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# A jitter after-effect reveals motion-based stabilization of vision

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A shaky hand holding a video camera invariably turns a treasured moment into an annoying, jittery momento. More recent consumer cameras thoughtfully offer stabilization mechanisms to compensate for our unsteady grip. Our eyes face a similar challenge in that they are constantly making small movements even when we try to maintain a fixed gaze[1]. What should be substantial, distracting jitter passes completely unseen. Position changes from large eye movements (saccades) seem to be corrected on the basis of extraretinal signals such as the motor commands sent to the eye muscle[2–5], and the resulting motion responses seem to be simply switched off[6,7]. But this approach is impracticable for incessant, small displacements, and here we describe a novel visual illusion that reveals a compensation mechanism based on visual motion signals. Observers were adapted to a patch of dynamic random noise and then viewed a larger pattern of static random noise. The static noise in the unadapted regions then appeared to 'jitter' coherently in random directions. Several observations indicate that this visual jitter directly reflects fixational eye movements. We propose a model that accounts for this illusion as well as the stability of the visual world during small and/or slow eye movements such as fixational drift, smooth pursuit and low-amplitude mechanical vibrations of the eyes.

The experimental setting required for this illusion has three conditions: (1) adaptation to dynamic random noise in a local region (referred to as the adapted area) for at least several seconds; (2) a successive test with static random noise in the adapted area plus static noise in a region somewhere near the adapted area (referred to as the unadapted area); and (3) maintained fixation throughout these two periods. During the adaptation period, static noise is typically presented in the unadapted region (Fig. 1a), although leaving it blank does not change the outcome (Fig. 1b). After adaptation, static noise presented in the unadapted region seems to jitter rigidly (all dots moving together) in random directions for several seconds. In contrast, the static noise in the
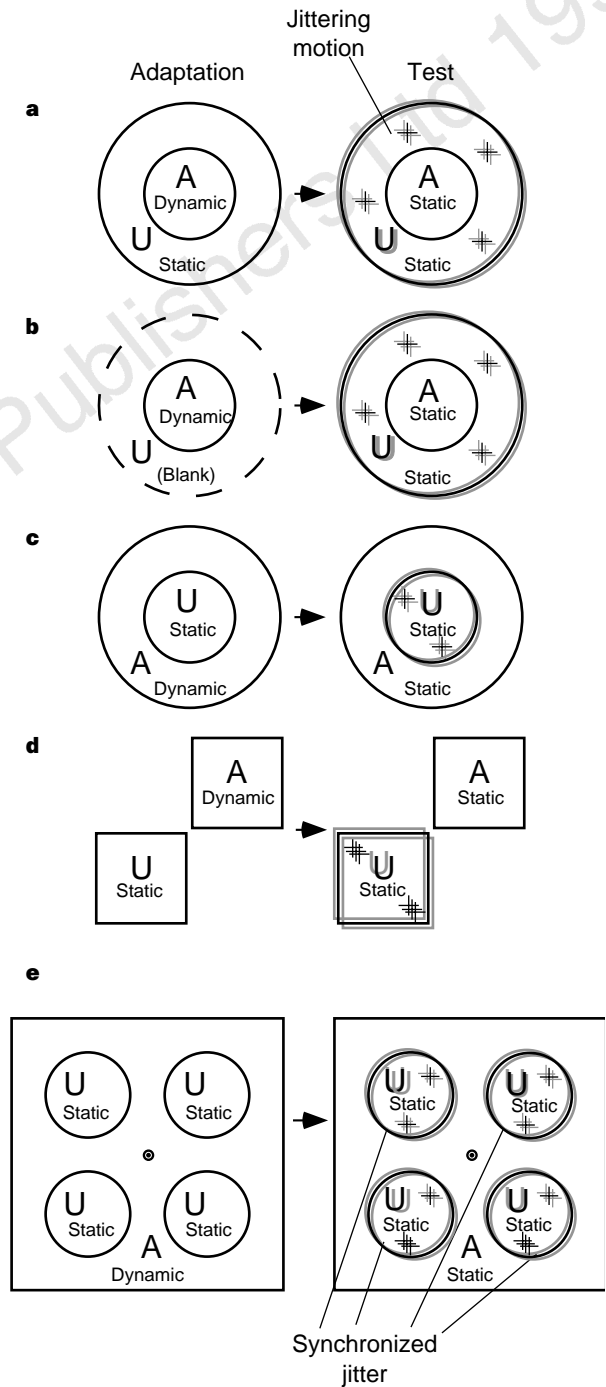
Figure 1 A schematic view of the stimulus configuration and perception. **a–e**, The various configurations used in these experiments and their outcomes. A fixation point is typically provided at the centre of the stimulus, but the illusion occurs for peripheral viewing as well. A and U stand for adapted and unadapted (static or blank) regions, respectively. The blur of circles and crosses in the test stimuli depicts the visual jitter schematically. See http://visionlab.harvard.edu/ for demonstrations.

adapted region seems stationary (as it actually is). Also, the unadapted region seems to jitter whether the adapted area surrounds the unadapted area or vice versa (Fig. 1c) or whether they are somewhat separated (Fig. 1d). These properties of the jitter were confirmed with tens of naive observers.

A similar adaptation procedure has been reported to produce a distinctly different aftereffect when a blank test field is used. In these experiments, a small uniform region was surrounded by dynamic random noise during adaptation, after which the whole display was turned to a uniform field; the small, unadapted region then seemed to be filled in with dynamic 'twinkle'[8–11]. The twinkle was an asynchronous, fine-grained scintillation with no sense of the common motion (jitter) seen on our static patterned test. Moreover, no 'twinkle' aftereffects could be observed in our displays when a uniform test field was used, probably because the unadapted regions in our test were slightly larger, and the adapted regions were much smaller, than those described in the 'twinkle' experiments.

We now describe several characteristics of the visual jitter effect. The duration of visual jitter was measured for the configurations shown in Fig. 1a, b for three conditions. The subjects observed the adaptation and test stimuli monocularly with either the same eye in both periods or different eyes, or binocularly in both periods. When the adaptation and test were in the same eye ('intraocular' and 'binocular' conditions), visual jitter lasted for approximately 5–12 s (Fig. 2). But when the adaptation and test were in different eyes ('interocular' condition), there was no transfer of the effect, unlike the substantial, although incomplete, transfer found for conventional motion aftereffects[12]. This result indicates that the adaptation is purely monocular and probably occurs at a very early level.

As motion adaptation in one region has been reported to induce a motion aftereffect in adjacent regions as well[13–15], visual jitter might be a type of induced jitter. However, one result in particular clearly differentiates visual jitter from other types of motion aftereffect: when unadapted areas were placed at four separate locations in the

adapting dynamic noise (Fig. 1e), the moment-to-moment directions of the jitter aftereffect seen in each were synchronized across all four regions. All regions moved together. No current model of the motion aftereffect could account for this synchronization. Rather, a single source of the illusory motion seems more likely, and fixational eye movements during the test period are the most obvious candidate.

If so, there should not be any visual jitter when the test stimulus is stabilized on the retina. We found this to be true. We analysed the subjective rating of jitter as a function of time after adaptation to (non-stabilized) dynamic noise. The magnitude of the jitter decreased gradually when non-stabilized noise was used throughout the test duration (Fig. 3, conditions N–N). However, when tested with retinally stabilized 'static' noise (the afterimage of a static noise field induced by a stroboscopic flash), no jitter was perceived (condition S–S). When the stabilized test was followed 3 s later by non-stabilized static noise, weaker but clear jitter re-emerged (condition S–N). In contrast, the jitter perceived in a non-stabilized image was extinguished when the stimulus was subsequently replaced by a stabilized image (condition N–S).

These characteristics suggest that the retinal slip caused by small eye movements during fixation is somehow unveiled in the unadapted area. However, the standard models of compensation for eye movements all predict that the jitter should be seen, if anywhere, in the adapted area. Efference copy of motor commands[3–5] or proprioceptive signals from extraocular muscles[16–18] are claimed to correct position changes caused by eye movements[19–21]. This would act to cancel the retinal slip in both adapted and unadapted areas. If we assume that appropriate position correction also suppresses motion responses, say by analogously subtracting the expected motion response, the correction should make the unadapted area appear motionless. But it would overcompensate in the adapted areas where the motion responses to the retinal slip have been attenuated (we verified that motion thresholds in, for example,
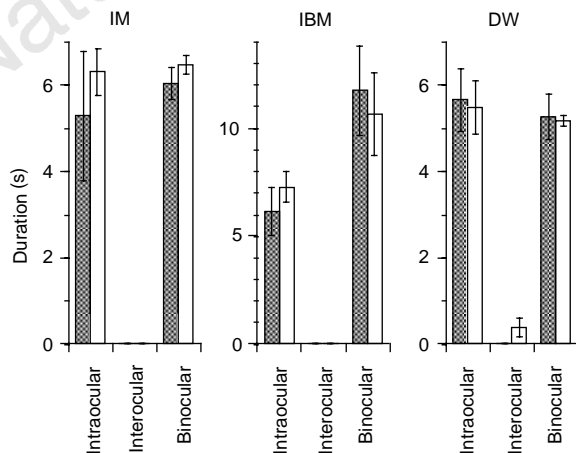
Figure 2 Duration of visual jitter for three subjects, I.M., I.B.M., and D.W. Filled and open bars indicate the results for the stimulus configuration shown in Fig. 1a (static unadapted) and Fig. 1b (blank unadapted), respectively. In the 'intraocular' condition, the same eye (either left or right) was used in both adaptation and test periods. In the 'interocular' condition, different eyes were used for adaptation and test. Both eyes were used in the 'binocular' condition. Each value is the mean of four repeated measurements; the error bars indicate ±1 s.e.m.
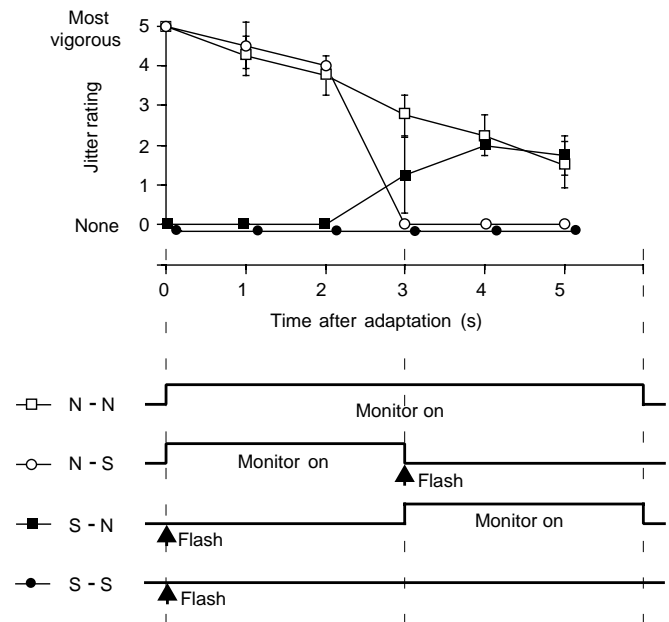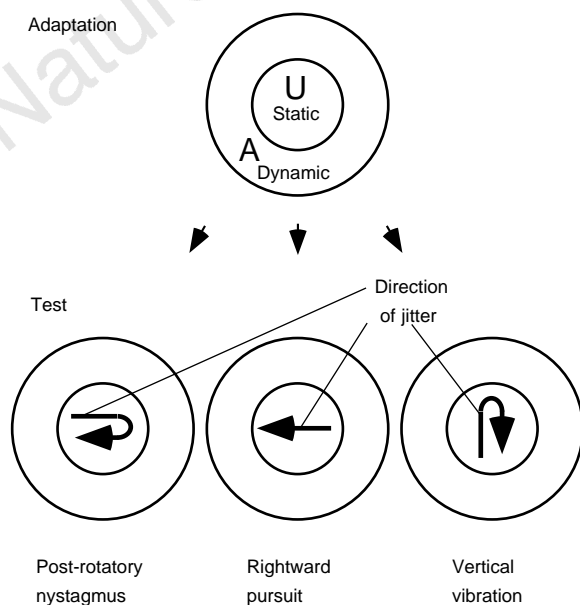

Figure 3 Typical rating data of a naive subject that show no visual jitter in the stabilized retinal image. Each data point is the mean of four repeats; error bars indicate ±1 s.d. The judged magnitude of jitter is plotted against the time at which the judgement was made, with the beginning of the test period set to time 0. different symbols indicate different test sequences (N, non-stabilized; S, stabilized). Below the graph the physical appearance and disappearance of the noise in the monitor is shown schematically alongside each symbol. The arrowheads indicate the time at which the flash was presented.

Gabor tests were elevated by adaptation to dynamic noise). Therefore, if any area should jitter, it should be the adapted areas, but the opposite is reported.

Indeed, small eye movements have often been considered to be too small to influence visual functions[22,23] and, unlike saccades, they are perhaps even too small to be accurately corrected by extraretinal signals. Nevertheless, they are quite capable of driving robust motion responses because motion mechanisms signal displacements at much smaller amplitudes than position mechanisms[24,25]. Motion responses triggered by saccades can be suppressed over the whole visual field[6,7], but this global suppression is not apparent during small eye movements as object motion is easily seen during the incessant small eye movements of normal fixation.

We propose that the motion signals triggered by small, low-velocity eye movements are compensated for solely on the basis of visual motion signals themselves. When one fixates a scene that has both moving and stationary components, the eye-movement velocity is added to the velocities of all image points on the retina. We suggest that a baseline value is recovered from the region of the retina having the lowest instantaneous velocity. The assumption is that this region corresponds to the stationary features of the scene and that motion in this region is due solely to eye movements. Subtract this baseline value from the velocities of all points on the retina and, if the assumption is appropriate, the result will be the desired zero velocity for the stationary regions and the correct velocity for the moving objects. This scheme convincingly explains two questions about visual jitter: (1) why jitter is perceived in the unadapted region and (2) why the adapted region seems stationary. Adaptation to dynamic random noise transiently attenuates responses to motion within the adapted region, creating a new baseline minimum velocity there. Other retinal velocities in the neighbourhood are computed as deviations from this new baseline. In the unadapted region, the unattenuated motion response to eye movements is now above the new, lower baseline. Therefore, the residual, after subtraction, is interpreted as a jittering motion in the unadapted region. In addition, because the adapted region is the source of the new baseline, it appropriately cancels its own motion to result in a zero vector, thereby seeming stationary.

Our proposal requires that the image points that have the current minimum velocity on the retina can be identified and that their velocity acts as the baseline representing the eye movements. We consider this rule quite plausible. It works well for generic situations and fails only if an object's motion accidentally correlates with eye movements over some period. Another point is that the minimum of retinal velocities is a simple statistical variable that early visual areas might find feasible to extract. Moreover, this approach is superior to simpler statistics such as a global motion average (as effectively computed by large surrounds of opponent-motion cells[26,27]). Simple averaging cannot differentiate between scene elements that are stationary and those that are moving. This inappropriate correction for motion responses to small eye movements would invariably make stationary parts of the field seem to drift. For example, the interior of a car would seem to rise continuously as we drive forward (the average motion visible through the car windows is downwards for a textured ground and featureless sky).

Is our scheme applied to other kinds of eye movement? Our observations have shown that several types of eye movement during the test period alter the pattern of jitter seen in the manner expected for our model (Fig. 4). For example, we can induce back-and-forth oscillations of gaze by rotating the observer rapidly around the vertical axis (post-rotatory nystagmus). In the interval between a standard adaptation and test, the subject was rotated for several seconds with eyes closed and suddenly stopped. The direction of the visual jitter seen in the unadapted area during the test was almost always horizontal with quick and slow phases, in accordance with the nature of the post-rotatory nystagmus. A similar result held for voluntary smooth pursuits. When, after standard adaptation, a pursuit is made to track a moving spot during test, the unadapted background slides rapidly in the opposite direction. This is the direction of the retinal slip, which is now visible. Furthermore, visual jitter was found to mimic even retinal slip in the eye moved by an external force. After adaptation, up-and-down eye displacements were induced externally by a rapid alternation of extension/relaxation in the nearby skin; at amplitudes of vibration that did not produce noticeable motions of the world, vertical jitter nevertheless occurred in the unadapted area of the static test with a temporal profile synchronized with this vibration. This result suggests that the motion-based correction can compensate for small-amplitude displacements whether driven by the eye muscles or mechanically (such as the vibrations of talking or eating which reach the eyes through bone conduction). Overall, the jitter aftereffect is only one of a wide pattern of motions that can be seen in the unadapted area, each mirroring the eye movements occurring during the test.

The nature of these aftereffects supports the claim that visual motion information is used to correct for the spurious motion signals generated by various eye movements including nystagmus, smooth pursuit and fixational eye movements. Our model complements the extraretinally based processes[2–5,16–18], which correct for large position shifts due to saccades but do not affect the motion signals elicited by small eye movements. Without the motion-based process that we propose, one would suffer from an incessant jittering of the visual world owing to the motion responses to small displacements of the eyes.

Note added in proof: We are happy to acknowledge that this jitter effect has been previously observed by Stuart M. Anstis and Richard Gregory (unpublished manuscript). □

Figure 4 Similar phenomena contingent on various kinds of eye movement during the test period. The black arrows in the central unadapted regions illustrate schematically the direction of illusory motion, which is consistent with the direction of retinal slip due to eye movements indicated below each figure.

Methods

I.M. and two naive subjects participated in formal data acquisition. All had corrected-to-normal visual acuity. In a darkened booth, the stimulus was presented on the screen of a 20-inch colour CRT monitor (Sony GDM2000TC, 832 pixels × 624 pixels) controlled by a computer (Apple Power Macintosh). The subject's head was immobilized with a chinrest; the viewing distance was 77.3 cm.

The adapting stimulus consisted of two concentric regions (see Fig. 1a). A

fixation point was provided in the centre throughout the trial. In the inner circle subtending 6.67° in diameter, black and white dynamic random noise was presented; each dot (8 arcmin × 8 arcmin) was randomly assigned black (0.18 cd/m^2) or white (52.4 cd/m^2) every frame (75 Hz). In the outer annulus with an outer diameter of 13.33°, static random noise was presented (or not, as in Fig. 1b). There was a uniform grey surround (23.5 cd/m^2) outside the stimuli. After 30 s of adaptation, the two regions were changed to a new pattern of static random noise. To quantify the magnitude of the effect, the total duration of the visual jitter in a 20-s test period was measured; the subject was asked to hold a computer button whenever jitter was apparent and to release the button whenever no jitter was perceived. There was at least 10 s of a blank display between trials.

A stroboscopic flash (Speedotron 4803CX) was used to generate an afterimage under the control of the same computer. It briefly illuminated a printout of the random noise, matched to that on the CRT display, that was otherwise unilluminated and invisible. It was optically superimposed onto the stimulus in the monitor by using a half-silvered mirror. The observation was made binocularly. To make the most conspicuous afterimage, the subject was dark-adapted for at least 10 min before experiments, and the luminance of stimuli in the monitor was reduced (3.1 cd/m^2 for white, 0.01 cd/m^2 otherwise). The monitor was kept totally dark during the period when the subject was to observe the afterimage. Likewise, the perception of afterimage was effectively suppressed when the monitor was turned on again. In this experiment, the method of subjective rating (choosing an integer from 0 to 5, with 0 as no jitter and 5 as the maximum) was used to quantify the magnitude of the illusion because the duration measurement was not appropriate for examining the time course of the effect. In a preliminary experiment, the duration of jitter in the stabilized test (the afterimage) was measured and gave a result of 0 s.

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The DCC gene product induces apoptosis by a mechanism requiring receptor proteolysis

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The development of colonic carcinoma is associated with the mutation of a specific set of genes[1]. One of these, *DCC* (*deleted in colorectal cancer*)[2–5], is a candidate tumour-suppressor gene, and encodes a receptor for netrin-1, a molecule involved in axon guidance[6–8]. Loss of DCC expression in tumours is not restricted to colon carcinoma[2], and, although there is no increase in the frequency of tumour formation in *DCC* hemizygous mice[5], re-establishment of DCC expression suppresses tumorigenicity[3,4]. However, the mechanism of action of DCC is unknown. Here we show that DCC induces apoptosis in the absence of ligand binding, but blocks apoptosis when engaged by netrin-1. Furthermore, DCC is a caspase substrate, and mutation of the site at which caspase-3 cleaves DCC suppresses the pro-apoptotic effect of DCC completely. These results indicate that DCC may function as a tumour-suppressor protein by inducing apoptosis in settings
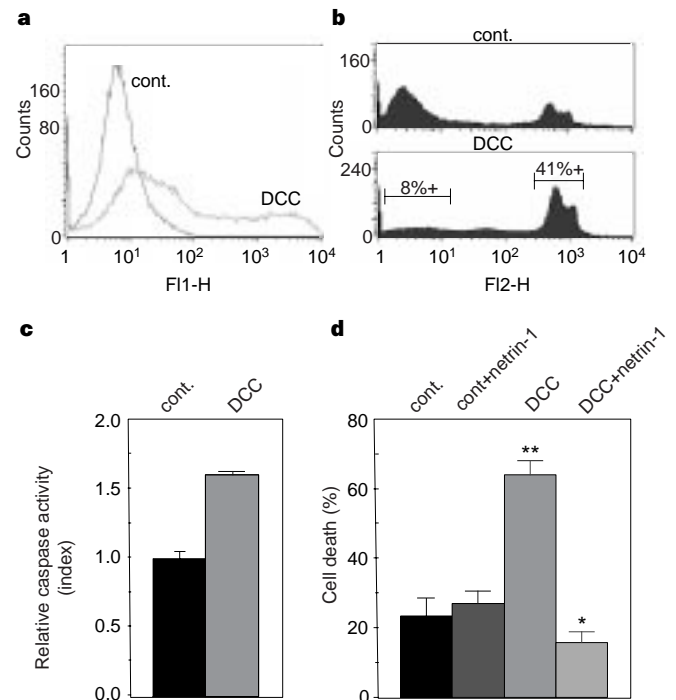
Figure 1 DCC expression induces cell death, which is blocked by netrin-1. 293T cells were transiently transfected with either the DCC expression plasmid pDCC-CMV-S (DCC) or the pCMV control plasmid (cont.). **a**, Expression of DCC 48 h after transfection. **b**, Cell death induced by DCC expression, monitored by flow cytometry using propidium iodide incorporation. Note the increase in dead cells (bottom right peak) after DCC expression. Only 8% of the live cells expressed DCC, in comparison to 41% of the dead cells. **c**, Increase in caspase activity after DCC expression. **d**, Netrin-1 expression blocks DCC-induced cell death. Standard deviations are indicated (n = 3). Double asterisk indicates $P < 0.005$ (unpaired t-test); single asterisk indicates $P < 0.05$ (paired t-test).