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Very Short Term Visual Memory Via Reverberation: A Role for the Cortico-thalamic Excitatory Circuit in Temporal Filling-in During Blinks and Saccades?

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There is a large projection of neurons from Layer VI of V1 that makes excitatory connections on LGN relay cells. It has been proposed that this circuit is involved in signal processing and thalamic sensitivity regulation. Alternatively, Crick has suggested that the circuit could be a reverberatory loop—a site for very short term (iconic) visual memory. This hypothesis is shown to be plausible if the reverberation is keyed to the onset of neurally initiated visual disruptions such as blinks and saccades. Neural mechanisms suppress perception during these events but little is known about temporal filling-in processes analogous to the mechanisms that fill-in spatial scotomas. Crick's reverberatory loop could provide a process for filling-in temporal scotomas with information acquired just before the disruption, thus maintaining the continuity of visual experience. © **1997 Elsevier Science Ltd. All rights reserved.**

LGN Cortex Saccadic suppression Temporal scotomas Filling-in

INTRODUCTION

Although a simplification, the notion of the lateral geniculate nucleus (LGN) as a simple relay in the retino-geniculate-cortical pathway is attractive. The deceptively similar numbers of primate retinal ganglion and LGN relay cells (Connolly & van Essen, 1984), the high ratio of relay cells to interneurons in the primate LGN (about 4:1; Garey *et al.*, 1991) and the preservation of retinal receptive field organizations in LGN cells (Casagrande & Norton, 1991) all reinforce the relay perspective. Considerable evidence exists that the LGN is something more. A striking clue is the enormous number of fibers contacting the LGN from the cortex (cortico-thalamic neurons may outnumber thalamo-cortical neurons by an order of magnitude; Sherman & Koch, 1986), mostly excitatory neurons from layer VI of V1. These cortico-geniculate fibers are organized topographically, and appear to divide into distinct populations, projecting to the parvocellular and magnocellular LGN layers (Lund *et al.*, 1975). Several suggestions for the role of the cortico-geniculate pathway have been made. Here, one possibility—reverberation—is shown to have the properties required for filling-in visual disruptions such as blinks and saccades.

BACKGROUND

Although the nature of the cortico-geniculate pathway is unclear, several intriguing suggestions have emerged. First, the cortical projection may act to regulate the sensitivity of thalamic neurons. Such a mechanism could play many roles from regulating system gain to mediating selective attention. In most of these theories, cortical excitatory connections on geniculate units lower the threshold for retino-geniculate transmission. If done selectively, features in the optic array can be enhanced. This attentional "searchlight", originally proposed by Crick (1984) for the reticular complex, generalizes to other thalamic nuclei (see Sherman & Koch, 1986; Koch, 1987; and Casagrande & Norton, 1991 for discussion of similar hypotheses).

Second, some investigators have proposed that the thalamus is a dynamic blackboard (Mumford, 1991) or volatile sketchpad (Harth *et al.*, 1987) on which the cortex writes its last best estimate of the visual stimulus. In this view, the thalamus acts like a spatially dislocated seventh layer of cortex. Images, after being processed and enhanced by cortex, are briefly stored in thalamus, where they are available to other areas of the cortex.

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Related theories postulate a role for the thalamus in stereopsis and in synchronization of cells responding to common features (Singer, 1977; Sillito *et al.*, 1994). These intriguing ideas have at least two problems. First, to avoid interference with the LGN's primary role as a visual relay, the backprojection from cortex to LGN would have to be spatially organized to coincide with the forward projection. There is evidence in cats that the cortico-thalamic projection is at least coarsely organized; Tsumoto *et al.* (1978) found that adding glutamate to locations in Layer VI facilitated the response of LGN cells whose receptive fields were within a few degrees of the retinotopic coordinates of the affected areas. Murphy & Sillito's (1996) anatomical study also shows a retinotopic projection, but the corticofugal cells have terminal fields that are extensive enough to influence geniculate regions well beyond those that constitute the corresponding inputs to the cortex. If the projection is not exact, blur (in the case of random displacement and large terminal fields) and smear (in the case of systematic displacement) would result from cortically processed information being added to relayed information. A second problem with the information processing hypothesis is that LGN and retinal ganglion cells have similar spatial tuning properties, whereas cortical cells have narrower tuning. If the LGN receives cortically processed data, one might expect LGN cells to have apparently narrower bandwidths than retinal cells (see, however, Cuderio & Sillito (1996) for evidence of a cortico-geniculate influence on the center/surround balance of LGN cells). This has not been observed, although it is possible that this system is compromised under some experimental conditions (e.g., anesthesia).

Alternatively, the excitatory nature of the Layer VI–LGN pathway suggests that the LGN–V1–LGN circuit could be reverberatory, with information traveling around the cortico-thalamic loop in relatively unaltered form. Crick (1994, pp. 240–241) suggests that a reverberatory loop provides a mechanism for very short term visual memory. Crick argues that the Layer VI–LGN pathway is suitable for this purpose because its lack of lateral connections (Tombol, 1984) would prevent the spread of memory into unintended circuits. Unfortunately, in addition to the spatial superposition problems discussed above, cortico-thalamic reverberation would interfere with the thalamic relay function by acting as a temporal lowpass filter*; the output of a LGN cell to Layer IV of V1 would be the weighted sum of its output to the current retinal information plus prior retinal information delayed by passage from LGN to V1/Layer VI and back to LGN. Reverberatory lowpass filtering could cost the visual system temporal resolution. If the LGN–V1–LGN loop time (Δt) is about 25–50 msec (Mumford, 1991; Swadlow, 1983), then the Nyquist frequency for the system is $1/2\Delta t$, or 10–20 Hz. This range of cut-off frequencies is low compared to perceptual flicker fusion frequencies of 40–80 Hz for moderate to high levels of illumination (Kelly, 1961). Apparently, the cortico-thalamic system does not reverberate during active viewing. However, this does not preclude a role for reverberation when visual data collection is briefly disrupted.

*The lowpass filtering nature of reverberation can be made explicit by expressing the reverberation as a difference equation similar to that used to recursively model the output of a lowpass filter: $G(t) = AG(t - \Delta t) + (1 - A)R(t)$, where $G(t)$ is the output of the LGN with reverberation, $R(t)$ is the retino-geniculate relay signal from the optic nerve, and A is an attenuation factor. If $A \propto e^{-a\Delta t}$, then this is an excellent approximation of exponential lowpass filtering (Fleet *et al.*, 1984). The loop time Δt determines the highest frequency the system can adequately represent ($F = 1/2\Delta t$). Δt is poorly studied in primates but Swadlow (1983) and Mumford (1991) give estimates of 25–50 msec (Nyquist frequencies of 10–20 Hz). Note however that these estimates are based on average conduction times and that there is substantial variation of conduction velocity of axons, even in the same pathways (Swadlow, 1983).

THEORY

Suppression and filling-in of temporal scotomas

Visual data collection is disrupted several times a second by fast saccades and several times a minute by blinking. These temporal scotomas are seldom noticed. Saccades, which can take place about every 300 msec, can reach extraordinary velocities (top velocities of 800–1000 deg/sec in humans for large amplitude saccades; Hyde, 1959; Alpern, 1982), badly blurring all but the shortest duration stimuli (Volkmann, 1962). Blinking occurs every few seconds, covering the pupil and darkening the retina for 100–150 msec. Experiments simulating the effect of a blink show that the blinks should be very noticeable (Riggs *et al.*, 1981). The fact that neither blinks nor saccades are noticed—either as perceptual degradation or as events (try seeing either in a mirror)—suggests a system that alters perception during neurally initiated visual disruptions. These alterations probably include both suppression of retinal information and filling-in of the resultant temporal scotoma. Suppression mechanisms increase detection thresholds by 0.4 to 2.0 log units during blinks and saccades (Volkmann, 1962, 1986; Volkmann *et al.*, 1980). The time courses (and other properties) of both suppression mechanisms are similar—beginning a few tens of milliseconds before the disruption and persisting for 100–200 msec—suggesting a common mechanism (Volkmann, 1986). Several lines of evidence suggest that the site of suppression is in the LGN, possibly restricted to the magnocellular layers (see Discussion). Although necessary, mere suppression of light decrements or blur is not sufficient to explain perceptual continuity during visual disruptions. It seems necessary to postulate an active filling-in mechanism (see Discussion) such as cortico-thalamic reverberation to bridge the gap between periods of actual viewing. Analogous mechanisms fill in spatial scotomas; e.g., textures and colors that surround blind spots and artificial scotomas are perceived as continuous

throughout the visual field and broken lines are completed (Ramachandran, 1993).

Anatomical and electrophysiological correlates of reverberation

To avoid disruption of ordinary vision, reverberation should be inhibited, except at the initiation of a blink or saccade. Given evidence for multiple roles for cortico-thalamic feedback, the most likely control site is Layer VI of V1. Similarly, to avoid disruption of reverberation, blink and saccadic suppression should take place at the transmission site from retinal ganglion cells to LGN relay cells. Thalamic information sent to the cortex just before the disruption would be relayed by Layer VI neurons back to the LGN. A few loops through the system would serve to fill in all saccades and most blinks. The anatomy of the cortico-geniculate pathway is consistent with this hypothesis. Cortico-thalamic cells contact LGN relay cells both directly and indirectly, from contacts made on local inhibitory interneurons and neurons of the thalamic reticular nucleus (RNT), which in turn contact LGN relay cells. Synapses from cortico-thalamic cells dominate the distal dendrites of relay cells, while synapses from retinal neurons, LGN interneurons, brainstem and RNT neurons are concentrated in the proximal portions of the dendritic tree (see Sherman & Koch, 1990 for a review). These relay cells are electrotonically compact and even distally located inputs are relatively unattenuated. Koch (1987) has suggested that if the receptor for the retino-geniculate synapse is of the nonlinear NMDA variety, this arrangement can act as a logical gate, selectively controlling the ability of retinal neurons to excite LGN relay cells. This mechanism seems ideal for saccadic suppression. Moreover, synapses from inhibitory LGN interneurons, RNT and brainstem neurons are in close proximity to the synapses of the retinal afferents and may play a role in regulating retinogeniculate information transfer; electrical stimulation of the pontine and mesencephalic reticular formation has much the same effect on LGN activity as saccades (Bartlett *et al.*, 1976). At the same time, cortico-thalamic excitatory synapses could drive the LGN relay cells. Electrophysiological evidence in primates, although sketchy, is supportive of a reverberatory model. Bartlett *et al.* (1976) implanted permanent electrodes in the optic tract, LGN, optic radiations and V1 of several monkeys. By applying electric shocks in the optic tract, LGN and optic radiations and measuring potentials evoked in optic radiations and V1 during and between saccades, Bartlett *et al.* were able to measure the excitability of segments of the retino-geniculate-cortical pathway. For 100 msec after the beginning of a saccade, transmission of shocks applied to the optic tract and measured in the optic radiations were reduced 5–54%, followed by a period of slight (5–15%) enhancement of sensitivity. The most dramatic reductions in sensitivity were in the magnocellular pathway, which ranged from 8 to 54% compared to 5–21% reductions in sensitivity for parvocellular neurons. Sensitivity changes were greatest for animals viewing patterned stimuli, and least for animals tested in total darkness. These indications of retino-geniculate signal regulation are roughly consistent with psychophysical studies of saccadic suppression discussed earlier and with similar electrophysiological studies in cat (Noda & Adey, 1974; Noda, 1975). A somewhat different picture emerges for the excitability of LGN relay fibers in the optic radiations for electric shocks applied in LGN. Geniculo-cortical excitability was reduced by only 4–17% during the first 60 msec after a saccade was initiated, followed by an increase in sensitivity of 11–122% for the 60–100 msec period. This combination of suppressing retino-geniculate transmission while enhancing geniculo-cortical excitability is intriguing. If the LGN–V1–LGN loop time is close to Mumford's (1991) 50 msec estimate, then the period of enhancement of sensitivity of LGN relay axons would coincide roughly with the rearrival of information acquired immediately prior to the suppression period.

DISCUSSION

Parvo/magno distinctions and filling-in mechanisms

The electrophysiological parvo/magno differences reported for saccadic suppression are supported by a growing body of psychophysical studies showing more suppression for the magno system for than the parvo system during temporal scotomas (Bartlett *et al.*, 1976; Ridder & Tomlinson, 1993, 1995; Burr *et al.*, 1994; Uchikawa & Sato, 1995). The physiological basis for this phenomenon is unknown, but may be related to evidence that magno relay cells receive far more input from inhibitory interneurons than parvo cells do (Winfield, 1980). This raises two interesting questions. First, is it really necessary to postulate an active filling-in process? After all, if the magno system (which is well suited to detecting the initial intensity and contrast changes induced by blinks and saccades) is more suppressed than the parvo system during temporal scotomas, then perhaps the role of suppression is to prevent detection of the onset of the scotoma. Then, sustained activity in the parvo system (believed to mediate perception of long duration afterimages; Ingling & Grigsby, 1990) might suffice to fill-in neural disruptions. However, this explanation is belied by the immediate loss of vision during nonblinking eyelid or artificial aperture closure, suggesting that an active mechanism mediates temporal filling-in. Another indication of an active mechanism is a slight enhancement of sensitivity for high spatial frequencies and for chromatic patterns (both putatively parvo functions; Burr *et al.*, 1994) during saccades. Additionally, the Riggs *et al.* (1981) experiments discussed below suggest that an incomplete filling-in occurs for some blinks. Active processes are known to occur in filling-in spatial scotomas; lines broken by a scotoma are completed in 4–5 sec, with misaligned parts moving slowly and perceptibly into alignment (Ramachandran, 1993)—indicative of a more sophisticated mechanism than that proposed for temporal filling-in. A second question is raised by the possible consequences of filling-in for

motion processing mechanisms. However, temporal filling-in works, it does not appear to cause directionally dependent cells that were active prior to a scotoma to continue to respond during the scotoma, otherwise motion extrapolation might occur. For example, there is a period of no motion during the blink scotoma. If you observe your blinks in a mirror, you see your eyelids come down—stop just above the pupil for a short period of time—then pop back up. You never paradoxically observe the reflection of your eyelids covering your eyes. Yet, directionally dependent motion sensitive cells should be triggered by the movement of the eyelids, and if their geniculo-cortical inputs are fed by the filling-in mechanism, then motion extrapolation should be possible. There are several possibilities why it might not occur. First, filling-in might be a predominately parvo phenomena, with only limited parvo inputs to the motion system. Evidence is sparse on this point; Bartlett *et al.* (1976) note that retinogeniculate transmission is more suppressed in magno cells, but do not address parvo/magno distinctions in geniculo-cortical excitability. Second, magno filling-in might be similar to parvo, with cortical motion systems actively inhibited during blinks and saccades. Finally, there might be a desynchronization of the form and motion systems during temporal scotomas, disrupting visual integration processes (Zeki, 1993).

Some proposed experiments

The hypothesis that cortico-thalamic reverberation fills in temporal scotomas is testable in primates. First, the theory predicts that firing rates in cortico-thalamic and thalamo-cortical neurons should become correlated (with a relative phase difference dependent on conduction velocities and locations of measurement) during blinks and saccades, roughly paralleling (see below) the time course of saccadic or blink suppression. Second, activity in LGN relay cells can be measured during blinks and saccades with cortico-thalamic feedback active or deactivated (this can be done either reversibly or irreversibly). Because suppression and temporal filling-in mechanisms appear to operate properly only for neurally initiated disruptions, and because it is suspected that anesthesia may inhibit the cortico-thalamic pathway (Livingstone & Hubel, 1981), humane ways should be found to conduct these experiments in alert animals, perhaps using permanently implanted electrodes (Bartlett *et al.*, 1976). Special attention should be paid to parvo/magno distinctions and to the time course of filling-in. Blinks (but not saccades), if attended, can be noticed as a very brief sensation of flicker. This sensation is psychophysically matched to a very short duration luminance decrement (but not to an equal energy long duration decrement; Riggs *et al.*, 1981). This suggests that the time course of filling-in and/or suppression is slightly mismatched to the time course of the blink. Similarly, there are anecdotal reports that some Parkinson's disease patients do perceive their own blinks and saccades. Primate models of Parkinson's disease exist and electrophysiological correlates of these perceptual phenomena can be sought.

Time-sharing the cortico-thalamic pathway

This temporal filling-in process does not exclude other roles for the cortico-thalamic pathway. During ordinary vision, the same pathway might be used to modulate or gate visual data or to fine tune the spatiotemporal properties of geniculate neurons (Singer, 1977; Sherman & Koch, 1986; Koch, 1987; Cuderio & Sillito, 1996). The two mechanisms (regulation and reverberation) could share the cortico-thalamic pathway—one active during data collection, the other during visual disruption—a form of time division multiplexing. Finally, the pathway might be involved in vivid sensory imagery—a role consistent with increased LGN activity in cats during REM sleep (Bizzi, 1966; Steriade *et al.*, 1989) and with neuro-imaging research linking thalamic dysfunction to schizophrenia (Andreasen *et al.*, 1994).

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