fixation are therefore necessary to overcome loss of vision due to uniform stimulation of the retinal receptors, even at the potential cost of a decrease in visual acuity⁷. The goal of oculomotor fixational mechanisms might be not retinal stabilization, but controlled image motion adjusted to be optimal for visual processing⁸. In the early 1950s, several groups showed that all eye movements could be eliminated in the laboratory, causing visual perception to fade to a homogeneous field^{1,2,9}. Although this might seem counterintuitive at first, it is a common experience in all sensory modalities — we do not generally notice that our shoes are on for 16 hours a day. When the eyes were released from artificial stabilization or if

the stabilized image was changed^{10–14}, visual perception reappeared; just as, if we wiggle our toes, we notice again that our shoes are on. BOX 1 shows how neural adaptation can lead to perceptual fading during normal visual fixation.

Retinal stabilization studies. Given the technical difficulties and potential health hazards of holding the eyes physically stationary in human subjects (with paralyzing drugs, for instance), retinal stabilization studies have generally used an alternative approach. In these studies, the visual stimulus is shifted in such a way that all eye movements are effectively cancelled. That is, the visual stimulus moves in the same direction, and at the same speed and amplitude, as the eye, so that the retinal image remains stable despite eye movements. Most retinal stabilization techniques fall into three categories: (i) a tiny slide projector or a mirror is mounted on a contact lens attached to the eye^{9,15} (FIG. 2). The target stimulus is projected through the pupil or bounced off the mirror, and therefore moves as the eye moves; (ii) the ENTOPTIC STRUCTURES in the eye are used as visual stimuli. For example, the retinal vasculature can be illuminated so it becomes visible, before fading rapidly due to adaptation^{13,14,16,17}; (iii) the movements of the eye are measured, either optically or through the rotation of a magnetic coil (which is surgically implanted in the eye, or mounted onto a contact lens). Data on eye position are then rapidly transmitted to a projection system that moves the stimulus and keeps it stable on the retina^{18–20}.

Early experiments found that images faded only after several seconds of stabilization, after which the visible image would regenerate for a few seconds. Then the images would fade again, and so on^{7,15,16,21}. However, recent studies have shown that even brief periods of stabilization lead to significant decreases in visibility²⁰. Moreover, it is now believed that the slow onset of visual fading and the occasional recurrent visibility of stabilized images were probably due to imperfect stabilization during eye movements²². In the past decade, Coppola and Purves¹⁷ found that images of entoptic vascular shadows (which are very stable) can disappear in less than 80 ms, indicating that normal visual processing might entail a very rapid mechanism for image creation and erasure. This is probably for the best, or we would presumably be unceasingly haunted by the ghostly image of our retinal vasculature under all viewing conditions.

Eye movements during fixation

It has long been known that our eyes are never still, even during fixation. Jurin, in 1738, referred to the “trembling of the eye”²³. Helmholtz, in 1860, also admitted the difficulty of maintaining perfect fixation, and proposed that this “wandering of the gaze” served to prevent retinal fatigue²³. Starting in the late 1800s, a variety of techniques for recording these eye movements were developed²⁴. Scientists today agree on the occurrence of three main types of eye movement during visual fixation in humans: tremor, drifts and microsaccades^{9,25} (FIG. 3).

ENTOPTIC STRUCTURES
Structures within the eye. When these become visible they give rise to entoptic images.

Box 1 | Visual fading outside the laboratory: Troxler's effect

Although perfect retinal stabilization is most easily achieved under laboratory conditions, fading of objects in our visual periphery occurs often in normal vision. Peripheral fading of stationary objects was first noticed by Troxler in 1804. Troxler reported that, under voluntary fixation, stationary objects in the periphery of vision tend to fade and disappear¹⁰⁶. In the late 1950s, Clarke made a connection between Troxler's fading and the fading of stabilized images in the laboratory^{1,2}, and attributed both phenomena to neural adaptation^{107–110}. The simplest explanation for Troxler's peripheral fading is that receptive fields in the periphery of our vision can be considerably larger than fixational eye movements (especially as accurate fixation tends to eliminate microsaccades^{56–59}). Drifts and tremor, being smaller than the peripheral receptive fields, do not provide effective visual stimulation to prevent peripheral visual fading, especially in the case of low-contrast stimuli. The figure is a demonstration of Troxler's effect. To experience it, fixate precisely on the red spot, while paying attention to the bluish annulus. After a few seconds of careful fixation, the annulus will disappear, and the red spot will appear to be surrounded by a completely white field. Movements of the eyes will immediately bring the blue annulus back to perception.



- NYSTAGMUS**
Involuntary rhythmical oscillations of one or both eyes.
- FOVEA**
The retinal region with maximal concentration of photoreceptors, where visual acuity is highest.
- FLICKER FUSION THRESHOLD**
The rate of flicker at which the flickering stimulus being viewed appears non-flickering (approximately 50–60 Hz in humans).
- CONJUGATE**
Coordinated in the two eyes.

Tremor. Sometimes called physiological NYSTAGMUS, tremor is an aperiodic, wave-like motion of the eyes⁷, with a frequency of ~90 Hz²⁵ (TABLE 1). Being the smallest of all eye movements (tremor amplitudes are about the diameter of a cone in the FOVEA^{9,24,25}), visual tremor is difficult to record accurately — tremor amplitudes and frequencies are usually in the range of the recording system's noise. The contribution of tremor to the maintenance of vision is unclear. It has been argued that tremor frequencies are much greater than the

FLICKER FUSION FREQUENCIES in humans, so the tremor of the visual image might be ineffective as a stimulus^{12,13,26}. But recent studies indicate that tremor frequencies can be quite low, below the flicker fusion limit²⁷. Greschner *et al.*²⁸ have shown that low frequencies (5 Hz) of tremor-like motion generate strong synchronous firing in the turtle's retina. Furthermore, early visual neurons can follow high-frequency flickering that is over the perceptual threshold for flicker fusion²⁹. So, it is possible that even high-frequency tremor is adequate to maintain activity in the early visual system, which might then lead to visual perception.

Tremor is generally thought to be independent in the two eyes. This imposes a physical limit on the ability of the visual system to match corresponding points in the retinas during stereovision^{27,30}.

Drifts. Drifts occur simultaneously with tremor and are slow motions of the eye that occur during the epochs between microsaccades (see TABLE 2 for a description of drift parameters). During drifts, the image of the object being fixated can move across a dozen photoreceptors²⁴. Initially, drifts seemed to be random motions of the eye³¹, generated by the instability of the oculomotor system³². However, drifts were later found to have a compensatory role in maintaining accurate visual fixation in the absence of microsaccades, or at times when compensation by microsaccades was relatively poor^{33–36}. Drifts have been reported to be both CONJUGATE^{27,31} and non-conjugate^{9,37}. As with tremor, drifts might result from the noise and variability of neuronal firing to the ocular muscles^{25,38}. If drifts and tremor are indeed conjugate, this might indicate that they have a central origin (at least in part). This idea supports observations of reduced or absent tremor in patients with brainstem lesions³⁹.

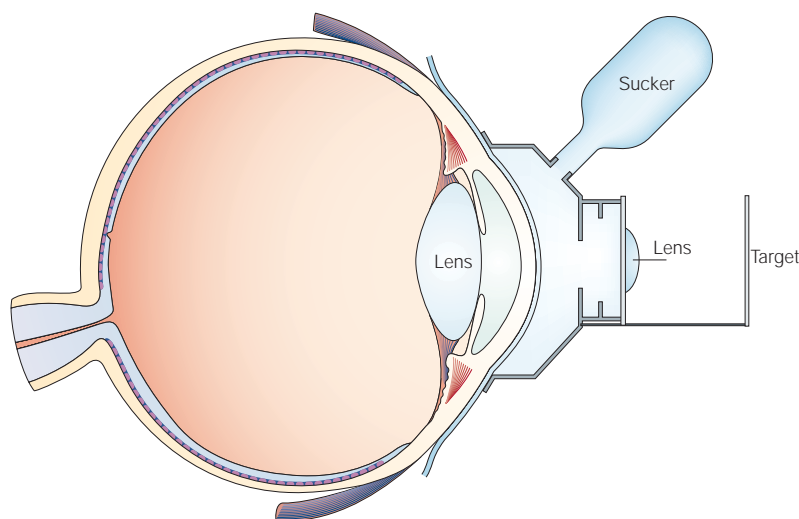


Figure 2 | **Early retinal stabilization studies.** This drawing illustrates the suction cup technique, used by Yarbus⁹ and others. This technique was very popular in early retinal stabilization studies for its simplicity, but it is now considered old-fashioned, and other, less invasive stabilization techniques are preferred. The target image is directly attached to the eyeball by means of a contact lens assembly. The target is viewed through a powerful lens. The assembly is firmly attached to the eye by a suction device.

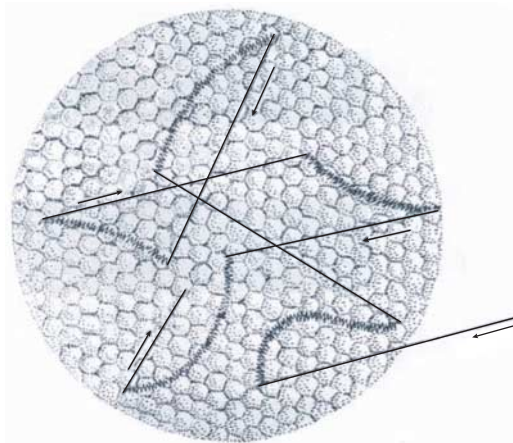


Figure 3 | **Fixational eye movements carry the image across the retinal photoreceptors.** High-frequency tremor is superimposed on slow drifts (curved lines). Microsaccades are fast jerk-like movements, which generally bring the image back towards the centre of vision (straight lines). The diameter of the patch of the fovea shown here is 0.05 mm. Reproduced, with permission, from REF. 21 © (1961) Scientific American, Inc.

Microsaccades. Fixational microsaccades, also called 'flicks' in early studies, are small, fast, jerk-like eye movements that occur during voluntary fixation. They carry the retinal image across a range of several dozen²⁴ to several hundred⁴⁰ photoreceptor widths, and are about 25 ms in duration⁴¹ (TABLES 3 and 4). Microsaccades cannot be defined on the basis of amplitude alone, as the amplitude of voluntary saccades can be as small as that of fixational microsaccades. In this review, we use the term microsaccade to refer to the small involuntary saccades that occur during fixation, sometimes called fixational saccades⁴². Microsaccades have been described in several species other than humans²⁵. However, they seem to be most important in species with foveal vision (such as monkeys and humans). Microsaccades in the macaque monkey are quite similar to those in humans⁴³⁻⁴⁵.

MAIN SEQUENCE
The linear correlation between saccadic speed and amplitude.

One of the possible roles of microsaccades is to correct displacements in eye position produced by drifts^{9,31,32}. The probabilities of occurrence, direction and amplitude of microsaccades are related to previous displacements of the image over the retina. For example, if drifts carry the fixation target away from the fovea, microsaccades will tend to bring the target back. No comparable correlation has been found for drifts³². The accuracy of potentially correctional microsaccades is nevertheless limited, and non-corrective microsaccades also occur^{24,46}. Recent studies indicate that microsaccades might help to counteract receptor adaptation on a short timescale and to correct fixation errors on a longer timescale⁴⁷.

The relationship between microsaccade velocity and amplitude (length) follows the 'MAIN SEQUENCE', similar to large saccades^{40,48,49}. It has therefore been proposed⁴⁸ that microsaccades and large saccades might be generated by the same mechanisms (that is, by circuits leading to saccade-related burst neurons in the superior colliculus^{50,51}). No correlation has been shown between visual acuity and changes in microsaccade amplitude⁴⁹. BOX 2 describes the production of 'saccades' in a subject without eye movements.

Microsaccades in the two eyes have generally been found to be conjugate^{9,31,49,52}. As microsaccades are performed involuntarily, this might indicate a subcortical control mechanism for microsaccade production⁴⁹. The problem of the binocular coordination of microsaccades has just begun to be investigated, and recent studies have distinguished between monocular and binocular microsaccades, with potentially different functional roles⁵³.

The role of microsaccades in perception
The function of microsaccades during visual fixation has been the subject of debate for more than thirty years. Cornsweet originally proposed that microsaccades serve the purpose of returning the eyes to the fixation target, and therefore correct the intersaccadic drifts of the eye³². It was also postulated that microsaccades probably have

Table 1 | Characteristics of tremor in humans

Amplitude	Tremor frequency	Duration	Maximum speed	Mean speed	Conjugate	Ref.
-	Up to almost 150 Hz, with peaks at 0-25 Hz and 60-90 Hz in both eyes	-	-	-	Yes	27
-8.5 s*	40-100 Hz	-	-	-	-	38
-	-	-	-	-	Yes	116
5-30 s (<1 min)	50-100 Hz	-	-	-	-	55
20-40 s	70-90 Hz	-	20 min s ⁻¹	-	-	9
9 s (median for one subject)	63 Hz (median for one subject)	-	-	-	-	117
10-30 s	30-80 Hz	-	18 min s ⁻¹ (maximum acceleration 20 deg s ⁻²)	-	-	31
10-20 s	30-100 Hz	-	-	-	-	7
About 15 s	Up to 90 Hz	-	-	-	No	30
0-2 min (rarely >1 min; median value 17.5 s)	30-70 Hz	-	-	-	-	24
1 min (mean) [†]	50-100 Hz	-	-	-	-	118

The different measurements obtained by different laboratories can be largely attributed to differences in recording methods^{31,116}. *, vector values from H-components — a conversion factor of $\sqrt{2}$ has been assumed; †, recalculated in Ref. 24.

an “important role in maintaining vision by counteracting retinal fatigue”^{11,34}. It was found that, after stabilizing a retinal image, superimposing eye movements similar to microsaccades could restore perception, whereas imposed movements of the image similar to drifts or tremor had a much smaller effect in preventing fading^{11,25}.

However, not all studies agreed with this result^{12–14}, and other suggestions for the role of microsaccades were proposed. Ditchburn and Foley-Fisher found microsaccades to be necessary to perceive hue differences at low contrast⁴¹, and Westheimer⁵⁴ suggested that microsaccades enhance stereoscopic hyperacuity. Carpenter²⁵ postulated that, of the three types of fixational eye movement, only microsaccades might contribute significantly to the maintenance of vision, as drift velocities are too low, and the amplitude and frequency of tremor would make it more detrimental than beneficial.

From the late 1960s and through the 1970s, a lively discussion on the importance of microsaccades for the maintenance of vision took place. Its main protagonists were Ditchburn, who argued that microsaccades have an essential role in normal vision, and Steinman, who argued that microsaccades serve no useful purpose. The strongest evidence against a role for microsaccades in preserving visual perception was twofold. First, trained subjects can suppress their microsaccades for several

seconds without fading of the images^{35,46,55}. Second, microsaccades are naturally suppressed in normal vision while subjects perform high-acuity tasks, such as shooting a rifle or threading a needle^{56–59}.

Ditchburn replied⁴¹ that the fact that subjects can learn to perform certain visual tasks without microsaccades does not contradict the essential role of microsaccades in normal vision. He argued further against Kowler and Steinman’s view⁵⁸ that microsaccades were an evolutionary mystery. In Ditchburn’s words: “Some acrobats can walk on their hands with amazing agility . . . Yet no one suggests, from these facts, that it is mysterious that feet have evolved.” Ditchburn’s letter was subsequently contested by Kowler and Steinman⁶⁰, who reiterated the uselessness of microsaccades and stated that these movements were perhaps “merely a kind of nervous tic.”

The debate ceased, unresolved, in the early 1980s and was revived in the second half of the 1990s. It was proposed that microsaccades could bring stationary stimuli in and out of RECEPTIVE FIELDS during free viewing, and therefore produce transient neural responses^{61,62}. It was further suggested that microsaccades could account for much of the response variability of neurons in visual area V1 of the awake monkey⁶³. The discussion was re-opened with a new focus — understanding the neurophysiological properties of microsaccade-induced activity. Microsaccades were found to generate neural

RECEPTIVE FIELD
The area of the sensory space in which stimulus presentation leads to the response of a particular sensory neuron.

Table 2 | Characteristics of drifts in humans and primates

Amplitude	Drift frequency	Duration	Maximum speed	Mean speed	Conjugate	Ref.
Human studies						
3–12 min	–	0.2–1 s	–	–	Yes	117
Rarely > 8 min	–	0.18–3.67 s (mean 0.8 s)	–	–	–	119
31.4 min (mean)*	–	–	–	24.6 min s ⁻¹	–	120
4.8–6.4 min (means for two subjects)	–	–	–	–	–	102
1.5–3.7 min (medians for two subjects)	–	–	–	–	–	121
–	95–97% fixation time	0.3–0.8 s	30 min s ⁻¹	6 min s ⁻¹	No	9
1.5–3.7 min (median values for different subjects) [†]	–	–	–	–	–	122
2–15 min (mean 6 min)	–	–	–	–	–	46
1.8–2.6 min (medians for two subjects)*	–	–	–	–	–	100
1.2–1.6 min (means for two subjects); 0.8–1.1 min (medians for two subjects) [‡]	–	–	–	–	–	34
–	–	–	–	–	No	37
1.5–4.0 min (medians for two subjects) [†]	–	–	–	–	–	33
Up to 6 min	–	–	–	–	Yes	31
Up to 5 min	–	–	–	–	–	24
Primate studies						
–	–	–	–	6 min s ⁻¹ (mean for one monkey)	–	65
–	–	–	–	0.42–11.91 min s ⁻¹ (means for four monkeys) [§]	–	43

*, vector values from H-components – a conversion factor of $\sqrt{2}$ has been assumed; †, recalculated in/taken from Ref. 127; ‡, calculated from horizontal and vertical components.

Table 3 | Characteristics of microsaccades in humans

Amplitude	Intersaccadic interval/ microsaccadic frequency	Duration	Max. speed	Mean speed	Conj.	Ref.
5–120 min (range monocular); ~15 min (median monocular); 6–120 min (range conjugate binocular); ~30 min (median conjugate binocular)*	0.51 Hz (mean monocular); 1.43 Hz (mean conjugate binocular)	–	–	6–120 deg s ⁻¹ (range monocular); ~18 deg s ⁻¹ (median monocular); 10–120 deg s ⁻¹ (range conjugate binocular); ~25 deg s ⁻¹ (median conjugate binocular)*	Yes	53
–	0.8–1.9 Hz (among different subjects)	–	–	–	Yes	47
32 min (mean)	1.3 ± 0.7 Hz (among different subjects)	–	–	–	–	70
12.7–60 min (range); ~42 min (median) ^{†,*}	–	–	–	11.3–127.3 deg s ⁻¹ (range); ~28.3 deg s ⁻¹ (median) ^{†,*}	–	69
12.7–65.9 min	0.23–0.93 Hz (mean 0.61 Hz)	–	28–97 deg s ⁻¹	16–40 deg s ⁻¹ (mean acceleration 2,322–6,440 deg s ⁻²)	Yes	49
<12 min	–	–	–	–	Yes	123
17 min (mean) [§]	–	–	–	–	–	124
5–32 min	2.1 Hz	–	~4.2–55 deg s ^{-1†}	–	Yes	117
4.2–53 min	1–3 Hz	–	21.3–36.5 deg s ⁻¹	–	–	119
20.8 min (mean) [†]	2.62 Hz (mean)	–	–	–	–	120
–	1.3–1.43 Hz (means for two subjects)	–	–	–	–	125
~7 min (mean)	~2 Hz (mean)	–	–	–	–	56
7.7 min (median)	0.6 s (median)	–	–	–	–	43
7.7–8.9 min (means for two subjects)	2.1–2.5 Hz (means for two subjects; range 1–4 Hz)	–	–	–	–	102
~40 min (mean)	–	–	–	–	–	55
1.8–5.8 min (medians for two subjects)	0.24–0.44 s (mean range for two subjects)	–	–	–	–	121
8.18 min (mean)	1.44 Hz [¶]	–	–	–	–	35
2–50 min	3–5% fixation time	0.01–0.02 s	–	10 deg s ⁻¹	Yes	9
3.7–9 min (median values for different subjects) [¶]	0.20–0.72 s (mean values for different subjects) [¶]	–	–	–	–	122
–	1.5 s (mean for one subject)	–	–	–	–	46
–	1.45–2.2 Hz (means for two subjects)	–	–	–	–	100
2–13 min*	–	–	–	3–14 deg s ^{-1*}	–	48
1.7–4.8 min (means for two subjects); 1.2–3.4 min (medians for two subjects) [¶]	–	–	–	–	–	34
2.5–5.6 min (median range for two subjects) [¶]	–	–	–	–	Yes	37
2–3.5 min (medians for two subjects) [¶]	0.45–0.5 s (means for two subjects); 0.35–0.4 s (medians for two subjects)	–	–	–	–	33
~3.5 min*	~0.4 Hz*	–	–	–	–	32
1–20 min	0.03–5 s	0.025 s	Max. acceleration 1,000 deg s ⁻²	10 deg s ⁻¹	Yes [#]	31
2–25 min	0.76–10 s (means for different subjects)	0.02–0.03 s	–	–	Yes	52
2.2–25.8 min (rarely > 10 min; mean 5.6 min; median 3.9 min) [¶]	0.2–4 s	0.02 s	–	–	–	24
3–14 min	1.5–2 Hz (mean frequencies for two subjects)	0.02–0.03 s	–	–	–	126
12.5–17.5 min (mean 8 min [¶] ; median 5.6 min [¶])	1 Hz (mean frequency)	–	–	–	–	118

*, values estimated from plots; †, vector values from H-components — a conversion factor of $\sqrt{2}$ has been assumed; §, calculated from horizontal and vertical components; ¶, recalculated in/taken from Ref. 127; ¶, microsaccade rates for 'large' and 'small' targets have been averaged; #, small differences in amplitude and direction were observed in one out of two subjects; Conj., conjugate.

Table 4 | Characteristics of microsaccades in primates

Amplitude	Intersaccadic interval/ microsaccadic frequency	Duration	Max. speed	Mean speed	Conj.	Ref.
8.4–16.2 min (means for two monkeys)	2.3–2.5 Hz (mean frequencies for two monkeys)	At least 8 ms	–	–	–	42
~40 min (mean)	–	–	–	~30 deg s ⁻¹	–	67
~20 min (mean)	~3–5 Hz	29 ms (mean)	–	~30 deg s ⁻¹	–	40
48 min (mean)	0.3–1.4 Hz (means for two monkeys)	25 ms (mean)	9–110 deg s ⁻¹ (median 40 deg s ⁻¹)	~30 deg s ⁻¹	–	65
10.1 min (median)	0.597 s (median)	20 ms (mean)	–	–	–	66
9.9–40.3 min (medians for four monkeys)	0.8–7.4 s (medians for four monkeys)	–	–	–	–	43
40 min (mean; minimum amplitude 23 min)	–	–	–	–	–	55

Conj., conjugate.

responses in every visual area examined by researchers^{29,40,64–68}, indicating a potentially important physiological role for microsaccades. Moreover, microsaccades were linked to fixation correction, control of BINOCULAR DISPARITY⁴⁷ and attentional shifts^{69–71}, but not all studies concurred^{72,73}.

It seems probable that all fixational eye movements are important for the maintenance of vision, and that their relative contributions depend on stimulation conditions. Gerrits and Vendrik⁷⁴ and Clowes⁷⁵ found that both drifts and microsaccades were necessary for optimal vision. Receptive fields near the fovea might be so small that drifts and tremor are sufficient to prevent visual fading in the absence of microsaccades. On the other hand, it is quite possible that if drifts and tremor were eliminated altogether, microsaccades would then suffice to sustain vision during fixation. Receptive fields in the periphery might be so large that only microsaccades are large and fast enough (compared to drifts and tremor) to prevent visual fading, especially with low-contrast stimuli^{40,41,74}.

In summary, it remains unresolved whether microsaccades contribute significantly to the maintenance of visibility and, if so, whether they have a specific function in visual perception; that is, does the role of microsaccades differ from those of drifts and tremor? Are microsaccades more important for peripheral vision, whereas drifts and tremor maintain foveal vision when

microsaccades are suppressed during specific tasks? It seems clear that microsaccades lead to neural activity in the visual pathway. Microsaccade-induced neural activity would be difficult to explain if it did not affect perception. The type of activity induced by microsaccades is qualitatively similar to neural activity that correlates with the perception of visibility during VISUAL MASKING^{62,76} and other visual illusions⁷⁷. However, a direct correlation between microsaccade-induced activity and visual perception has yet to be shown.

Neuronal responses during visual fixation

Decrypting the NEURAL CODE is crucial to understanding virtually all brain function. If we assume that the fundamental action of the visual system is to generate visible percepts, and if we consider that fixational eye movements are correlated with the maintenance of visibility during fixation, it follows that, by measuring the neuronal responses that are produced by fixational eye movements, we might determine the encoding strategy that is used by the visual system to generate the perception of visibility. The neural responses to fixational eye movements have been recently studied in the retina²⁸, lateral geniculate nucleus (LGN)^{29,68}, area V1^{29,40,66,67} (FIGS 4 and 5) and extrastriate cortex^{65,66}. During these experiments, macaque monkeys are usually trained to fixate their gaze on a small fixational spot while a stationary stimulus of optimal characteristics (for example, a bar with optimal dimensions and orientation when recording from area V1) is placed over the receptive field of the neuron that is being recorded. Fixational eye movements are then correlated with subsequent neural activity. Because the visual stimulus does not move and the head is fixed, modulation of neural activity occurs only when fixational eye movements move the visual receptive field in and out of the stationary stimulus.

Neural responses to microsaccades. It is generally agreed that fixational eye movements, especially microsaccades, have predominantly excitatory effects at all levels of the visual system^{28,29,40,65,67,68}. In the LGN and area V1, neuronal responses after microsaccades are visual in nature — microsaccades lead to an increase in neural activity when a stationary bar of light is centred

BINOCULAR DISPARITY

The difference in gaze position of the two eyes that gives rise to stereovision.

VISUAL MASKING

An illusion in which a normally visible target object is rendered invisible by a mask object.

NEURAL CODE

The language, expressed as a pattern of neuronal impulses, that neurons use to send information to each other.

Box 2 | Vision without eye movements: a clinical case

A.I. is a young woman who has never made eye movements; she has extraocular muscular fibrosis. Despite her condition, her visual perception is surprisingly normal — she can read at normal speed and she can perform complicated visuomotor tasks, such as making a cup of tea, with no problems. The strategy that A.I. uses is to move her head in a 'saccadic' fashion (with both voluntary and automatic saccades) which seems to compensate for the absence of eye movements. The saccadic movements of A.I.'s head closely resemble the saccadic eye movements of normal subjects, indicating that the same saccade-generating circuits that operate in normal subjects (neurons in the superior colliculus) might participate in generating A.I.'s head-saccades. Although studies of A.I. have not addressed fixational eye movements *per se*, they indicate that saccadic movements, either of the head or of the eye, might be the optimal sampling method for the brain, as compared, for example, to smooth scanning of the visual scene^{111–113}. Studies of A.I. might also help to resolve an old debate on the importance of head movements versus fixational eye movements for visual perception^{8,114}, as they indicate that in the absence of eye movements, normal head movements alone might not suffice to maintain vision.

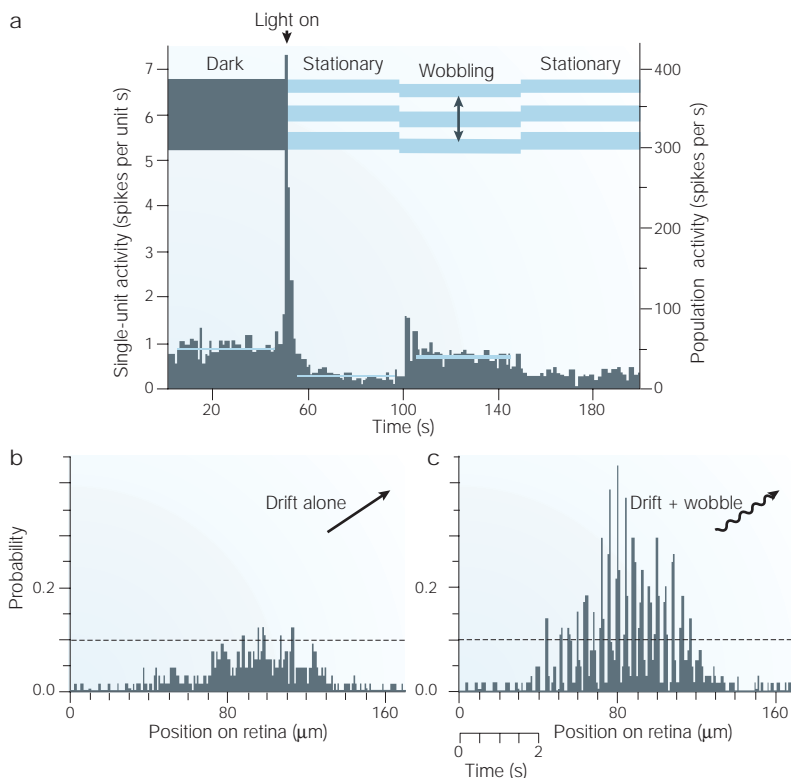


Figure 4 | Fixational eye movements increase retinal activity. **a** | Continuous recording from 54 retinal ganglion cells in the turtle during darkness, after switching on a stationary grating, and during wobbling of this grating. The grating was wobbled to simulate periodic (tremor-like) eye movement. **b** | Responses of a single ganglion cell to a drifting contrast border of velocity comparable to ocular drifts. **c** | When the drift is superimposed on wobbling, the responses of the cell shown in **b** markedly increase. Reproduced, with permission, from REF. 28 © (2002) Macmillan Magazines Ltd.

EXTRARETINAL ACTIVATION
Responses in the visual system that occur in the absence of visual stimuli (such as one might see due to feedback from motor areas).

SPATIAL SUMMATION
The way in which non-overlapping retinal stimulation is integrated within dendrites to produce a stronger neuronal response.

BURSTS
Clusters of action potentials.

TEMPORAL SUMMATION
The way in which non-simultaneous synaptic events combine in time. One of the basic elements of synaptic integration.

LONG, TIGHT BURST
A type of burst consisting of a large number of spikes that occur in rapid succession.

over the neuron's receptive field. However, when the bar is removed from the receptive field (and the monitor facing the monkey is blank except for the fixation point), microsaccades do not lead to changes in neural activity. This shows that microsaccade-induced activity in early visual neurons is visual, rather than motor, because these neurons are excited only when their receptive fields sweep across stationary stimuli and not during equivalent action by the motor system in the absence of a visual stimulus^{29,40} (FIG. 5a). However, the debate on the existence of EXTRARETINAL ACTIVATION owing to microsaccades is not settled. Leopold and Logothetis⁶⁶ reported that extraretinal influences cause decreases in V1 activity in response to microsaccades, whereas Snodderly *et al.*⁶⁷ claimed that these extraretinal influences lead to increases in V1 activity (however, they found this in only one subject). In summary, some studies show excitation, some show inhibition, and some show no effect in response to microsaccades in the absence of visual stimuli.

Presumably, fixational eye movements first generate neural activity at the level of retinal photoreceptors, by moving their receptive fields over otherwise stationary stimuli. This photoreceptor activity would then be transmitted (perhaps without substantial transformation for the first several steps) to subsequent levels in the visual hierarchy.

To investigate the effectiveness of microsaccades in generating neural activity, neural responses induced by microsaccades have been compared with neural responses induced by flashing bars (a type of visual stimulus commonly used in visual research). Onset responses to flashing bars in the LGN and area V1 are about seven times larger than the responses to stationary bars moved across the neurons' receptive fields by microsaccades, perhaps because of the relative abruptness of flashes as stimuli²⁹. No similar comparison has been made for drifts or tremor.

There has been speculation that microsaccades might help to disambiguate latency and brightness in visual perception, allowing us to use latency in our visual discriminations⁴⁰. Changes in contrast can be encoded as changes in the latency of neuronal responses^{78–80}. But how can the brain use the latency information as a code for contrast, without first knowing the timing of events? In principle, microsaccades could solve this problem and be used by the brain to measure latency. As the brain knows when a microsaccade is generated, differential latencies in visual responses could be used by the brain to indicate differences in contrast and salience.

Microsaccades could enhance SPATIAL SUMMATION by synchronizing the activity of neurons with neighbouring receptive fields^{40,66}. Increases in neural firing after microsaccades usually occur in 'clumps', or BURSTS of spikes. Bursts are better correlated with previous microsaccades than single spikes or instantaneous firing rate. So, transient bursts of spikes are a more reliable code for stimulus visibility than single spikes or fast rates of firing, as bursts are best correlated with microsaccades, and microsaccades, as with all fixational eye movements, are themselves correlated with visibility⁴⁰. Similar transient bursts of spikes are also correlated with stimulus visibility during visual masking illusions. It would be difficult to imagine a system in which transient bursts were the code for visibility in one case, but not in the other^{62,76}.

By generating bursts of spikes, microsaccades might also enhance TEMPORAL SUMMATION of responses from neurons with neighbouring receptive fields⁴⁰. Synaptic facilitation for closely spaced pairs of spikes has been shown *in vivo* in retinogeniculate synapses, and in disinaptic connections from the retina to the visual cortex^{81,82}.

Bursts that are highly correlated with previous microsaccades are characterized by high spike numbers and short interspike intervals. So, LONG, TIGHT BURSTS of spikes are the type of activity that most effectively sustains a visible image^{29,40}. Moreover, the optimality of the stationary visual stimulation has an effect on the size of bursts after microsaccades. When the stationary stimulus over the neuron's receptive field has optimal characteristics (for instance, an optimally oriented bar of light), microsaccades during fixation generate long bursts. By contrast, when the stimulus on the receptive field has non-optimal characteristics, microsaccades induce shorter bursts. So, long bursts are correlated with salient, optimal stimuli, whereas short bursts are correlated with non-optimal visual stimulation²⁹ (FIG. 5c).

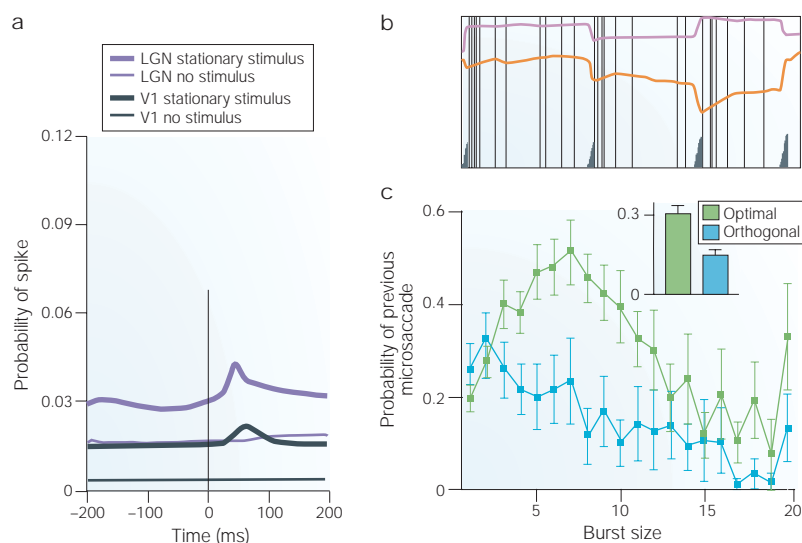


Figure 5 | Neural responses to microsaccades in the primate. **a** | Microsaccades increase spike probabilities in neurons of the lateral geniculate nucleus (LGN) ($n = 57$) and visual area V1 ($n = 308$) of the awake primate. Time 0 indicates start time for all microsaccades ($n = 1, 246, 791$). In the absence of visual stimulation, microsaccades do not generate spikes in the LGN ($n = 42$) or in V1 ($n = 37$). Modified, with permission, from REF. 29 © (2002) National Academy of Sciences. **b** | Correlation between microsaccades and bursts in primate area V1. The pink and orange traces represent horizontal and vertical eye positions, respectively. The triangles at the bottom indicate occurrence of a microsaccade (the height of the triangles represents microsaccade amplitudes). The vertical black lines represent the spikes of a single V1 neuron. Microsaccades tend to be followed by a rapid burst of spikes. Reproduced, with permission, from REF. 40 © (2000) Macmillan Magazines Ltd. **c** | The size (number of spikes) of bursts following microsaccades depends on the type of visual stimulation that is presented to the neuron's receptive field. Optimal stimuli (in this case, optimally oriented bars of light) lead to longer bursts; non-optimal stimuli (bars of light with orientations that are orthogonal to the optimal) lead to shorter bursts. Reproduced, with permission, from REF. 29 © (2002) National Academy of Sciences.

Neural responses to drifts. Neural responses to drifts have received considerably less attention than neural responses to microsaccades in recent studies. Part of the reason for this is that drifts are more difficult to characterize objectively than microsaccades (which are more easily detected by automatic algorithms that combine amplitude and velocity thresholds). So, drifts have usually been identified indirectly as the eye-position changes that occur during periods between microsaccades. This method has the potential flaw of unintentionally attributing non-drift related activity (such as undetected tremor or specific relationships between the stimulus and the receptive field) to drifts⁶⁷. Snodderly *et al.*⁶⁷ concluded that drifts caused increases in firing in a subset of V1 neurons. Gur *et al.*⁶³ found that drifts caused less variability in neuronal responses in V1 than a combination of drifts and microsaccades.

Neural responses to tremor. Greschner *et al.*²⁸ have recently shown that ganglion cells in the turtle retina respond strongly to a simulation of periodic, tremor-like fixational eye movements, whereas simulated drift movements have little effect (there are no microsaccadic eye movements in the turtle). Moreover, neurons with receptive fields located along contrast borders are synchronized and their activity reliably indicates preceding movements. This synchronization of retinal activity

could potentially be used by the brain to improve the estimation of stimulus features, such as spatial frequency²⁸. Future physiological studies should address the neuronal responses to tremor (and to tremor versus drifts) in the primate visual system.

A continuum of responses to eye movements. Snodderly *et al.*⁶⁷ reported three different groups of neurons in area V1, classified according to their responses to eye movements during fixation: microsaccade-activated cells; position/drift-activated cells; and mixed cells (which were activated by both drifts and microsaccades). The authors found that these response categories fell on a continuum of transient/sustained neuronal responsiveness in such a way that transient neurons responded best to the fast stimulation provided by microsaccades and sustained neurons responded best to the slow stimulation that occurred between microsaccades (such as drifts). Most neurons fell in the middle of the transient/sustained continuum and responded in a mixed fashion.

Perceptual stability during fixation
Why does the world remain perceptually stable when we fixate, despite the continual motion caused by fixational eye movements? Even if we were to account for tremor by its being too small and too fast to notice, and for drifts by their being too slow to notice, there is no doubt that we can easily see small shifts in the visual field that are equivalent in size and speed to microsaccades. It has been suggested that there must be a neural mechanism that accounts for these eye movements and excludes them from our perception. This hypothetical mechanism is called 'microsaccadic suppression'; that is, the suppression of neural firing that is associated with the occurrence of a microsaccade.

Suppression of neural firing during large saccades is known to exist^{83–87}, but the existence of microsaccadic suppression has been more controversial. Some studies have reported elevation of visual thresholds^{26,88}, but others have found little or no threshold elevation during microsaccades^{89,90}. In the early visual system (LGN, area V1), microsaccades generate increases in neural activity, but not suppression^{29,40} (FIG. 5a). A theoretical model proposed by Rucci *et al.*⁹¹ argues that fixational eye movements might be important for the generation of orientation selectivity during development. Because orientation selectivity occurs first in V1, this model supports the idea that microsaccadic suppression would occur after V1. The issue is not resolved, however.

Oliveczky *et al.*⁹² have recently shown that a subset of ganglion cells in the rabbit and salamander retinas show microsaccadic suppression. As this subset of cells is restricted to the motion pathway, it might mean that this mechanism does not exist in the primate retina (in which directionally selective retinogeniculate neurons have not been found). Furthermore, microsaccadic suppression failed in all retinal ganglion cells when the authors presented stimuli on a blank background, whereas perceptual suppression of microsaccades clearly occurs on presentation of sparse stimuli to humans. So, although the results of Oliveczky *et al.*

might help to explain how a moving foreground is parsed from a complex background⁹³, it is hard to imagine that microsaccadic suppression in humans occurs at the level of the retina.

Other potential mechanisms for microsaccadic suppression include a common motor-to-sensory feedback mechanism (for both saccadic and microsaccadic suppression), probably located at the level of the brainstem ocular motor nuclei^{94,95}. But Murakami and Cavanagh have suggested that the suppression mechanism for fixational eye movements is based solely on visual motion signals⁹⁶. They have developed a remarkable visual illusion — visual jitter — that arises when our visual system fails to compensate for fixational eye movements. As the name of the illusion indicates, in the absence of fixational eye movement compensation, we can easily notice our own fixational eye movements, and the world is seen as unstable and jittery.

Murakami and Cavanagh have proposed that this suppression system determines the region within the stimulus that has the minimum instantaneous velocity vector (which would be due to the contribution of eye movements only). This baseline velocity vector could be subtracted from the velocities of all points in the scene⁹⁶ to account for the effects of fixational eye movements. The extrastriate cortex, especially the middle temporal region (MT)^{97,98}, could be the locus for such a compensatory mechanism. However, physiological studies indicate that microsaccades induce strong excitatory responses in macaque area MT⁶⁵. This seems to contradict a specific role for MT in microsaccadic suppression, although we cannot rule out that the responses in MT might drive a microsaccadic-suppression system later in the visual hierarchy — the question remains open.

Modulation of fixational eye movements

As fixational eye movements seem to enhance the visibility of the world, a pertinent question is whether environmental factors (such as dim light levels) or cognitive factors (such as the level of attention) can control the generation of fixational eye movements.

Visual modulation. If the role of fixational eye movements is to prevent visual fading, we might expect the magnitude or the frequency of fixational eye movements to increase in response to a decrease in visibility⁵⁵. However, microsaccades are less frequent in the dark and during retinal stabilization^{32,45}. But there might be some effect of environmental factors on the generation of fixational eye movements, because microsaccades tend to become larger in the dark that is, under SCOTOPIC CONDITIONS^{31,32,45}, and drifts are both larger and more frequent under the same conditions^{31,34}. The increased amplitude of fixational eye movements in the dark might be accounted for by the fact that the eyes cannot maintain accurate fixation in complete darkness, and that a visual target is crucial to normal fixation³². Horwitz *et al.*⁴² showed that the onset of peripheral stimuli evokes microsaccades with a latency of ~70 ms. It might have been expected that, if the role of microsaccades is to increase the visibility of

the stimulus, turning peripheral stimuli off (and not on) should evoke microsaccades.

Precise fixation depends on the luminance of the background of the fixation spot^{45,99}, as dark backgrounds lead to a fixational upshift; that is, the eyes fixate above the fixation spot. This upshift is mainly driven by microsaccades, and depends solely on the background luminance — it is not evoked by low levels of contrast between the fixation spot and its background. This effect cannot be explained by scotopic foveal blindness, as the targets in these experiments were bright and well within PHOTOPIC CONDITIONS.

There is some evidence that the parameters of the fixation target, such as its size¹⁰⁰, shape³⁶, colour¹⁰¹ or eccentricity¹⁰², can have a small influence on the amplitude, direction and/or retinal disparity of fixational eye movements.

Attentional modulation. Attentional and oculomotor processes are greatly intertwined in the brain¹⁰³. Visual modulation of microsaccades could therefore be the by-product of attentional modulation driven by visual inputs. Barlow¹⁰⁴ proposed that microsaccades represented shifts in visual attention, and several recent studies have addressed the role of attention in microsaccade dynamics. Engbert and Kliegl⁷⁰ reported an initial decrease in microsaccade rate, followed by an increase, in response to shifts in visual attention. Moreover, the average direction of microsaccades is correlated with the direction of attention⁶⁹⁻⁷¹. These results challenge the interpretation of microsaccades as strictly low-level oculomotor phenomena⁷⁰. However, Tse *et al.*^{72,73} have shown that when the goal of the task is to maintain very accurate fixation^{56,59,105}, sudden stimulus onsets, which capture attention, have no influence on the frequency or characteristics of microsaccades and drifts.

Other cognitive factors. Although microsaccades are generally considered to be involuntary movements, it has been reliably shown that humans and primates can be trained to suppress their microsaccades for several seconds^{35,46,55}. However, microsaccades cannot be elicited voluntarily, unlike the larger voluntary or exploratory saccades. It therefore remains possible that different neural mechanisms are involved in the generation of voluntary saccades and involuntary microsaccades.

Conclusions

Most of our viewing is conducted while we fixate our gaze. Fixational eye movements not only help to keep objects of interest in the centre of our vision, but also prevent sensory adaptation in our visual path by refreshing our retinal images. Without fixational eye movements, we would be blind during visual fixation, and the world would become visible only when we moved our eyes voluntarily, when we moved our heads or when the world moved in front of us. As fixational eye movements induce firing of visual neurons in response to stationary objects, they ensure that we retain our vision during fixation.

SCOTOPIC CONDITIONS

Dim light conditions in which only the rods of the retina are active.

PHOTOPIC CONDITIONS

Bright light conditions in which only the cones of the retina are active.

The neural responses that are generated during this process are similar to the responses that would be generated if we moved a visual stimulus over the stationary receptive field of a visual neuron (although completely stationary receptive fields can only be produced in the laboratory). Qualitatively similar activity is also correlated with the visibility of stimuli during visual masking. The neural responses that are best correlated with fixational eye movements are clustered in a specific manner, forming long, tight bursts. These bursts are an important clue to the language that our brain uses to represent the visibility of the world and the salience of specific visual features. So, although the debate on the significance of microsaccades, drifts and tremor for our perception might rage on, we would certainly have a greater mystery at hand if it was found that all of the robust neural activity induced by microsaccades did not lead to correlated perception.

The field is moving in the direction of further recording the neural responses that correlate with fixational eye movements. Following this trend, the field promises to discover the underpinnings of fixational eye movements and the neural responses they generate: the neural circuits that control fixational eye movements; the neural correlates of microsaccadic suppression; the physiological mechanisms that account for the effects of

cognitive factors during fixation; the biophysical explanation of why long, tight bursts are best correlated with microsaccades; and the neural correlates of tremor and drifts, and their relative contributions to visibility during fixation in comparison to microsaccades.

Several techniques under development will further improve our understanding of fixational eye movements and the neural activity that maintains the visibility of a scene. These techniques include implementing simultaneous psychophysical tests during the recording of neural responses to fixational eye movements, and the development of fast and reliable non-invasive retinal stabilization methods to be used in humans and primates. Once these techniques are perfected, it will become possible, for the first time, to carry out the crucial experiment that will end the debate on the importance of microsaccades and the other fixational eye movements to visual perception. This experiment will include the accurate recording of microsaccades, drifts and tremor during visual fixation, the superimposition of each type of eye movement during fast retinal stabilization conditions, and the recording of neural responses to superimposed microsaccades, drifts or tremor at multiple levels in the visual pathway during a perceptual task in which the subject reports the perceptual state of the stimulus — visible or invisible.

1. Riggs, L. A. & Ratliff, F. The effects of counteracting the normal movements of the eye. *J. Opt. Soc. Am.* **42**, 872–873 (1952).
This classic paper, together with references 2 and 9, showed that when all eye movements are counteracted in the laboratory, visual perception rapidly fades owing to sensory adaptation.
2. Ditchburn, R. W. & Ginsborg, B. L. Vision with a stabilized retinal image. *Nature* **170**, 36–37 (1952).
3. Judge, S. J., Richmond, B. J. & Chu, F. C. Implantation of magnetic search coils for measurement of eye position: an improved method. *Vision Res.* **20**, 535–538 (1980).
4. Hartline, H. K. The nerve messages in the fibers of the visual pathway. *J. Opt. Soc. Am.* **30**, 239–247 (1940).
5. Kuffler, S. W. Neurons in the retina: organization, inhibition and excitation problems. *Cold Spring Harb. Symp. Quant. Biol.* **17**, 281–292 (1952).
6. Hubel, D. H. & Wiesel, T. N. Receptive fields and functional architecture in two non-striate visual areas (18 and 19) of the cat. *J. Neurophysiol.* **28**, 229–289 (1965).
7. Riggs, L. A., Ratliff, F., Cornsweet, J. C. & Cornsweet, T. N. The disappearance of steadily fixated visual test objects. *J. Opt. Soc. Am.* **43**, 495–501 (1953).
8. Skavenski, A. A., Hansen, R. M., Steinman, R. M. & Winterson, B. J. Quality of retinal image stabilization during small natural and artificial body rotations in man. *Vision Res.* **19**, 675–683 (1979).
9. Yarbus, A. L. *Eye Movements and Vision* (Plenum, New York, 1967).
10. Krauskopf, J. Effect of retinal image motion on contrast thresholds for maintained vision. *J. Opt. Soc. Am.* **47**, 740–744 (1957).
11. Ditchburn, R. W., Fender, D. H. & Mayne, S. Vision with controlled movements of the retinal image. *J. Physiol. (Lond.)* **145**, 98–107 (1959).
12. Gerrits, H. J. & Vendrik, A. J. Artificial movements of a stabilized image. *Vision Res.* **10**, 1443–1456 (1970).
13. Sharpe, C. R. The visibility and fading of thin lines visualized by their controlled movement across the retina. *J. Physiol. (Lond.)* **222**, 113–134 (1972).
14. Drysdale, A. E. The visibility of retinal blood vessels. *Vision Res.* **15**, 813–818 (1975).
15. Gerrits, H. J., De Haan, B. & Vendrik, A. J. Experiments with retinal stabilized images. Relations between the observations and neural data. *Vision Res.* **6**, 427–440 (1966).
16. Ratliff, F. Stationary retinal image requiring no attachments to the eye. *J. Opt. Soc. Am.* **48**, 274–275 (1958).
17. Coppola, D. & Purves, D. The extraordinarily rapid disappearance of entoptic images. *Proc. Natl Acad. Sci. USA* **93**, 8001–8004 (1996).
This study showed that visual fading due to retinal stabilization can happen extremely quickly, in less than 80 ms. This fast fading of retinal images in the absence of eye movements has made the field reconsider the dynamics of neural adaptation during normal vision.
18. Kelly, D. H. New method of stabilizing retinal images. *J. Opt. Soc. Am.* **65**, 1184 (1975).
19. Gur, M. & Snodderly, D. M. Studying striate cortex neurons in behaving monkeys: benefits of image stabilization. *Vision Res.* **27**, 2081–2087 (1987).
20. Rucci, M. & Desbordes, G. Contributions of fixational eye movements to the discrimination of briefly presented stimuli. *J. Vis.* **3**, 852–864 (2003).
21. Pritchard, R. M. Stabilized images on the retina. *Sci. Am.* **204**, 72–78 (1961).
22. Barlow, H. B. Slippage of contact lenses and other artifacts in relation to fading and regeneration of supposedly stable retinal images. *Q. J. Exp. Psychol.* **15**, 36–51 (1963).
23. Helmholtz, H. *Helmholtz's Treatise on Physiological Optics* (ed. Southall, J. P. C.) (Gryphon Editions, Birmingham, 1985).
24. Ratliff, F. & Riggs, L. A. Involuntary motions of the eye during monocular fixation. *J. Exp. Psychol.* **40**, 687–701 (1950).
25. Carpenter, R. H. S. *Movements of the Eyes* (Pion, London, 1988).
26. Ditchburn, R. W. Eye-movements in relation to retinal action. *Opt. Acta (Lond.)* **1**, 171–176 (1955).
27. Spauschus, A., Marsden, J., Halliday, D. M., Rosenberg, J. R. & Brown, P. The origin of ocular microtremor in man. *Exp. Brain Res.* **126**, 556–562 (1999).
28. Greschner, M., Bongard, M., Rujan, P. & Ammermüller, J. Retinal ganglion cell synchronization by fixational eye movements improves feature stimulation. *Nature* **5**, 341–347 (2002).
Ganglion neurons from the turtle retina were stimulated by stimuli that moved in a fashion that simulated eye movements during fixation. Fixational eye movements not only led to increased activity of isolated retinal ganglion neurons, but also to increased synchronization in the retinal ganglion cell network.
29. Martínez-Conde, S., Macknik, S. L. & Hubel, D. H. The function of bursts of spikes during visual fixation in the awake primate lateral geniculate nucleus and primary visual cortex. *Proc. Natl Acad. Sci. USA* **99**, 13920–13925 (2002).
The parameters of bursts of spikes following microsaccades in the lateral geniculate nucleus and in area V1 depend on whether the visual stimuli presented are optimal or non-optimal for each of these areas.
30. Riggs, L. A. & Ratliff, F. Visual acuity and the normal tremor of the eyes. *Science* **114**, 17–18 (1951).
31. Ditchburn, R. W. & Ginsborg, B. L. Involuntary eye movements during fixation. *J. Physiol. (Lond.)* **119**, 1–17 (1953).
32. Cornsweet, T. N. Determination of the stimuli for involuntary drifts and saccadic eye movements. *J. Opt. Soc. Am.* **46**, 987–993 (1956).
33. Nachmias, J. Two-dimensional motion of the retinal image during monocular fixation. *J. Opt. Soc. Am.* **49**, 901–908 (1959).
34. Nachmias, J. Determiners of the drift of the eye during monocular fixation. *J. Opt. Soc. Am.* **51**, 761–766 (1961).
35. Steinman, R. M., Cunitz, R. J., Timberlake, G. T. & Herman, M. Voluntary control of microsaccades during maintained monocular fixation. *Science* **155**, 1577–1579 (1967).
36. St Cyr, G. J. & Fender, D. H. The interplay of drifts and flicks in binocular fixation. *Vision Res.* **9**, 245–265 (1969).
37. Krauskopf, J., Cornsweet, T. N. & Riggs, L. A. Analysis of eye movements during monocular and binocular fixation. *J. Opt. Soc. Am.* **50**, 572–578 (1960).
38. Eizenman, M., Hallett, P. E. & Frecker, R. C. Power spectra for ocular drift and tremor. *Vision Res.* **25**, 1635–1640 (1985).
39. Shakhnovich, A. R. & Thomas, J. G. Micro-tremor of the eyes of comatose patients. *Electroencephalogr. Clin. Neurophysiol.* **42**, 117–119 (1977).
40. Martínez-Conde, S., Macknik, S. L. & Hubel, D. H. Microsaccadic eye movements and firing of single cells in the striate cortex of macaque monkeys. *Nature Neurosci.* **3**, 251–258 (2000).
Fixational microsaccades during the presentation of stationary stimuli led to increases in the activity of primate area V1 neurons. Increases in neural firing after microsaccades were clumped in bursts of spikes.
41. Ditchburn, R. W. The function of small saccades. *Vision Res.* **20**, 271–272 (1980).
42. Horowitz, G. D. & Albright, T. D. Short-latency fixational saccades induced by luminance increments. *J. Neurophysiol.* **90**, 1333–1339 (2003).
43. Skavenski, A. A., Robinson, D. A., Steinman, R. M. & Timberlake, G. T. Miniature eye movements of fixation in rhesus monkey. *Vision Res.* **15**, 1269–1273 (1975).

44. Snodderly, D. M. & Kurtz, D. Eye position during fixation tasks: comparison of macaque and human. *Vision Res.* **25**, 83–98 (1985).
45. Snodderly, D. M. Effects of light and dark environments on macaque and human fixational eye movements. *Vision Res.* **27**, 401–415 (1987).
46. Fiorentini, A. & Ercoles, A. M. Involuntary eye movements during attempted monocular fixation. *Atti Fond. Giorgio Ronchi* **21**, 199–217 (1966).
47. Engbert, R. & Kliegl, R. Microsaccades keep the eyes' balance during fixation. *Psychol. Sci.* (in the press)
48. Zuber, B. L. & Stark, L. Microsaccades and the velocity–amplitude relationship for saccadic eye movements. *Science* **150**, 1459–1460 (1965).
- This classic study showed a linear relationship between microsaccade amplitudes and velocities. This followed the extrapolation of the same relationship for large saccades, and indicated that both large saccades and microsaccades might be generated by the same neural mechanisms.**
49. Moller, F., Laursen, M. L., Tygesen, J. & Sjolle, A. K. Binocular quantification and characterization of microsaccades. *Graefes Arch. Clin. Exp. Ophthalmol.* **240**, 765–770 (2002).
50. Wurtz, R. H. Vision for the control of movement. The Friedenwald Lecture. *Invest. Ophthalmol. Vis. Sci.* **37**, 2130–2145 (1996).
51. Sparks, D. L. The brainstem control of saccadic eye movements. *Nature Rev. Neurosci.* **3**, 952–964 (2002).
52. Lord, M. P. Measurement of binocular eye movements of subjects in the sitting position. *Brit. J. Ophthalmol.* **35**, 21–30 (1951).
53. Engbert, R. & Kliegl, R. in *The Mind's Eyes: Cognitive and Applied Aspects of Eye Movements* (eds Hyona, J., Radach, R. & Deubel, H.) 103–117 (Elsevier, Oxford, 2003).
54. Westheimer, G. The spatial sense of the eye. Proctor lecture. *Invest. Ophthalmol. Vis. Sci.* **18**, 893–912 (1979).
55. Steinman, R. M., Haddad, G. M., Skavenski, A. A. & Wyman, D. Miniature eye movement. *Science* **181**, 810–819 (1973).
56. Winters, B. J. & Collewijn, H. Microsaccades during finely guided visuomotor tasks. *Vision Res.* **16**, 1387–1390 (1976).
57. Kowler, E. & Steinman, R. M. The role of small saccades in counting. *Vision Res.* **17**, 141–146 (1977).
58. Kowler, E. & Steinman, R. M. Miniature saccades: eye movements that do not count. *Vision Res.* **19**, 105–108 (1979).
59. Bridgeman, B. & Palca, J. The role of microsaccades in high acuity observational tasks. *Vision Res.* **20**, 813–817 (1980).
60. Kowler, E. & Steinman, R. M. Small saccades serve no useful purpose: reply to a letter by R. W. Ditchburn. *Vision Res.* **20**, 273–276 (1980).
61. Livingstone, M. S., Freeman, D. C. & Hubel, D. H. Visual responses in V1 of freely viewing monkeys. *Cold Spring Harb. Symp. Quant. Biol.* **61**, 27–37 (1996).
62. Macknik, S. L. & Livingstone, M. S. Neuronal correlates of visibility and invisibility in the primate visual system. *Nature Neurosci.* **1**, 144–149 (1998).
63. Gur, M., Beylin, A. & Snodderly, D. M. Response variability of neurons in primary visual cortex (V1) of alert monkeys. *J. Neurosci.* **17**, 2914–2920 (1997).
64. Gur, M. & Snodderly, D. M. Visual receptive fields of neurons in primary visual cortex (V1) move in space with the eye movements of fixation. *Vision Res.* **37**, 257–265 (1997).
65. Bair, W. & O'Keefe, L. P. The influence of fixational eye movements on the response of neurons in area MT of the macaque. *Vis. Neurosci.* **15**, 779–786 (1998).
66. Leopold, D. A. & Logothetis, N. K. Microsaccades differentially modulate neural activity in the striate and extrastriate visual cortex. *Exp. Brain Res.* **123**, 341–345 (1998).
67. Snodderly, D. M., Kagan, I. & Gur, M. Selective activation of visual cortex neurons by fixational eye movements: implications for neural coding. *Vis. Neurosci.* **18**, 259–277 (2001).
68. Reppas, J. B., Usrey, W. M. & Reid, R. C. Saccadic eye movements modulate visual responses in the lateral geniculate nucleus. *Vision Res.* **35**, 961–974 (2002).
69. Hafed, Z. M. & Clark, J. J. Microsaccades as an overt measure of covert attention shifts. *Vision Res.* **42**, 2533–2545 (2002).
- This study, together with references 70 and 72, comprised the first systematic attempts to characterize the influence of cognition on the dynamics of fixational eye movements. These studies provided a rich basis for what is likely to become a lively field of enquiry.**
70. Engbert, R. & Kliegl, R. Microsaccades uncover the orientation of covert attention. *Vision Res.* **43**, 1035–1045 (2003).
71. Rolfs, M., Engbert, R. & Kliegl, R. Microsaccade orientation supports attentional enhancement opposite to a peripheral cue: commentary on Tse, Sheinberg, and Logothetis. *Psychol. Sci.* (in the press).
72. Tse, P. U., Sheinberg, D. L. & Logothetis, N. K. Fixational eye movements are not affected by abrupt onsets that capture attention. *Vision Res.* **42**, 1663–1669 (2002).
73. Tse, P. U., Sheinberg, D. S. & Logothetis, N. K. The distribution of microsaccade directions need not reveal the location of attention. *Psychol. Sci.* (in the press).
74. Gerrits, H. J. & Vendrik, A. J. The influence of stimulus movements on perception in parafoveal stabilized vision. *Vision Res.* **14**, 175–180 (1974).
75. Clowes, M. B. A note on colour discrimination under conditions of retinal image constraint. *Opt. Acta (Lond.)* **9**, 65–68 (1962).
76. Macknik, S. L., Martinez-Conde, S. & Haglund, M. M. The role of spatiotemporal edges in visibility and visual masking. *Proc. Natl Acad. Sci. USA* **97**, 7556–7560 (2000).
77. Leopold, D. A. & Logothetis, N. K. Activity changes in early visual cortex reflect monkey percepts during binocular rivalry. *Nature* **379**, 549–553 (1996).
78. Albrecht, D. G. & Hamilton, D. B. Striate cortex of monkey and cat: contrast response function. *J. Neurophysiol.* **48**, 217–237 (1982).
79. Albrecht, D. G. Visual cortex neurons in monkey and cat: effect of contrast on the spatial and temporal phase transfer functions. *Vis. Neurosci.* **12**, 1191–1210 (1995).
80. Gawne, T. J., Kjaer, T. W. & Richmond, B. J. Latency: another potential code for feature binding in striate cortex. *J. Neurophysiol.* **76**, 1356–1360 (1996).
81. Usrey, W. M., Reppas, J. B. & Reid, R. C. Paired-spike interactions and synaptic efficacy of retinal inputs to the thalamus. *Nature* **395**, 384–387 (1998).
82. Kara, P. & Reid, R. C. Efficacy of retinal spikes in driving cortical responses. *J. Neurosci.* **23**, 8547–8557 (2003).
83. Bridgeman, B. B. & Macknik, S. L. Saccadic suppression relies on luminance information. *Psychol. Res.* **58**, 163–168 (1995).
84. Macknik, S. L., Fisher, B. D. & Bridgeman, B. Flicker distorts visual space constancy. *Vision Res.* **31**, 2057–2064 (1991).
85. Wurtz, R. H. Visual cortex neurons: response to stimuli during rapid eye movements. *Science* **162**, 1148–1150 (1968).
86. Wurtz, R. H. Comparison of effects of eye movements and stimulus movements on striate cortex neurons of the monkey. *J. Neurophysiol.* **32**, 987–994 (1969).
87. Ross, J., Morrone, M. C., Goldberg, M. E. & Burr, D. C. Changes in visual perception at the time of saccades. *Trends Neurosci.* **24**, 113–121 (2001).
88. Beeler, G. W. Visual threshold changes resulting from spontaneous saccadic eye movements. *Vision Res.* **7**, 769–775 (1967).
89. Krauskopf, J. Lack of inhibition during involuntary saccades. *Am. J. Psychol.* **79**, 73–81 (1966).
90. Sperling, G. in *Eye Movements and Their Role in Visual and Cognitive Processes* (ed. Kowler, E.) 307–351 (Elsevier, Amsterdam, 1990).
91. Rucci, M., Edelman, G. E. & Wray, J. Modeling LGN responses during free-viewing: a possible role of microscopic eye movements in the refinement of cortical orientation selectivity. *J. Neurosci.* **20**, 4708–4720 (2000).
92. Olveczky, B. P., Baccus, S. A. & Meister, M. Segregation of object and background motion in the retina. *Nature* **423**, 401–408 (2003).
93. Masland, R. H. The retina's fancy tricks. *Nature* **423**, 387 (2003).
94. Zuber, B. L., Crider, A. & Stark, L. Saccadic suppression associated with microsaccades. *Q. Prog. Rep.* **74**, 244–249 (1964).
95. Zuber, B. L. & Stark, L. Saccadic suppression: elevation of visual threshold associated with saccadic eye movements. *Exp. Neurol.* **16**, 65–79 (1966).
96. Murakami, I. & Cavanagh, P. A jitter after-effect reveals motion-based stabilization of vision. *Nature* **395**, 798–801 (1998).
- A striking visual illusion that showed how jitter our perception of the world would be if fixational eye movements were not systematically compensated for by the visual system.**
97. Murakami, I. & Cavanagh, P. Visual jitter: evidence for visual-motion-based compensation of retinal slip due to small eye movements. *Vision Res.* **41**, 173–186 (2001).
98. Sasaki, Y., Murakami, I., Cavanagh, P. & Tootell, R. H. Human brain activity during illusory visual jitter as revealed by functional magnetic resonance imaging. *Neuron* **35**, 1147–1156 (2002).
99. Barash, S., Mellikyan, A., Sivakov, A. & Tauber, M. Shift of visual fixation dependent on background illumination. *J. Neurophysiol.* **79**, 2766–2781 (1998).
100. Steinman, R. M. Effect of target size, luminance, and color on monocular fixation. *J. Opt. Soc. Am.* **55**, 1158–1165 (1965).
101. Fender, D. H. Variation of fixation direction with colour of fixation target. *Br. J. Ophthalmol.* **39**, 294–297 (1955).
102. Sansbury, R. V., Skavenski, A. A., Haddad, G. M. & Steinman, R. M. Normal fixation of eccentric targets. *J. Opt. Soc. Am.* **63**, 612–614 (1973).
103. Corbetta, M. *et al.* A common network of functional areas for attention and eye movements. *Neuron* **21**, 761–773 (1998).
- This imaging study showed extensive anatomical overlap between the attentional and eye-movement related areas of the brain.**
104. Barlow, H. B. Eye movements during fixation. *J. Physiol. (Lond.)* **116**, 290–306 (1952).
105. Kowler, E. & Steinman, R. M. The effect of expectations on slow oculomotor control. I. Periodic target steps. *Vision Res.* **19**, 619–632 (1979).
106. Troxler, D. in *Ophthalmologische Bibliothek* (eds Himly, K. & Schmidt, J. A.) 1–53 (Springer, Jena, 1804).
107. Clarke, F. J. J. Rapid light adaptation of localised areas of the extra-foveal retina. *Opt. Acta (Lond.)* **4**, 69–77 (1957).
108. Clarke, F. J. J. A study of Troxler's effect. *Opt. Acta (Lond.)* **7**, 219–236 (1960).
109. Clarke, F. J. J. Visual recovery following local adaptation of the peripheral retina (Troxler's effect). *Opt. Acta (Lond.)* **8**, 121–135 (1961).
110. Clarke, F. J. J. & Belcher, S. J. On the localization of Troxler's effect in the visual pathway. *Vision Res.* **2**, 53–68 (1962).
111. Gilchrist, I. D., Brown, V. & Findlay, J. M. Saccades without eye movements. *Nature* **390**, 130–131 (1997).
112. Gilchrist, I. D., Brown, V., Findlay, J. M. & Clarke, M. P. Using the eye-movement system to control the head. *Proc. R. Soc. Lond. B* **265**, 1831–1836 (1998).
113. Land, M. F., Furness, S. M. & Gilchrist, I. D. The organization of visually mediated actions in a subject without eye movements. *Neurocase* **8**, 80–87 (2002).
114. Steinman, R. M. & Collewijn, H. Binocular retinal image motion during active head rotation. *Vision Res.* **20**, 415–429 (1980).
115. Verheijen, F. J. A simple after image method demonstrating the involuntary multidirectional eye movements during fixation. *Opt. Acta (Lond.)* **8**, 309–312 (1961).
116. Simon, F., Schulz, E., Rassow, B. & Haase, W. Binocular micromovement recording of human eyes—methods. *Graefes Arch. Clin. Exp. Ophthalmol.* **221**, 293–298 (1984).
117. Riggs, L. A., Armington, J. C. & Ratliff, F. Motions of the retinal image during fixation. *J. Opt. Soc. Am.* **44**, 315–321 (1954).
118. Adler, F. H. & Flegelman, M. Influence of fixation on the visual acuity. *Arch. Ophthalmol.* **12**, 475–483 (1934).
119. Schulz, E. Binocular micromovements in normal persons. *Graefes Arch. Clin. Exp. Ophthalmol.* **222**, 95–100 (1984).
120. Srebro, R. Fixation of normal and amblyopic eyes. *Arch. Ophthalmol.* **101**, 214–217 (1983).
121. West, D. C. & Boyce, P. R. The effect of flicker on eye movement. *Vision Res.* **8**, 171–192 (1968).
122. Boyce, P. R. Monocular fixation in human eye movement. *Proc. R. Soc. Lond. B* **167**, 293–315 (1967).
123. Malinov, I. V., Epelboim, J., Herst, A. N. & Steinman, R. M. Characteristics of saccades and vergence in two types of sequential looking tasks. *Vision Res.* **40**, 2083–2090 (2000).
124. Kingstone, A., Fendrich, R., Wessinger, C. M. & Reuter-Lorenz, P. A. Are microsaccades responsible for the gap effect? *Percept. Psychophys.* **57**, 796–801 (1995).
125. Sabrin, H. W. & Kertesz, A. E. Microsaccadic eye movements and binocular rivalry. *Percept. Psychophys.* **28**, 150–154 (1980).
126. Lord, M. P. & Wright, W. D. Eye movements during monocular fixation. *Nature* **162**, 25–26 (1948).
127. Ditchburn, R. W. & Foley-Fisher, J. A. Assembled data in eye movements. *Opt. Acta (Lond.)* **14**, 113–118 (1967).

Acknowledgements
We thank Y. Duran for technical assistance and X. G. Troncoso for comments on the manuscript. This study was funded by the Barrow Neurological Foundation and the National Eye Institute.

Competing interests statement
The authors declare that they have no competing financial interests.

 **Online links**

FURTHER INFORMATION
An online algorithm for microsaccade detection: <http://www.agnld.uni-potsdam.de/~ralfi/micro/>
Fading dot demonstration: http://www.exploratorium.edu/exhibits/fading_dot/fading_dot.html
Shimmer demonstration: <http://www.exploratorium.edu/exhibits/shimmer/shimmer.html>
Susana Martinez-Conde's page: <http://neuralcorrelate.com>
Troxler studies: <http://www.troxlerforum.ch/>
Visual jitter demonstration: <http://www.visionlab.harvard.edu/Members/Alumni/ikuya/html/memorandum/VisualJitter.html>
Access to this interactive links box is free online.