Neuronal latencies and the position of moving objects

Bart Krekelberg and Markus Lappe

Neuronal latencies delay the registration of the visual signal from a moving object. By the time the visual input reaches brain structures that encode its position, the object has already moved on. Do we perceive the position of a moving object with a delay because of neuronal latencies? Or is there a brain mechanism that compensates for latencies such that we perceive the true position of a moving object in real time? This question has been intensely debated in the context of the flash-lag illusion: a moving object and an object flashed in alignment with it appear to occupy different positions. The moving object is seen ahead of the flash. Does this show that the visual system extrapolates the position of moving objects into the future to compensate for neuronal latencies? Alternative accounts propose that it simply shows that moving and flashed objects are processed with different delays, or that it reflects temporal integration in brain areas that encode position and motion. The flash-lag illusion and the hypotheses put forward to explain it lead to interesting questions about the encoding of position in the brain. Where is the 'where' pathway and how does it work?

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In the flash-lag illusion, observers compare the position of a continuously moving stimulus to the position of a brief flash of light. When the moving stimulus and the flashed stimulus are physically aligned in space and time, observers nonetheless perceive a spatial offset: the moving stimulus appears advanced relative to the position of the flashed stimulus (Fig. 1).

Earlier reports of similar phenomena can be found as far back as the 1920s (Refs 5–7). But Nijhawan's rediscovery and new interpretation in 1994 created a surge of interest, certainly because we now know much more about the neural processes underlying perception, appreciate the problem of neural latencies and have psychophysical as well as neurophysiological tools to unravel the mechanisms that underlie these phenomena. This article discusses the properties of the flash-lag effect known from a host of studies carried out since Nijhawan's report. The hypotheses on the origin of the flash-lag effect will be discussed mainly in the light of these psychophysical findings. It is important, however, to put the psychophysical hypotheses on a neurophysiological basis, and review some of the pertinent data.

Motion extrapolation

Nijhawan has proposed that the visual system uses the motion signals to extrapolate the position of a moving object[1]. Accordingly, the representation faithfully reflects the position of the object at the time it is perceived and, as such, compensates for neuronal delays. Extrapolation cannot occur for a briefly flashed object, either because it does not move, or because its duration is too short to initiate extrapolation mechanisms. Hence, the flash is seen at its true position, but the moving object is seen at an extrapolated position (Fig. 2b). This extrapolated position is the position the moving object occupies 'now', not the position it occupied when the flash hit the retina: hence, the flash will appear to lag behind.

If motion extrapolation compensates for neuronal latencies, the magnitude of the lag-effect should depend on the actual latency of the moving object. Hence, a crucial test manipulates the latency of the moving object and determines whether the lag co-varies. Neuronal latency increases when stimulus luminance decreases[8,9]. Therefore, when the luminance of the moving object in the flash-lag illusion is decreased, the lag should increase to compensate for the increased latency. Contrary to this prediction, however, the lag decreases with a decrease in luminance[3] and can even turn into a flash-lead[10].

Moreover, motion extrapolation should become incorrect when a moving object abruptly reverses direction; it should yield apparent positions beyond the reversal point. This, however, is not observed experimentally[2,11,12]. The failure to compensate for latency variations correctly and the lack of a predicted overshoot has prompted several researchers to look for alternative explanations of the flash-lag effect.

Differential latencies

The differential latencies hypothesis proposes that the flash-lag effect occurs simply because the visual system responds with a shorter latency to moving stimuli than to flashed stimuli[2,10,11]. Thus, temporal coincidence of the neural representation of the two stimuli occurs when the moving stimulus is farther along its path rather than when both stimuli coincide physically (Fig. 2c).

The differential latencies hypothesis provides a natural explanation for the linear speed dependence of the flash-lag effect (Fig. 1c). The linearity simply reflects that the difference in latency is independent of speed. In addition, because a reduction in luminance of the moving object reduces the difference in latencies, this hypothesis easily incorporates the findings discussed above.

If a flashed object indeed has a longer latency than a moving object, one would expect this to be reflected in temporal order judgements. Eagleman and

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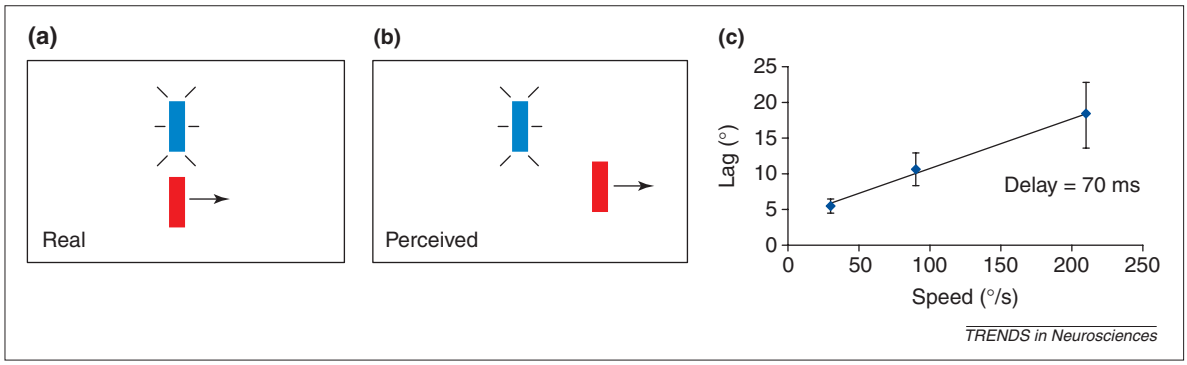

Fig. 1. The flash-lag illusion[1]. (a) The stimulus. A bar (shown in red) moves. A second bar (shown in blue) is flashed in perfect alignment. (b) The percept: the moving bar (red) appears to be ahead of the flashed bar (blue). Note that the illusion is not because observers fail to stick to the instructions and report the alignment of flash-offset (which could possibly be delayed as a result of persistence of the flash) with the moving stimulus. Whitney *et al.* convincingly demonstrated this by extending the flash-duration as well as by backward masking[2]. For technical reasons most studies have used rotating line segments rather than the linear motion shown here. The illusion is strong in both cases. Moreover, the flashed object does not have to be stationary. A flash-lag also occurs between a continuously lit moving object and another moving object that is visible only briefly[3] or intermittently[4]. (c) The magnitude of the offset depends linearly on the speed of the moving object. The slope of the speed dependence can be used to express spatial offsets as equivalent temporal delays. In Nijhawan's experiments[1], the perceived spatial offset corresponded to a time difference of about 80 ms, roughly the typical latency of early visual cortical neurones. In this figure, the equivalent delay is 70 ms (Fig. based on data published in Ref. 3).

Sejnowski have tested this by flashing a bar at the time a moving bar came to a halt. Subjects perceived the temporal order of these events correctly. But, as earlier experiments have shown that there is no flash-lag effect at motion offset[12], this experiment does not argue against differential latencies. It would be interesting to repeat the temporal order experiment for an object that remains in motion after the flash.

The differential latencies hypothesis loses some of its appeal when one considers the rich phenomenology of the flash-lag effect. In a set-up with repetitive sequences of flashes, increasing the number of flashes, the duration of the flash or the flash frequency all lead to an orderly decrease of the lag[3,4]. Moreover, flash-lag-like phenomena not only apply to motion but also to other dimensions of stimulus change[13]. In one experiment, the colour of a spot gradually changed from green to red. At some time during the change a second spot was flashed with exactly the same colour the changing spot had at that time. Subjects reported that the changing spot was redder than the flashed spot. The changing dot had 'moved' more than 400 ms along the colour dimension – much more than the equivalent temporal shift for position changes (~80 ms). Similar results, though with different temporal shifts, were obtained for changes in luminance, spatial frequency and even pattern entropy. To support the differential latencies hypothesis, independent evidence is needed that these different differential latencies really exist in each of these feature dimensions.

Attentional delays

Differences in latencies could arise not only from the properties of single neurones, but also from higher level processing. For example, the flash could trigger an attentional process to estimate the position of the flash. This process takes time and by the time the system gets round to judging the position of the moving object, the latter will have moved on and a spatial offset will be perceived[14–16].

To test whether attention plays a role in the flash-lag effect, several authors have used paradigms that compare the lag in situations where the occurrence of the flash is predictable or unpredictable. Unfortunately, the results are mixed: although some authors report that cueing[15] or predictability[23] of the flash reduces the lag, others claim that there is no attentional effect[17].

Temporal integration

The temporal averaging model proposes that the visual system collects position signals over time and estimates the position based on the integrated signals[18]. The flash-lag occurs because position information of the flashed stimulus is hypothesized to persist for some time[19] and therefore biases the position estimate towards the last seen position of the flash (Fig. 2d).

Unlike motion extrapolation, temporal averaging predicts an influence of post-flash changes on the percept. The finding that reversing[12] or slowing down[15] the moving object after the flash reduces the lag, confirms this. The data diverge on the answer of how long after the flash the trajectory of the moving object still influences the lag. Some experimental data show a temporal horizon of about 500 ms (Ref. 3), Brenner and Smeets report 150 ms, Eagleman and Sejnowski argue for an 80 ms window, and Whitney *et al.* suggest an even shorter 50 ms window[11] (albeit in combination with differential latencies). A 500 ms integration period is certainly longer than the reaction time for many natural tasks. For the temporal averaging model this implies that only part of the integration period is normally used. Eagleman and Sejnowski ascribe the ability to modify the integration window to the salience of the stimuli[20]. It would be interesting to put this hypothesis on a more solid footing.

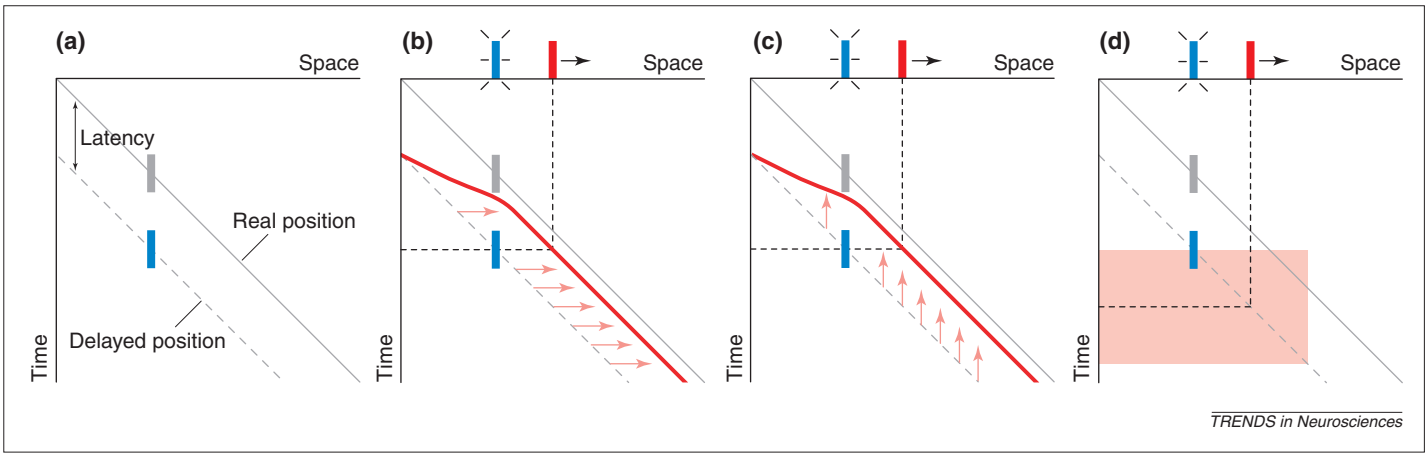

Fig. 2. Proposed explanations of the flash-lag effect. In a space–time plot, the changing position of a moving object is depicted as a diagonal line. The solid grey line in each plot represents the true position of the moving object. The grey bar is the true position of the flash, which appears for a short time and does not change position. The broken grey line shows the position of the moving object as it is seen by a neurone with a fixed latency. The offset between this line and the unbroken grey line reflects the delay introduced by the latency. The blue bar shows the delayed representation of the flash. It is perceived at the correct spatial position, but with a delay given by the latency. Elements in red depict how the perceived position of the moving object is accounted for in the different hypotheses. (b) The motion extrapolation hypothesis spatially shifts the representation of the moving object (arrows). This brings its position (unbroken red line) closer to the true position (grey line). At the time of the flash (unbroken grey line) the extrapolated position of the moving object is spatially ahead of the flash. Presumably, extrapolation requires some time to set up; hence, in the early parts of the trajectory, the object gradually shifts from the fully delayed to the extrapolated position. (c) The differential latencies hypothesis assumes that the latency is shorter for moving than for flashed objects. Again, the representation of the position of the moving object (red line) is pushed closer to its real position. Now the change is along the temporal rather than the spatial dimension (arrows). Here too, the reduced latency of a moving object can only make itself felt after the object has been moving for a while. This gradual reduction of the latency can be seen in the first part of the trajectory. (d) In the temporal averaging hypothesis and in postdiction all events are processed with the same latency. The position of the moving object is not determined instantaneously, but rather averaged over some time interval (red area). The perceived position is the mean position during this interval. The position signal of the flash is also averaged over time, but as the position does not change, the average position equals the actual position. The mismatch between the mean position of the moving object and the mean position (which equals the actual position) of the flashed object causes the flash-lag effect. In postdiction, the red time interval can be shifted depending on the saliency of the flash and moving object and the temporal integration can be more complicated than a simple average.

Postdiction

Recently, Eagleman and Sejnowski have extended temporal integration to a more general concept called postdiction. In postdiction, the brain constructs a percept by combining an internal model of the world (based on recent history) with the current external input. How the brain combines these sources of information depends on their respective salience. When a flash occurs, for example, the external input has high salience and partially resets the internal model of the world. While the internal model is being rebuilt, the moving object moves on; hence, it is perceived beyond the position of the flash. Once the internal model is 'ready', the percept is postdicted back to the time of the flash, hence the flash-lag. Precisely how internal models are set-up and which internal and external details are combined has not yet been clearly specified, and has recently led to considerable debate[20–24].

Neural mechanisms

Where in the visual pathways does the flash-lag effect occur? Although claims about neural substrates are still speculative, the psychophysical data have narrowed down the range of brain areas where one might look for neural substrates of the representation of the position of moving objects. Conversely, it is important to investigate possible neural mechanisms to narrow down the range of flash-lag interpretations.

Early visual processing

Conceivably, the spatiotemporal integration properties of early visual neurones might give rise to differential latencies for moving and flashed objects. Is there neurophysiological evidence that supports such a mechanism?

Orban *et al.* have used the spatial-lag method[25] to compare latencies to moving and flashed stimuli in the cat lateral geniculate nucleus (LGN)[26] and macaque medial temporal area (MT)[27]. In the LGN, the response latencies of neurones to moving light bars are indeed shorter than to the onset of flashed bars. The magnitude of the latency differences (~15 ms), however, is smaller than that required for an explanation of the flash-lag effect in humans (~45–80 ms)[1,2,10]. In macaque MT neurones[27], latencies to moving stimuli are even slightly longer than latencies to flashed stimuli (by 5 ms on average).

Even if the difference in latency is small (and of the wrong sign in MT), a moving stimulus will nevertheless elicit an earlier response. The reason for this is that, with extended spatial receptive fields, a moving stimulus enters the receptive field of a neurone before the flashed stimulus is turned on. A simple neuronal place code in which the response of a neurone signals the position of an object at its receptive field centre would therefore transmit the message that indicates the arrival of a moving object earlier than the arrival of a flash. Electrophysiological data from the retina[28] and from primary visual cortex[29] support this.

However, for an individual cell, the temporal advantage of a moving stimulus becomes smaller with increasing speed because it takes the moving bar less time to travel from the edge to the centre of the receptive field. This predicts that the flash-lag should

decrease with speed. Berry *et al.* claim that this is consistent with the properties of the flash-lag effect[28]. They point out that the flash-lag is higher for the low-speed stimuli of Nijhawan[1] than for the high-speed stimuli of Whitney and Murakami[2]. It seems, however, that differences in the experimental design between these studies caused the lag difference: two studies that directly measured the speed dependence of the flash-lag effect, in fact found a linear increase with speed[1,3] (see Fig. 1c). A linear increase might be achieved in a population code, for example, if receptive field sizes scale with speed preference.

Spreading activation

Lateral interactions in visual area V1 are thought to underlie many visual illusions[30]. Spreading activation could set up a bow-wave of activation around a moving object and enhance neural responses at future positions of the object. The focal attention model[31], as well as the temporal integration model[18], use this to explain the large spatial range of the flash-lag effect. The temporal integration model, however, also requires a temporal integration window[18] of ~500 ms – much longer than that observed for lateral spreading in striate cortex[32] (~100 ms). It is unclear where in cortex this long temporal integration could take place.

Higher level

The previous paragraphs show that the flash-lag effect is unlikely to be completely understood in terms of low-level visual processing. Latency differences and spatiotemporal interactions, however, could also be caused by processes beyond striate cortex. Some psychophysical findings indeed also point towards higher-level cortical origins of the flash-lag effect. Schlag *et al.*[33] asked subjects to fixate a head-fixed target while rotating their head left to right. (Therefore, the target is stationary on the retina, but it moves in space, and is perceived to move in space.) When a second target is flashed aligned with the first, subjects perceived a spatial lag[33]. In this experiment, perceived motion of the target is created solely from an extra-retinal signal of head movement[34]. Therefore, although retinal motion might be sufficient[35,36], it is not necessary for a flash-lag effect to occur. This indicates that the origin of the flash-lag effect must lie beyond the point of integration of retinal and extra-retinal signals. The findings of Sheth *et al.*[13] suggest that the representation of changing stimuli uses common mechanisms across stimulus qualities. This implicates cortical areas beyond V1 and suggests that these mechanisms might be present in many cortical areas. Both the latency differences hypothesis and temporal integration could be extended to encompass higher level processing. Such models, however, are increasingly less amenable to physiological scrutiny.

Position encoding in a changing world

The data reviewed do not unequivocally point to a single explanation of the flash-lag effect. Nevertheless, motion extrapolation is an unlikely possibility. The influence of differential latencies on the perception of the position of moving objects, however, is undeniable. Moreover, there is no doubt that some neurones respond faster to one type of stimulus than to another. Whether the differences in latency found in early visual areas are sufficient to explain all aspects of the flash-lag effect is, however, questionable. Temporal integration deals naturally with the long-term influence of the trajectory of an object on its perceived position and could easily be extended to other feature dimensions. The time window over which this integration takes place and whether it should be extended to such a general concept as implied in postdiction, however, is not yet known.

Given that the ability to localize moving objects in the visual world is such an important aspect of our daily lives, it is somewhat surprising that so little is known about its neural implementation. Maybe the reason for this is that there seems to be an almost trivial solution: cells in early visual cortex respond only when visual objects are inside their small receptive field. Thus, in principle, these cells have all the information that is needed to signal where an object is. The flash-lag effect is a compelling demonstration that there is more to position encoding than simple spatial receptive fields. First, this visual illusion brings home the point that, in a changing environment, processing latencies influence perception: the question of when a cell encodes a stimulus becomes as important as where the receptive field of the cell is. Second, the flash-lag shows that the perception of position depends on the trajectory of an object. This spatiotemporal influence of the trajectory can extend over many degrees of the visual field and over hundreds of milliseconds. In neural terms, this presumably reflects the importance of lateral interactions and local feedback loops. It shows that setting up a neural representation of position involves more than taking a snapshot of the environment and progressively passing this up through the hierarchy of visual areas.

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Properties and modulation of mammalian 2P domain K^+ channels

Amanda J. Patel and Eric Honoré

Mammalian 2P domain K^+ channels are responsible for background or 'leak' K^+ currents. These channels are regulated by various physical and chemical stimuli, including membrane stretch, temperature, acidosis, lipids and inhalational anaesthetics. Furthermore, channel activity is tightly controlled by membrane receptor stimulation and second messenger phosphorylation pathways. Several members of this novel family of K^+ channels are highly expressed in the central and peripheral nervous systems in which they are proposed to play an important physiological role. The pharmacological modulation of this novel class of ion channels could be of interest for both general anaesthesia and ischaemic neuroprotection.

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Leak or background K^+ selective channels – defined by a lack of voltage- and time-dependency, and with a linear current to voltage relationship in a symmetrical K^+ gradient – play an essential role in setting the resting membrane potential, tuning the action potential duration and modulating the responsiveness to synaptic inputs. Regulation of background K^+ channels by neurotransmitters and second messengers is central for synaptic function[1,2]. The most extensively studied native background K^+ channel is the S channel in the marine snail *Aplysia* sensory neurones[1]. Closing of the S-type background K^+ channel by 5-HT receptor activation is involved in presynaptic sensitization, a simple form of learning[2]. Additionally, neuronal background K^+ channels are the targets of an important class of pharmacological agents, the volatile general anaesthetics[3–7].

Mammalian K^+ channel subunits (~80 genes) can be divided into three main structural classes comprising two transmembrane segments (TMS), four-TMS or six-TMS (Ref. 8). The common feature of all K^+ channels is the presence of a conserved motif called the P domain, which is part of the K^+ conduction pathway[9]. The two-TMS channels comprise a single P domain and encode the inward rectifiers. These K^+ channels, which operate at negative membrane potentials, contribute to the setting of the resting membrane potential. The six-TMS channels, including the voltage-gated and the Ca^{2+}-activated K^+ channels, similarly comprise a single P domain. These channels, which are usually activated at depolarized membrane potentials, mostly contribute to the repolarization of the action potential. By contrast, the most recently discovered class of four-TMS subunits is characterized by the

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