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Vision: In the Blink of an Eye

Although we blink every 4 to 6 seconds, we notice neither the act of blinking nor the mini-blackouts they cause. A new study using imaging techniques identifies the neural structures in humans involved in suppressing vision processing and visual awareness during blinking.

David Burr

We blink reflexively to protect our eyes from potential damage that might be caused by external objects such as flying insects and unruly hair, and also spontaneously, 10 to 15 times a minute, to moisten and oxygenate our corneas. We can intentionally blink or inhibit blinking, as in the child's game of 'stare down', but in general blinking is as automatic, and goes as unheeded, as breathing.

Each blink lasts for 100–150 milliseconds, during which time it obstructs all pattern vision and attenuates light levels 100-fold. Not only do blinks disrupt visual input, they generate a strong, transient whole-field decrement in luminance that would normally be highly disturbing. So why do these continuous mini-blackouts escape notice completely? One strategy would be to blink one eye at a time, so vision is never completely disrupted. This is what birds do. In mammals, however, blinking always involves both eyes simultaneously — except for the intentional social signal, the wink — possibly to minimise downtime of binocular vision, fundamental for depth perception and for breaking camouflage.

So there is a problem. The solution is that vision is transiently suppressed during each blink. Twenty five years ago, Frances Volkmann and colleagues [1] devised an ingenious technique to measure the neural consequences of blinks on visual function, bypassing the physical consequences of eyelid closure by stimulating the retinae via the mouth. Light passes through the palatine bone to trans-illuminate the photoreceptors without being affected by eyelid closure (Figure 1). In an elegant series of studies they described the timecourse and magnitude of blink suppression, usually a factor of about three (reviewed in [2]).

Eye-blinking is not the only disruption vision has to cope with. A more frequent, and in some ways more disruptive, problem arises from the rapid ballistic movements called saccades, with which we actively scan the world. As with blinks, we are normally unaware both of the fact that our eyes are continuously moving, and of the image motion and image displacement the movement causes. And as with blinks, vision is actively suppressed at the time of saccades (reviewed in [3]).

Saccadic suppression shares much in common with blink suppression, implicating a common mechanism: the magnitude and timecourse are similar [2]; they show similar spectral and spatial-frequency selectivity [4–7]; and blink and saccadic suppression co-vary between individuals in a similar way [8]. Indeed blinks and large-saccades are often coordinated [9], presumably to minimise downtime in visual processing. Importantly, there is good evidence to suggest that both blinks and saccades affect primarily the magnocellular visual pathway [6,7], the pathway tuned to low spatial and high temporal frequencies, strongly implicated in motion and flash perception. The suppression seems to occur via contrast gain control [10], an important component of early visual processing, particularly for the magnocellular pathways.

Although much work points to active suppression of vision during blinks, the neural mechanisms of the suppression are yet to be unveiled. As they report in this issue of *Current Biology*, Bristow *et al.* [11] investigated the neural consequences of blinking on visual processing with functional

magnetic imaging (fMRI). Using the clever technique of trans-palatine retinal stimulation [1], they compared BOLD activity in response to whole-field flicker during periods of rapid blinking with activity during periods of no intentional blinks (red and green bars of Figure 2). They found strong suppression of the visual response during blinking in many cortical areas, most notably in V3 — a motion-sensitive area that also responds to flashes — as well as in many parietal and pre-frontal areas.

Interestingly, visually driven BOLD activity was not directly reduced during blinking in early visual areas — the lateral geniculate nucleus of the thalamus (LGN), and the primary and secondary visual cortices V1 and V2. However, we should not rush to conclude that no suppression occurs in those areas. As blink suppression seems to be selective to the magno-cellular system [6,7], any suppression of this pathway may be swamped by the response of the non-suppressed cells from the parvo-system, which are intermingled at voxel resolution and outnumber magno-driven cells in LGN, V1 and V2.

Another curious aspect of this and previous [12] studies is that, in the absence of any visual stimulation, blinks cause a significant increase in BOLD activity in many early visual areas, as do saccades [13], reinforcing claims that blink and saccadic suppression share common neural mechanisms. Bristow *et al*. [11] suggest that this activation reflects a motor signal associated with blinking, presumably a form of 'corollary discharge' that accompanies the motor command.

Why should a motor signal be present in early visual areas? A strong possibility is that the BOLD activity shown by the dark blue bars of Figure 2 is associated with the motor signal that down-regulates the neural responsiveness of visual neurones. If this were the case, then the increase of BOLD response should also occur during retinal stimulation, and should therefore be subtracted from the visually driven activity shown by the green bars, to reveal the purely visual response (light blue bars of Figure 2).

Figure 1. The technique devised by Volkmann *et al.* [1] for visual stimulation bypassing the eyelids.

Light is flashed in the mouth at 7 Hz and passes through the palatine bone to stimulate the retinae without being affected by the eyelid closure.

If we were to accept this logic, the new results [11] imply almost total suppression in LGN and V3, and a more modest suppression in V1 and V2. This is strongly consistent with selective suppression of the magno-pathway, as V3 has a very strong magno-cellular input from the layer 4B of V1 [14], which receives primarily magno-cellar input, and the magno-cells of the LGN, although inferior in number, form a larger relative mass than do the magno-driven cells of V1 and V2. Unfortunately area MT, also driven by the magno-cellular pathway, does not respond well to diffuse flicker, so it was not possible to show suppression, but there was a strong blink signal both in dark and during stimulation (reinforcing the suggestion that it is always there, and needs to be subtracted).

Bristow *et al*.'s [11] result is also consistent with both the psychophysical [6] and electrophysiological [15] evidence that saccadic suppression occurs at early vision stations, probably as early as LGN. Perhaps what is now needed is to search for suppression at sub-voxel resolution, using techniques such as adaptation [16]. It would also be interesting to look for effects in sub-cortical, magno-driven visual areas, like the superior colliculus, although this would seem to be technically unfeasible at this stage.

Ever since blink and saccadic suppression were first observed, there has been a debate about the evidence and the need for active neural suppression of visual signals, with some suggesting that purely visual mechanisms such as 'masking' may suffice [17,18]. This view is clearly not compatible with these latest results: under conditions where the blinks could have no visual effects whatsoever, strong suppression was observed in many cortical areas. This does not rule out a possible subsidiary role of visual factors in blink and saccade suppression, but it demonstrates unequivocally the existence of a non-visual suppression, supporting much previous psychophysical evidence [3].

A very interesting aspect of the Bristow *et al*. [11] paper is that they report blink suppression, not only in early visual areas, but in 21 regions of parietal and pre-frontal cortices. One possibility is that the reduction of activity in the higher areas merely reflects the reduced output from early visual areas that are actively suppressed. The other

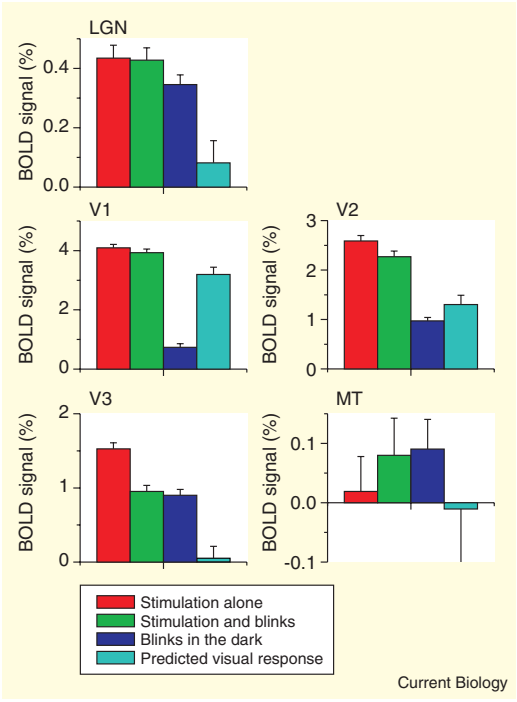

Figure 2. Summary of Bristow *et al.*'s [11] results, showing BOLD response in five visual areas.

Red bars, response to whole field flicker of 6.7 Hz, via the roof of the mouth; green bars, response to the same stimuli while subjects blinked frequently; dark blue bars, response in darkness to similar blink frequencies; light blue bars, the response to retinal stimulation during blinking, after subtracting the response to blinking in the dark (green bars minus dark blue bars). Note that this result was not shown in the authors' paper [11], and the assumptions behind making the subtraction — such as linearity in the neural signal and of the BOLD response – are not necessarily justified. (The results for MT were communicated directly by the authors, the others reproduced from their Figure 2.)

possibility, favoured by the authors, is that these higher areas are actively suppressed during blinking. This is an exciting idea, as many of the parietal and prefrontal areas have been associated with fluctuations in consciousness [19,20]. Although evidence for a suppression of primary visual function during blinks and saccades is now indisputable, it remains a mystery of how this modest (0.5–1 log unit) suppression can completely eliminate all sensation of motion or flash that should accompany rapid motion or a 150 millisecond blackout. Fast, whole-field motion is particularly attention-grabbing: but if it occurs during saccades, one can be intellectually aware of a change in position but perceive no sense of motion or of *startle* [4]. Perhaps we are not startled by the blink black-out or the saccade-induced motion because those areas that register awareness of these events are momentarily anaesthetised.

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Retinal Development: Second Sight Comes First

Mammals are functionally blind at birth because responses to rod and cone photoreceptor activation are immature. Recent studies show that the newborn retina is nevertheless sensitive to light. Indeed, intrinsically photosensitive retinal ganglion cells are present from birth and already make functional connections with the suprachiasmatic nucleus, the site of the central circadian clock.

Evelyne Sernagor

In vertebrates, visual processing starts at the back of the eye, where the retina converts light into neural signals. Images of our surrounding world are transduced by rod and cone photoreceptors, which generate neural responses that are processed through several layers of specialised neurones. Retinal ganglion cells