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Motion-induced blindness in normal observers

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Cases in which salient visual stimuli do not register consciously are known to occur in special conditions, such as the presentation of dissimilar stimuli to the two eyes[1] or when images are stabilized on the retina[2]. Here, we report a striking phenomenon of 'visual disappearance' observed with normal-sighted observers under natural conditions. When a global moving pattern is superimposed on high-contrast stationary or slowly moving stimuli, the latter disappear and reappear alternately for periods of several seconds. We show that this motion-induced blindness (MIB) phenomenon is unlikely to reflect retinal suppression, sensory masking or adaptation. The phenomenology observed includes perceptual grouping effects, object rivalry and visual field anisotropy. This is very similar to that found in other types of visual disappearance, as well as in clinical cases of attention deficits, in which partial invisibility might occur despite the primary visual areas being intact[3]. Disappearance might reflect a disruption of attentional processing, which shifts the system into a winner-takes-all mode, uncovering the dynamics of competition between object representations within the human visual system.

The phenomenon reported here adds to a class of known phenomena of 'visual disappearance' in which salient stimuli disappear from visual awareness, as if erased in front of the observer's eyes. Such phenomena, in which information is ignored owing not to a failure to notice[4] but to explicit 'erasing', include binocular[1,5] and monocular[6,7] rivalry (in which superimposed dissimilar patterns presented to different eyes or in different colours disappear in alternation), stabilized images that fade away[2], after-images that similarly disappear and reappear[8], and Troxler fading (in which low-contrast peripheral stimuli disappear under strict fixation[9]). Clinical cases of explicit disappearance have also been reported in patients with simultanagnosia[10,11]. The phenomenon of MIB occurs in normal observers under normal (monocular) viewing conditions and might occur in natural situations. It was first described by Grindley and Townsend[12,13], who studied 'movement masking' in binocular rivalry, but went largely ignored until now, probably because its compelling strength in normal viewing was never observed.

We presented high-contrast yellow patterns (targets) together with a dynamic blue random dot pattern (mask), as described in Fig. 1. With steady fixation, but not with strict fixation (small eye movements could be tolerated), observers reported seeing long periods (several seconds) of complete disappearance of one or more target patterns, which disappeared and reappeared in a seemingly spontaneous way. We used the accumulated duration of disappearance as a measure for studying the parameters that affect this phenomenon and the mechanisms involved. Several hundred observers[14] have already confirmed the effect by subjective report, and very few have failed to experience MIB.

The results are summarized in Fig. 2. The properties of MIB do not seem to reflect sensory suppression or adaptation. First, targets of higher luminance contrast disappeared more (Fig. 2a), as opposed to the Troxler fading effect[15], and thus cannot be explained by a contrast-gain-control mechanism. Second, moving or dynamic targets disappeared too (Fig. 2c, d), producing the striking phe-

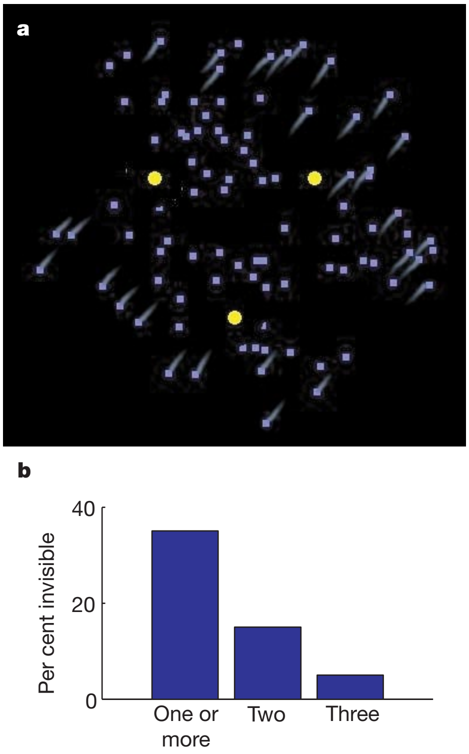

Figure 1 Typical stimuli used to measure the motion-induced blindness (MIB) effect and typical data showing its magnitude. Observers viewed high-contrast target patterns together with a dynamic random dot pattern ('rotating sphere') and reported the disappearance of the targets (see Methods). **a**, A typical snapshot of the dynamic display used. **b**, The percentage of accumulated invisibility period for the disappearance of one or more patches, exactly two patches and exactly three patches. The dots disappeared for about 30% of the trial duration, with disappearance episodes extending up to 10 s.

nomenon of target dots that disappeared in one quadrant and reappeared in another after a few seconds. Such disappearance is unlikely to be explained by local adaptation or retinal stabilization effects. Third, the effect did not depend on local masking, as targets continued to disappear even when surrounded by background-colour circular 'protection zones' that occluded the moving mask (Fig. 2e). In such cases, targets disappeared without filling-in of the empty zones by the moving surround. However, targets did not disappear when positioned far outside the area of the mask (data not shown), suggesting that the effect is spatially limited. We also investigated the possibility that the effect depends on three-dimensional interpretation of the image; that is, on the structure from motion and occlusion. We found advantage for the three-dimensional dot sphere mask (Fig. 2h), although two-dimensional masks and Brownian motion could also induce the effect, leaving this issue open. Movement was critical in all cases but colour was not.

To investigate the functional level of visual processing affected by MIB, and to relate the MIB to other phenomena, we tested gestalt properties of figural organization (Figs 3 and 4). Good gestalts, defined by proximity or contour smoothness, tended to disappear entirely (as 'wholes') or to resist disappearance, as previously reported for stabilized images[16]. More surprising was the observed object rivalry that occurred between two partially overlapping (ellipses of different colours) or adjacent (orthogonal Gabor

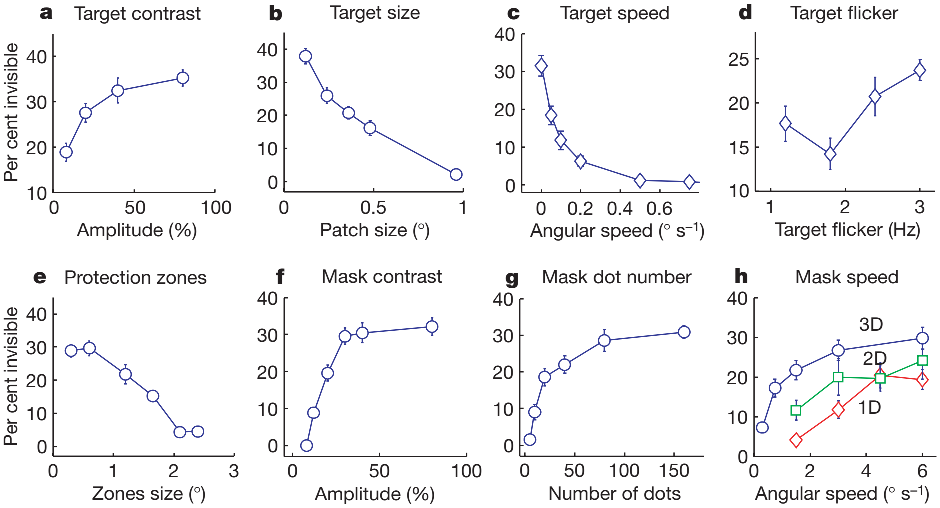

Figure 2 The effects of different parameters on the motion-induced blindness phenomenon. The percentage of accumulated invisibility period (any target) is plotted against different target and mask parameters (see Methods). **a**, Target luminance contrast. **b**, Target size. **c**, Target speed; the three-dot configuration was slowly rotated. **d**, Target flicker. **e**, The locality of the masking effect; size (diameter) of empty 'protection zones' around the targets. **f**, Mask luminance contrast. **g**, Mask number of dots. **h**, Mask speed and motion type: three-dimensional (3D) sphere (standard), two-dimensional (2D) rotation and one-dimensional (1D) left-to-right linear motion. Brighter targets disappeared more than dim ones (**a**), and disappearance was not eliminated with dynamic targets (**c**) or when the mask dots were distant from the target (**e**), and did not occur with static masks (**h**).

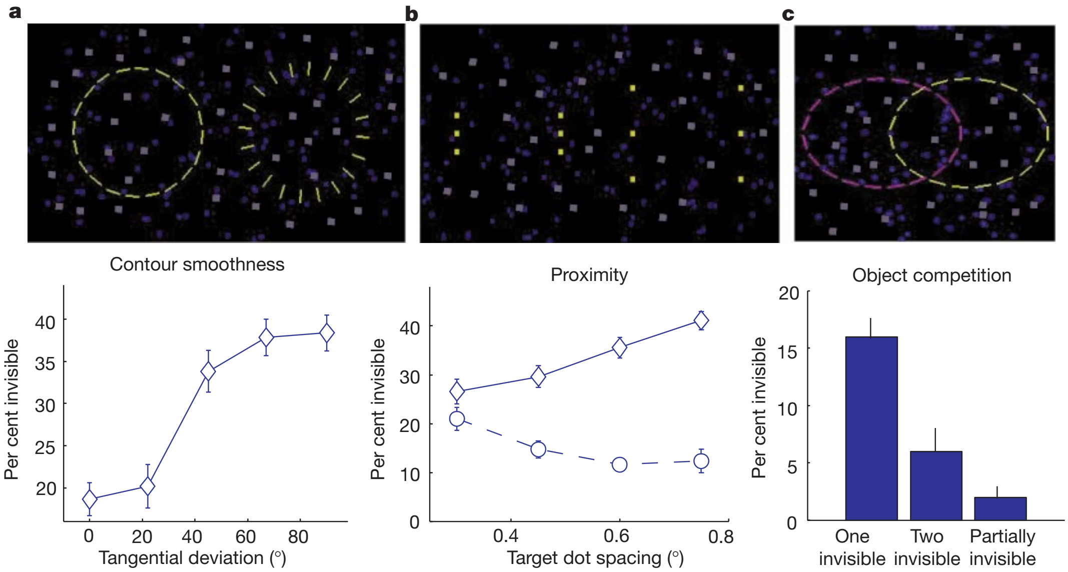

Figure 3 Gestalt effects reflected in the motion-induced blindness phenomena. The percentage of accumulated invisibility periods is plotted for different conditions. Snapshots of the two extreme conditions for each stimulus are presented at the top. The size of the mask dots was increased for clarity. **a**, Contour smoothness effect: the disappearance of any part of the circle is plotted against the tangential deviation of the circle elements, showing that smooth contours disappear less frequently. **b**, Proximity effect: the disappearance of any dot ('parts' condition) and of whole groups ('wholes' condition) are plotted against the interdot spacing. Increasing the spacing increased the disappearance of 'parts' but decreased the disappearance of 'wholes'. **c**, Object competition effect: complete and partial disappearance of each of two elliptical line configurations of different colours. Perception alternated between states of complete invisibility of each object for over 15% of the time.

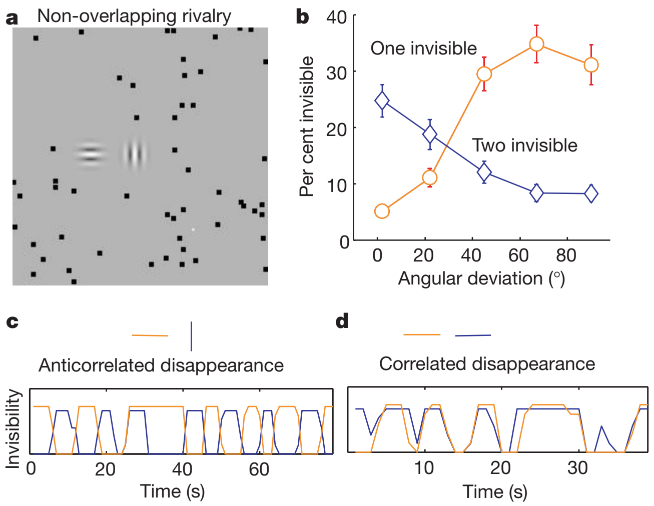

Figure 4 Co-operation and rivalry of non-overlapping stimuli under MIB conditions. **a**, A snapshot of stimuli used to measure anticorrelated disappearance (see Methods). **b**, Accumulated invisibility periods of one or two patches as a function of deviation from collinearity. With increased deviation, the percentage of 'one invisible' (rivalry) increased and the percentage of two invisible (co-operation) decreased. **c**, **d**, Invisibility against time (smoothed) as measured for one observer for orthogonal (**c**) and collinear (**d**) pairs of patches. The orthogonal patches engaged in anticorrelated disappearance (rivalry, $R = -0.75$), whereas the collinear patches were correlated (disappeared and reappeared together, $R = 0.76$).

patches) objects, which disappeared in alternation when superimposed by a 'rotating sphere' of dots. In Fig. 4c, we show an example of anticorrelated disappearance (rivalry) between two orthogonal patches compared with correlated disappearance for collinear patches (Fig. 4d). Overall, correlated (0.3–0.9) and anticorrelated or poorly correlated (−0.8 to 0.2) disappearance were observed for the collinear and orthogonal configurations respectively. Alternating disappearance of similar competing objects has been observed in simultanagnosia patients[10]. This suggests that MIB does not merely reflect suppression by movement but uncovers mechanisms of object competition that shift to operate in a winner-takes-all mode, as observed in the clinical cases.

We further observed that MIB is not uniformly distributed across space. Seven observers (two of them tested with each eye (see Methods)) reported more (typically twice as much) disappearance in the upper left field (two degrees of eccentricity) compared with the lower field. In comparison, we observed similar anisotropy in disappearance during figure–ground binocular rivalry, with targets presented to one eye and a static background mask (without motion) to the other (three observers). Moreover, two orthogonal Gabor patches presented to one eye, with a background of static random dot pattern presented to the other eye, often engaged in anticorrelated disappearance as in MIB. These similarities and the known disappearance of wholes and spatial configuration effects found in binocular[17–20] and monocular rivalry[8,18] suggest a common mechanism that gates or modulates conscious perception at the level of objects.

Why do salient stimuli disappear? Current explanations of other phenomena divide between sensory suppression[5,21] and 'higher-level' selection[22,23]. Our study shows that MIB is unlikely to be caused by sensory suppression or local adaptation. Recent evidence suggests the involvement of non-sensory or attention mechanisms. Using transcranial magnetic stimulation (TMS) with our basic stimuli (Fig. 1), it has been found[24] that a suppressive TMS pulse applied to parietal areas immediately after disappearance increased or decreased disappearance depending on the stimulated hemisphere. This suggests the involvement of attention mechanisms, which are assigned to competing objects and tend to divide between hemispheres. Additional evidence for the link between attention and disappearance comes from dorsal simultanagnosia patients, who report alternating disappearance of objects following bilateral occipito-parietal damage[10,11].

Taken together, these data suggest the following conclusions. (1) Under MIB conditions, or more generally for stimuli with sensory dissociation (different dynamics, eyes or colours), the visual system shifts to operate in a winner-takes-all mode. (2) This mode could be described as a disruption and slow-down of the commonly assumed, but usually unnoticed, fast attentional switching between objects in the scene. With such disruption, competing objects are perceived one at a time, with phenomenology similar to that observed in the clinical cases of attention deficits. (3) This disruption might occur because attentional mechanisms cannot be allocated or divided between dissociated or 'unfused' elements at the same time and location. (4) The actual rivalry and suppression could occur between competing object representations modulated by attention[25] or between attention mechanisms assigned to objects in space. The recent evidence for parietal mechanisms that represent visual space, and objects in space[26], as well as converging clinical evidence[3], might suggest the neural substrates for the mechanisms that gate visual appearance and disappearance. Finally, it is intriguing to consider the possibility that MIB, and visual disappearance in general, are just one manifestation of stimuli discarded by the visual system while fitting a consistent and useful interpretation to a fuzzy sensory input. In some rare cases, we are aware of the input being discarded, but most of the time we are not. ☐

Methods

Observers and stimuli

Ten observers (eight of them naive) took part in the experiments. In each experiment, four observers (three of them naive) viewed high-contrast patterns in a dark background together with a blue random dot (150 dots) pattern (mask) (see Supplementary Information). This pattern was displayed as if arranged on the surface of a 6° diameter rotating (x, y and z axes) sphere and viewed from 1 m for 60 s, repeated five times. In the experiments reported in Fig. 2, the targets consisted of three 0.2° yellow patches arranged along a 1° radius circle forming a triangle. Observers were instructed to attend to the rotating mask without following it with their eyes, while simultaneously reporting the disappearance of the targets by depressing three buttons, one for each target. Display luminance was set to 100 and 20 $\mathrm{cd\,m^{-2}}$ for 100% luminance contrast of the yellow and blue stimuli, respectively. Background luminance was set to 40 $\mathrm{cd\,m^{-2}}$ for the Gabor stimuli.

Parametric manipulations

Luminance contrast (Fig. 2a) varied in the range 10–80%. Size (Fig. 2b) varied in the diameter range 0.2–1°. Speed (Fig. 2c) was varied by slowly rotating the three-dot target configuration at angular speeds of 0–0.8° $\mathrm{s^{-1}}$. Flicker (Fig. 2d) was varied by a linear modulation of target luminance between 0 and maximum luminance at 1–3 Hz. Locality of the masking effect was measured (Fig. 2e) by creating circular black 'protection zones' that occluded the mask dots with diameters of 0.2–2.5°. Mask luminance contrast (Fig. 2f) was varied in the range 10–80% and the number of mask dots (Fig. 2g) in the range 0–180. Mask speed and motion type were tested (Fig. 2h) by changing the angular speed (expressed as maximum visual angle displacement per s) for three types of motion: 3D sphere (standard); 2D rotation of an array of crosses (approximately the same size as the sphere); and 1D left-to-right linear motion of a similar array.

Gestalt effects

To test the contour smoothness effect (Fig. 3a), we varied the tangential deviation of line segments arranged in a 2.1° (diameter) circle, between smooth (0° deviation) and sun-shaped (90° deviation). Observers reported disappearance of any part of the circle.

To test the effect of proximity, we varied the spacing in two groups of three dots each, from 0.3° (Fig. 3b, left) to 0.75° (Fig. 3b, right). The groups were separated by 2.1° along the *x* axis. Observers reported the disappearance of any dot ('parts' condition) and the disappearance of whole groups ('wholes' condition).

To test object competition, we presented two elliptical line configurations (1.8° × 2.2°, with 0.6° displacement) of different colours (Fig. 3c, top right), with observers reporting the complete disappearance of each configuration and partial disappearance of any part.

To test the co-operation and rivalry of non-overlapping stimuli under MIB conditions (Fig. 4), we used two adjacent Gabor patches. These had 8 cycles per degree and were three wavelengths apart, 2° from fixation in the upper left quadrant, on a grey background with a 'rotating sphere' made of black dots. They were shown to five observers.

To test the distribution of MIB across space, we presented a single yellow dot at 30 different locations within 5° from fixation, with a 5.5° radius sphere, three times for 45 s periods. These were shown to seven observers.

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Interaction with the NMDA receptor locks CaMKII in an active conformation

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Calcium- and calmodulin-dependent protein kinase II (CaMKII) and glutamate receptors are integrally involved in forms of synaptic plasticity that may underlie learning and memory. In the simplest model for long-term potentiation[1], CaMKII is activated by Ca^{2+} influx through NMDA (*N*-methyl-D-aspartate) receptors and then potentiates synaptic efficacy by inducing synaptic insertion[2,3] and increased single-channel conductance[4] of AMPA (α-amino-3-hydroxy-5-methyl-4-isoxazole propionic acid) receptors. Here we show that regulated CaMKII interaction with two sites on the NMDA receptor subunit NR2B provides a mechanism for the glutamate-induced translocation of the kinase to the synapse in hippocampal neurons. This interaction can lead to additional forms of potentiation by: facilitated CaMKII response to synaptoc Ca^{2+}; suppression of inhibitory autophosphorylation of CaMKII; and, most notably, direct generation of sustained Ca^{2+}/calmodulin (CaM)-independent (autonomous) kinase activity by a mechanism that is independent of the phosphorylation state. Furthermore, the interaction leads to trapping of CaM that may reduce down-regulation of NMDA receptor activity[5]. CaMKII–NR2B interaction may be prototypical for direct activation of a kinase by its targeting protein.

The generation of autonomous CaMKII activity has an important role in various forms of synaptic plasticity, and has been regarded as 'molecular memory', as the kinase remains active after the initial Ca^{2+} stimulus has subsided (see refs 1, 6, 7 for review). Autonomous CaMKII activity depends on autophosphorylation of T286 in its auto-inhibitory domain. Autophosphorylation requires coincident binding of at least two Ca^{2+}/CaM molecules to a dodecameric CaMKII holoenzyme[8,9] and enables Ca^{2+}-spike-frequency decoding by the kinase[10]. T286 autophosphorylation also results in greatly enhanced CaM binding (CaM trapping)[11] by the highly abundant α-CaMKII, which may sequester CaM and limit its availability for the NMDA receptor and other synaptic proteins. The initial Ca^{2+} stimulus for CaMKII activation can be provided by the NMDA receptor, the only known activity-dependent binding partner for CaMKII at the synapse[12–14]. Binding of α-CaMKII to the NMDA receptor was reported to require autophosphorylation[14], whereas NMDA-stimulated translocation of the kinase to neuronal synapses does not[15,16]; thus the two events did not seem to be linked. Our binding studies, however, now demonstrate that stimulation by Ca^{2+}/CaM is sufficient to induce binding of α-CaMKII to the cytoplasmic carboxy terminus of NR2B (residues 839–1,482) and that autophosphorylation is not required. In fact, this NR2B domain contains two sites with different modes of regulated CaMKII binding (Fig. 1), a Ca^{2+}/CaM-regulated site within residues 1,120–1,482 of NR2B (NR2B-C) and a phosphorylation-regulated site within residues 839–1,120 (NR2B-P). CaMKII binds to NR2B-

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