Control of Recurrent Inhibition of the Lateral Posterior-Pulvinar Complex by Afferents from the Deep Layers of the Superior Colliculus of the Rabbit

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Zhu, J. Julius and Fu-Sun Lo. Control of recurrent inhibition of the lateral posterior-pulvinar complex by afferents from the deep layers of the superior colliculus of the rabbit. J. Neurophysiol. 80: way may provide an attentional "window" for the primary visual cortex to select and route important visual information to the higher cortical areas.

In the retino-geniculo-cortical pathway, LGN serves as a gate or a filter, which controls and modulates the visual information en route to the cortex (Sherman and Guillery 1996; Sherman and Koch 1986). The "gating mechanisms" rely heavily on the overwhelming extraretinal inputs received by LGN. One such input is the recurrent inhibitory input, which is provided by the recurrent interneurons located in the visual sector of the thalamic reticular nucleus. The recurrent inhibitory circuits themselves are also under the influence of other extraretinal inputs, and these inputs can dramatically alter the recurrent inhibition patterns in LGN relay cells by changing the activity of the recurrent interneurons (Ahlén et al. 1984; Sherman and Guillery 1996; Sherman and Koch 1986; Singer 1977). For example, the recurrent inhibitory circuit of LGN is regulated by the afferents from the deep layers of SC (Lo 1988; Lo and Xie 1987b; Zhu and Lo 1995). When predorsal bundle-projecting cells in the deep SC are excited to initiate a gaze shift (e.g., a saccadic eye movement), they also exert an excitatory effect on the recurrent interneurons for LGN via the central lateral nucleus of the thalamus (CL). Activation of these recurrent interneurons will then elicit a prolonged inhibition in relay cells of LGN (Lo 1988; Lo and Xie 1987b; Zhu and Lo 1995, 1996b).

Although the functional role of LGN as the gate and filter of the primary visual ascending pathway is well established, it is still unclear whether LP services similar roles in the extrageniculate pathway. However, anatomical evidence suggests that LP is also targeted by numbers of extraretinal projections (Patel and Bickford 1997; Sherman and Guillery 1996). In addition, single unit recordings have shown that visual responses of LP cells are suppressed during the saccadic eye movements, presumably due to the modulatory effects of the extraretinal inputs (Benevento and Port 1995; Robinson et al. 1991). In this study, we report that LP cells are inhibited by the afferent inputs from the deep SC and this effect is mediated by the recently identified recurrent inhibitory circuit of LP (Zhu and Lo 1996a, 1997).
METHODS

Male New Zealand rabbits weighing 2.3–3.0 kg were anesthe-
tized by an intravenous injection of pentobarbital sodium (Nembutal, 40 mg/kg). Supplemental doses (10 mg/kg) were given as needed to maintain the animals free from pain reflex and/or in a state of slow-wave sleep, as determined by monitoring the cortical electroencephalograph (EEG). All pressure points and incisions were infiltrated with lidocaine. During recording, the rabbits were also immobilized by intermittent administration of gallamine triethiodide (Flaxedil, 3–5 mg·kg⁻¹·h⁻¹) and were respirated artificially. End-tidal CO₂ of animals was kept in a range of 3.8–4.5%. Body temperature was maintained within normal range (37.3 ± 0.3°C).

Four silver electrodes were fixed on the surface of the primary visual cortex (Cx), so that we could record EEG and evoked potentials from stimulation of the optic chiasm (OX). Several insulated stainless steel electrodes, with an exposed tip of 60 μm in diameter, were stereotaxically inserted into the ipsilateral SC (coordinates: P11, L3, D7), OX (coordinates: A3, L1, D14), and contralateral predorsal bundle (coordinates: P13.5, L1.5, D11.5) according to the atlas of Sawyer et al. (1954). The final electrode tip position for OX stimulation was ascertained by finding the position where the threshold of evoked cortical potentials was <50 μA. The final positions of the SC and predorsal bundle stimulating electrode tip were determined by evoking visible saccadic eye movements at the lowest stimulating current, ranged from 40 to 70 μA for three-pulse stimulation at 300 Hz and ranged from 150 to 220 μA for single-pulse stimulation. This task was performed before the recording and injection of Flaxedil and when the animals were in a lighter anesthetized state (i.e., EEG began to show a tendency of increasing frequency and decreasing amplitude). The rabbits did not appear disturbed during the task, which typically took only a few minutes. Supplemental Nembutal was given right after the task. Stimulating electrode tracks were reconstructed after conventional histological processing. In some experiments, the SC stimulating electrode was moved up and down to plot threshold-depth curves. The test pulse was typically composed of a negative pulse (200 μs, 5–250 μA) passing through stimulating electrodes. Extracellular and intracellular recordings were made in the ventral part of the visual sector of the ipsilateral TRN (coordinates: P3.5, L6, D8.5) and LP (coordinates: P4.5, L4, D7). Recurrent inhibitory interneurons in the extraglomerular pathway were identified by the criteria described before (Zhu and Lo 1997). Because relay cells in LP project to the primary visual cortex (Townes et al. 1982; Weyand and Swadlow 1986), they were first identified by antidromic invasion from the visual cortex and then confirmed by their histological locations. Recording microelectrodes were filled with 3 M NaCl, 3 M K-acetate or 2% Pontamine sky blue dye in 0.5 M K-acetate solution. The resistance of micropipettes ranged from 20 to 35 MΩ. Pontamine sky blue was injected by passing negative current (5–10 μA, 5–10 min) to localize positions of recorded neurons. Recording electrode tracks were reconstructed after conventional histological processing. Electrical signals were displayed on an oscilloscope and recorded photographically. Each photographic record contained four to six superimposed sweeps.

RESULTS

We first performed intracellular recording from 23 relay cells in LP to examine whether SC stimulation can induce an inhibitory postsynaptic potential (IPSP) in LP cells. We then recorded extracellularly from 31 reticular cells and intracellularly from 9 reticular cells in the ventral part of the visual sector of TRN to study whether the same SC stimulation can evoke an excitation in these reticular cells and whether the excitation is responsible for the IPSP in LP cells.

Responses of relay cells of LP to SC stimulation

We identified 74 relay cells in LP in extracellular recordings. All of them showed an antidromic spike (Fig. 1A) with a fixed latency and a clear initial segment spike-somadendritic spike (IS-SD) notch in response to Cx stimulation. The antidromic latency ranged from 3.8 to 10.8 ms, with a mean of 7.4 ± 1.9 (SD) ms (Fig. 2B). They also responded to OX stimulation trans-synaptically at jittering latencies. The response latency to OX stimulation ranged from 2.7 to 9.3 ms, with a mean of 5.9 ± 1.8 ms (Fig. 2A). The antidromic invasion from Cx was further confirmed by collision test (Fig. 1A, 2–4). If Cx stimulation was preceded by an OX stimulation at a proper interval, the antidromic spike was blocked. A small increase in the OX-Cx interval resulted in a partial recovery of the antidromic spike, while further increase in the OX-Cx interval resulted in a complete recovery. All recorded cells were histologically localized in LP, one of them is shown in Fig. 1B.

Twenty-three LP cells were impaled after the extracellular recording. The mean resting membrane potential of relay cells in LP was −60.8 ± 3.5 mV. Stimulation of SC induced an IPSP in each of the relay cells (Fig. 1C, top). The latency of the IPSP ranged from 3.1 to 6.1 ms with a mean value of 4.6 ± 0.6 ms. Stimulation of the contralateral predorsal bundle, the axonal pathway of predorsal bundle-projecting cells in SC, induced a similar IPSP (Fig. 1C, bottom) with a mean latency of 5.2 ± 0.9 ms (ranging from 3.7 to 6.8 ms). The averaged latency difference between the IPSPs from the two sites was 0.6 ms, implying that the IPSP in LP cells resulted from the activation of predorsal bundle-projecting cells in SC. This was supported by plotting threshold-depth curves for 6 LP cells. Figure 1D shows one such threshold-depth curve in which the most effective sites (threshold <50 μA) for inducing IPSP in that cell are located in the deep SC, the same place where predorsal bundle-projecting cells are located (Zhu and Lo 1995). This was also true for five other LP cells.

There was a linear correlation between the IPSP latency from SC stimulation and that from OX or Cx in LP cells (Fig. 2). These linear correlations were statistically significant, and the regression formulas were: y = 0.27x + 3.00 (r = 0.60, P < 0.005) and y = 0.24x + 2.83 (r = 0.58, P < 0.005), respectively. The results suggest that if an LP cell receives inhibitory inputs from the deep SC via a fast conducting pathway, it will also be connected with the retina or cortex via fast conducting fibers and vice versa. Namely, there is a specificity of conduction velocity in the extraglomerular visual pathway.

The IPSP in LP cells had a mean duration of 140.2 ± 19.6 ms (n = 23). Sometimes, a rebound burst of spikes followed the preceding IPSP (Fig. 3B, top). Holding the membrane potential over −75 mV by injecting hyperpolarizing current reversed only the early part of the IPSP but not the later part (Fig. 3B, bottom), suggesting the involvement of both γ-aminobutyric acid A-type (GABA_A) and B-type (GABA_B) receptors in the IPSP (Bal et al. 1995; Crunelli et al. 1988; Huguenard and Prince 1994).
Fig. 1. Location and responses of a lateral posterior-pulvinar complex (LP) relay cell to stimulation of the deep layers of the superior colliculus (SC). A: antidromic responses to visual cortex (Cx) stimulation. Note that the antidromic spikes had a fixed latency (A1) and were confirmed by collision test (A, 2–4). Arrowhead points to the initial segment spike–somasensory spike (IS-SD) notch. B: location of the relay cell in the LP. C: stimulation of the SC or predorsal bundle induced an inhibitory postsynaptic potentisl (IPSP) in the LP cell. Resting membrane potential of the cell was −65 mV. Scale bars in C also apply to A. D: depth-threshold curve for the IPSP evoked by SC stimulation. Note that the most effective stimulating sites were located in the deep SC and the lowest threshold was as low as 11 µA. SC or predorsal bundle stimuli are marked by dots, whereas Cx stimuli, by triangles. OX, optic chiasm; PDB, predorsal bundle; MD, medial dorsal nucleus; LGN, dorsal lateral geniculate nucleus; OT, optic tract; II, stratum opticum of superior colliculus; IV, stratum griseum intermediolateral; VI, stratum griseum profundum (all symbols and abbreviations are the same for following figures).

Responses of recurrent inhibitory neurons in the LP-cortical pathway to SC stimulation

We made extracellular recordings from 31 reticular cells. All of them responded to OX and Cx stimulation with a burst of spikes (Fig. 4A). The response latency to OX stimulation ranged from 3.5 to 7.2 ms with a mean of 4.9 ± 1.0 ms (Fig. 5A), while that to Cx stimulation ranged from 4.0 to 10.9 ms with a mean of 7.2 ± 1.8 ms (Fig. 5B). For each cell, the response latency to Cx stimulation was always longer than to OX stimulation. In addition, all recorded cells were located in the ventral part of the visual sector of TRN (Fig. 4B). Therefore these cells met the criteria for the recurrent inhibitory neurons in the extrageniculate pathway (Zhu and Lo 1997).

These reticular cells also responded to stimulation of SC with a burst of spikes (Fig. 4C, top). The response latency ranged from 1.5 to 4.5 ms with a mean value of 3.0 ± 0.4 ms (n = 31). Stimulation of the contralateral predorsal bundle induced a similar burst of spikes in these reticular cells (Fig. 4C, bottom). The response latency ranged from 1.9 to 4.9 and had a mean value of 3.5 ± 0.5 ms (n = 31). Similar to the IPSPs in LP cells, the latency difference from the two stimulation sites was 0.5 ms on average. These results suggest that the burst response in reticular cells also results from the activation of predorsal bundle-projecting cells in SC. We thus further measured the threshold-depth curve for seven reticular cells. We found that the most effective sites (threshold <50 µA) were located in the deep SC (Fig. 4D).

In reticular cells, the response latency to SC stimulation exhibited a linear correlation with that to OX stimulation (Fig. 5A) or with that to Cx stimulation (Fig. 2B). For the former correlation, the regression formula was $y = 0.47x + 0.66$ ($r = 0.74$, $P < 0.005$), whereas for the latter, the regression formula was $y = 0.24x + 1.22$ ($r = 0.65$, $P <$...
0.005). These results confirm the specificity of conduction velocity in the extrageniculate pathway.

Of the 31 recorded reticular cells, 9 were impaled after extracellular recording (Fig. 6). The resting membrane potential of these cells was $-66.2 \pm 4.1$ mV. Stimulation of SC or the predorsal bundle evoked an excitatory postsynaptic potential (EPSP; Fig. 6B). The latency of the EPSP from SC stimulation ranged from 1.1 to 4.0 ms, with a mean of $2.3 \pm 1.0$ ms ($n = 9$), whereas that from the predorsal bundle stimulation ranged from 1.5 to 4.3 ms, with a mean of $2.8 \pm 0.8$ ms ($n = 9$). The latency difference from the velocity in the extrageniculate pathway.

The EPSP always was followed by a long IPSP (Fig. 6, B and C) lasting for $143.3 \pm 24.0$ ms ($n = 9$). This IPSP was expected because reticular cells form mutual inhibitory connections (Ahlsén and Lindström 1982; Cox et al. 1996; Lo 1985; Spreafico et al. 1988; Yen et al. 1985; Zhu and Lo 1997). To determine the nature of the IPSP, we hyperpolarized the membrane potential over

![FIG. 2. Linear correlation between orthodromic latencies from SC stimulation and orthodromic latencies from OX (A) or antidromic latencies from Cx (B) stimulation in LP relay cells.](image)

![FIG. 3. Intracellular responses of a LP relay cell to stimulation of the deep layers of SC. A: location of the LP cell (*). B: intracellular responses of the LP relay cell to SC stimulation at different holding potentials. Resting membrane potential of the cell was $-59$ mV. Note that the IPSP was followed by a rebound burst at the resting membrane potential and the early part of the IPSP was reversed at $-81$ mV. Action potentials are truncated.](image)
FIG. 4. Location and extracellular responses of a recurrent interneuron to stimulation of the deep layers of SC. A: responses to OX and Cx stimulation. B: location of the recorded cell in the ventral thalamic reticular nucleus (TRN). C: responses to SC and PDB stimulation. D: depth-threshold curve for orthodromic activation of the cell to SC stimulation. Note that the most effective stimulating sites were located in the deep SC and the lowest threshold was 15 μA. Electrical stimuli are marked by dots. AD, dorsal anterior nucleus; CL, central lateral nucleus; VL, ventral lateral nucleus.

−75 mV by current injection. This reversed the early part of the IPSP, but not the later part (Fig. 6C, bottom), suggesting that the SC stimulation-evoked IPSP is mediated by both GABA<sub>A</sub> and GABA<sub>B</sub> receptors in reticular cells (Ulrich and Huguenard 1996).

DISCUSSION

In the present study, we observed that stimulation of the deep layers of SC elicited an IPSP in the relay cells of LP and an EPSP with a burst of spikes in reticular cells in the ventral part of the visual sector of TRN. On average, the response latency of reticular cells was 1.6 ms shorter than that of LP cells. Stimulation of the contralateral predorsal bundle induced similar responses in both LP cells and reticular cells at latencies which were 0.5–0.6 ms longer than those from SC stimulation.

Inhibitory neuronal circuit from SC to LP

Analysis of response latencies plays a crucial role in determining the neuronal connections. In the present study, we demonstrated that stimulation of the deep SC first induced a burst firing in reticular cells and then induced an inhibition in LP relay cells. The averaged latency difference between the two responses is 1.6 ms (4.6 – 3.0 = 1.6 ms). We have shown previously that the conduction time of axons of reticular cells, namely the antidromic latencies from LP stimulation, is ~1 ms (Zhu and Lo 1997). Therefore the latency difference just allows one synaptic delay between reticular cells and LP relay cells. This agrees well with the anatomic finding that a group of neurons in the ventral part of the visual sector of TRN project to and terminate in LP (Conley and Diamond 1990; Crabtree and Killackey 1989; Fitzgibbon et al. 1995; Pinault et al. 1995; Rodrigo-Angulo and Reinoso-Suárez 1988). The timing of the responses in LP relay cells and reticular cells thus indicates that the IPSP in LP relay cells is mediated by the reticular cells located in the ventral part of the visual sector of TRN.

We found that the response latencies from the predorsal bundle stimulation in both LP and reticular cells were 0.5–0.6 ms longer than those from SC stimulation. Because the antidromic latencies from the predorsal bundle to the deep SC neurons have a peak value ~0.4–0.6 ms (Zhu and Lo 1995), the latency difference from the two stimulating sites will allow no extra synaptic delay time. Therefore both the
inhibition in LP cells and excitation in reticular cells must originate from predorsal bundle-projecting neurons in the deep SC.

Because predorsal bundle-projecting cells in the deep SC do not project to TRN or LP directly (Harting 1977; Holstege and Collewijn 1982; Huerta and Harting 1984; Wells et al. 1989), there must be some relay(s) between SC and TRN or LP. There are several nuclei that receive the projections from predorsal bundle-projecting cells and may function as the relay(s). The best candidate for the relay is CL because CL cells not only receive a direct input from predorsal bundle-projecting neurons in the deep SC (Bickford and Hall 1989; Chevalier and Deniau 1984; Grantyn and Grantyn 1982; Lo 1988; Yamasaki and Krauthamer 1990; Zhu and Lo 1995), they also project to TRN (Deschénes et al. 1996; Jones 1975; Lo 1988). Recently, we have identified an inhibitory neuronal circuit from SC to LGN via CL and TRN (Zhu and Lo 1996b). The time delay for this circuit from SC to TRN varies from 1.3 to 4.8 ms, matching closely to the response latency in reticular cells from SC stimulation found in this study (1.5–4.5 ms). Thus the inhibitory neuronal circuit from SC to LP is likely to be: predorsal bundle-projecting neurons in the deep SC-CL-ventral part of the visual sector of TRN-LP (Fig. 7).

Comparison of the circuits from SC to LP and to LGN

Although LGN and LP relay visual inputs from the retina to the cortex via different pathways, there is good anatomic evidence that these nuclei receive their main extraretinal inputs from the same sources. Anatomic studies from a variety of mammals, including rabbits, rats, cats, and monkeys, have shown that both LGN and LP are innervated by corticothalamic fibers and at least some of the fiber terminals form the similar synaptic patterns in these nuclei (Bourassa and Deschénes 1995; Giolli et al. 1978; Hollander et al. 1979; Paré and Smith 1996; Rockland 1996; Vidnyanszky et al. 1996). There are also rich projections from neuromodulatory nuclei (e.g., cholinergic fibers from the parabrachial region in the brain stem and histaminergic fibers from the hypothalamus) to these nuclei in cats and monkeys (Fitzpatrick et al. 1989; Manning et al. 1996; Patel and Bickford 1997), and some of these cholinergic and monoaminergic fibers have been shown to branch into both LGN and LP (Manning et al. 1996; Uhlrich et al. 1988). These results have been used to argue that a similar synaptic organization may exist in LGN and LP (Patel and Bickford 1997; Sherman and Guillery 1996).

Previously we showed that both LGN and LP receive recurrent inhibitory inputs from the GABAergic cells located in the visual sector of TRN (Lo and Xie 1987a; Zhu and Lo 1995). In this study, we extend this finding and show that both recurrent inhibitory circuits are controlled by the same afferents from predorsal bundle-projecting cells in the deep SC (Zhu and Lo 1995). Interestingly, the response characteristics of relay cells in LP and recurrent inhibitory interneurons in ventral part of the visual sector of TRN to SC stimulation are strikingly similar to those of LGN relay
FIG. 6. Intracellular responses of recurrent interneurons of LP to stimulation of the deep layers of SC. A: locations of 2 recorded cells in the TRN. B: both SC and PDB stimuli induced an excitatory postsynaptic potential (EPSP) in a reticular cell (* in A). Note that lower stimulation intensity was applied during the intracellular recordings to avoid action potentials. Resting membrane potential of the cell was −69 mV. C: intracellular responses of another reticular cell, marked by a dot in A, to SC stimulation. Note that the EPSP was followed by an IPSP, and the early part of the IPSP was reversed by hyperpolarization. Resting membrane potential of the cell was −60 mV. Action potentials are truncated.

cells and recurrent inhibitory interneurons in the dorsal part of the visual sector of TRN (Lo 1988; Lo and Xie 1987b; Zhu and Lo 1995, 1996b). In particular, the activities in the two inhibitory circuits are of almost identical temporal properties. These similarities may result from a similar circuit used by these two inhibitory pathways (Fig. 7) (see also Zhu and Lo 1995). Thus our data support the ideas that a very similar set of extraretinal inputs innervate LGN and LP and that they may also gate and modulate the ascending visual information transmitted in the geniculocortical and extrageniculate visual pathways in a very similar way.

Functional considerations

According to morphological and physiological observations, predorsal bundle-projecting neurons in the deep SC are believed to initiate gaze shifts including saccadic eye movements (Dorris et al. 1997; Harting 1977; Holstege and Collewijn 1982; Huerta and Harting 1982; Moschovakis et al. 1988; Sparks and Hartwich-Young 1989; Wells et al. 1989). We induced visible eye movements in the rabbit by stimulating the deep layers of SC or the predorsal bundle. The same SC or the predorsal bundle stimulation evoked a prolonged IPSP in LGN (Zhu and Lo 1996b) and LP (this study) relay cells, suggesting that the inhibitory circuits from the deep SC to LGN and LP may provide a prolonged inhibition to the geniculate and extrageniculate visual pathways during saccadic eye movements. This is consistent with the observations on the awake, behaving animals that the activity of LP and LGN cells is suppressed for ~150 ms during saccadic eye movements (Guido and Weyand 1994; Robinson et al. 1991). It also agrees with the duration of a typical saccadic suppression (Beeler 1967; Burr et al. 1994; Collewijn 1969; Latour 1962; Mohler and Cechner 1975).

The notion that inhibitory circuits from SC to LGN and LP are involved in saccadic suppression is also supported by other temporal properties of the activities in the circuits. For example, the latency of the IPSP in LGN and LP relay cells from SC stimulation range from 3 to 6 ms, which is much shorter than the delay between the firing in predorsal bundle-projecting cells and the subsequent saccadic eye movement (20–60 ms) (Moschovakis et al. 1988). This will result in a visual suppression started earlier than the saccadic eye movement, consistent with the early saccadic
suppression found in physiological and psychological studies (Beeler 1967; Duffy and Lombroso 1968; Latour 1962; Mohler and Cechner 1975). In addition, the IPSP in LGN and LP cells is sometimes followed by a rebound burst of spikes. This is in line with a study on awake cats (Guido and Weyand 1995), which showed that a burst of spikes occurred sometimes after spontaneous saccadic eye movements in LGN cells. Besides, there is a late IPSP in reticular cells after the initial EPSP. Together the rebound burst in relay cells and the IPSP in reticular cells may help to terminate the suppression and to facilitate the transmission of novel visual information after saccades. Physiological and psychological studies did observe a facilitation of visual transmission after saccadic eye movements (Bartlett et al. 1976; Beeler 1967).

Although it is well established that saccadic eye movements are always accompanied by a visual suppression, recent studies suggested that the suppression is not absolute (Benevento and Port 1995; Cai et al. 1997; Robinson et al. 1991; Ross et al. 1997). These studies have demonstrated that some visual signals still can reach the visual cortex during saccadic eye movements. It was proposed that the saccade-evoked inhibition may help to generate the visual salience in LGN and LP by suppressing insignificant signals (Guido and Weyand 1994; Robinson et al. 1991). The prolonged IPSP generated by the inhibitory circuits from SC to LGN and LP may serve this function. By hyperpolarizing the membrane potential in LGN and LP relay cells, the IPSP not only prevents small EPSPs from reaching threshold, it also deactivates a low-threshold calcium conductance (Guido et al. 1992; Lo et al. 1991). The deactivated low-threshold calcium conductance is then ready to be activated by large EPSPs, and it can nonlinearly amplify them by generating a burst of action potentials (Lo et al. 1991; Lu et al. 1992). Thus during saccadic eye movements, only salient visual information may be selectively amplified and transducted to the cortex.

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INHIBITORY CIRCUIT FROM SC TO LP


