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DIRECTIONAL MODULATION OF VISUAL RESPONSES OF PRETECTAL NEURONS BY ACCESSORY OPTIC NEURONS IN PIGEONS

Y. GU, Y. WANG and S. R. WANG*

Laboratory for Visual Information Processing, Institute of Biophysics, Chinese Academy of Sciences, 15 Datun Road, Beijing 100101, PR China

Abstract—The nucleus lentiformis mesencephali and the nucleus of the basal optic root in birds, homologous to the nucleus of the optic tract and the terminal nuclei of the accessory optic tract in mammals, are involved in optokinetic nystagmus. The present study provides the first electrophysiological evidence that reversible blockade of the pigeon nucleus of the basal optic root by lidocaine can change visual responsiveness of pretectal neurons in a direction-dependent manner. Thirty pretectal cells examined were classified as unidirectional (80%), bidirectional (10%) and omnidirectional (10%) cells according to their directional selectivity. Among the unidirectional cells, seven cells changed firing rates in all directions of motion, 11 changed visual responses only in the preferred directions and six others did not change their responsiveness during lidocaine. Most of the bidirectional cells changed firing rates in the temporonasal direction, and two-thirds of the omnidirectional cells showed these changes in all directions. Thirteen lidocaine administration sites were marked within the nucleus of the basal optic root and 19 recording sites were marked within the nucleus lentiformis mesencephali. This histological verification indicates that the effects of lidocaine blockade in the accessory optic nucleus on the directional selectivity and visual responsiveness of pretectal cells appear to be related, to some extent, to the location of drug injections in the nucleus of the basal optic root.

This study has found that visual neurons in the nucleus of the basal optic root, which predominantly prefer vertical and backward motion, could modulate the directional selectivity and visual responsiveness of neurons in the nucleus lentiformis mesencephali, which mainly prefer horizontal motion. It is conceivable that both nuclei work together in coordination and in competition during optokinetic nystagmus.

Key words: accessory optic system, directional selectivity, lidocaine, optokinetic nystagmus, pretectum, receptive field.

It has been shown that the nucleus lentiformis mesencephali (nLM) in the pretectum and the nucleus of the basal optic root (nBOR) of the accessory optic system in birds are primary visual nuclei involved in generating optokinetic nystagmus, which stabilizes an object image on the retina by slow tracking and rapid resetting movements of the eyes. Their mammalian homologues are thought to be the nucleus of the optic tract and the terminal nuclei of the accessory optic tract, respectively. Visual neurons in these optokinetic nuclei are selective for direction and velocity of motion, and respond to motion of large-field patterns.[2,9,10,17,20,28,30–34,37] In pigeons, receptive fields of optokinetic neurons are usually composed of an excitatory receptive field and an inhibitory receptive field, both of which are overlapped or spatially separated but possess opposite directionality.[9,31,37] Most directional cells in the pigeon nLM prefer horizontal motion, but others prefer vertical motion,[2,9,10,30] whereas directional neurons in the pigeon nBOR are predominantly sensitive to vertical and backward motion.[5,12,28,31,33,34,37] It appears that both nuclei may play complementary roles in generating the optokinetic reflex.

In fact, neuroanatomical studies[1,4,35] have verified neuronal connections between the nLM and nBOR in birds, indicating the existence of functional interactions between both optokinetic nuclei. Previous studies with pigeons have found that the nBOR mainly exerts an inhibitory action on pretectal neurons,[2] whereas the nLM mainly excites accessory optic units with temporonasal directionality and inhibits those with nasotemporal preference.[22] However, these results were obtained by examining changes in spontaneous activity of pretectal or accessory optic cells following electrical stimulation of the nBOR or nLM. Though these physiological interactions may also exist between the nucleus of the optic tract and the terminal nuclei of the accessory optic tract in mammals,[13,14,21,23–25] very little is known about physiological interactions between these optokinetic nuclei in terms of directional modulation of visual responsiveness of optokinetic neurons in all species studied so far.

To further reveal physiological effects of the accessory optic nucleus on directional responses of visual neurons in the pretectal nucleus in pigeons, the present study was therefore undertaken to quantitatively analyse changes in visual responsiveness of pretectal neurons following

*Corresponding author. Tel.: +86-10-6488-9858; fax: +86-10-6487-1293.

E-mail address: wangsr@sun5.ibp.ac.cn (S. R. Wang).

Abbreviations: nBOR, nucleus of the basal optic root; nLM, nucleus lentiformis mesencephali.

reversible blockade of the accessory optic nucleus by lidocaine, which is a useful tool for studying interactions between visual structures.[8,19,27,29]

EXPERIMENTAL PROCEDURES

Seventeen adult homing pigeons (*Columba livia*) of either sex, weighing 370–440 g, were used following the Policy on the Use of Animals in Neuroscience Research approved by the Society for Neuroscience in 1995. All efforts were made to minimize the number of animals used and their suffering. Surgical procedures were as described previously.[9,28] In brief, the pigeon was anesthetized with urethane (20%, 1 ml/100 g body weight, i.m.) and then placed in a stereotaxic apparatus. Body temperature was maintained at 41°C by a heating pad. The left rostral tectum and caudal forebrain were surgically exposed and the overlying dura mater was excised. The nictitating membrane of the right eye was removed and the eye kept open. The left eye was occluded with a cover. A screen of 130° (vertical angle) × 140° (horizontal angle) was positioned 40 cm distant from the viewing eye. To examine visual responses and their changes in nLM cells, a black bar (2.0–6.0° wide × 130° long) was generated by a workstation (SiliconGraphics Indigo 2) and back-projected onto the screen with a three-color projector (Electrohome ECP4). The long bar was always oriented perpendicular to its direction of motion and therefore scanned over the whole receptive field, whose location and extent were plotted with a hand-held target. The bar was randomly moved at velocities of 6–45°/s in eight directions (nasal: 0°, 45°; dorsal: 90°, 135°; temporal: 180°, 225°; ventral: 270°, 315°) in the pigeon visual field to determine the preferred direction of a particular cell. The horizontal meridian of the visual field was rotated by 38°[9] to meet the pigeon's normal conditions for flying, walking, standing and perching.[7] The effects of reversible blockade of the nBOR by lidocaine on the visual responses of pretectal neurons were examined in four orthogonal directions, including the preferred directions.

Following an injection of lidocaine into the nBOR, the total number of spikes was accumulated and an average firing rate obtained in each direction of motion. Pretectal cells were each examined for three injections with an interval of 20–30 min, with a subsequent injection being made after a given cell had recovered from a previous injection. Paired *t*-tests were then made between firing rates obtained before and during lidocaine for each of the directions. Within the same group of cells, *t*-tests were made between the average firing rates obtained with three injections for each direction across the cells. In all cases, $P < 0.01$ was considered statistically significant.

Action potentials of pretectal neurons were recorded extracellularly with a micropipette (1–3 μm tip diameter) filled with 2 M sodium acetate and 2% Pontamine Sky Blue.[16] A two-barrel pipette (5–10 μm tip diameter), one of whose channels was filled with 2 M sodium acetate and the dye, and the other filled with 2% lidocaine hydrochloride and connected to a pneumatic picopump (PV800, Medical Systems Corp.), was used both for electrophysiological confirmation of the nBOR together with stereotaxic coordinates,[18] and for marking electrode tip sites and applying drug into the nucleus. Lidocaine was injected into the nBOR in volumes of 40–100 nl to block nBOR–nLM transmission. Blocking action of lidocaine in the nucleus usually lasted up to 20 min, during which visual examination could be completed. In some cases where pretectal cells were not influenced by lidocaine in smaller volumes, a larger dosage of lidocaine (120–200 nl) was applied. If no observable effects occurred, these cells were thought not to be influenced by lidocaine blockade. Spikes recorded from pretectal cells were amplified and displayed on an oscilloscope, and were fed into the workstation computer for on-line analysis.

At the end of experiments, the recording sites of some pretectal neurons and drug administration sites within the nBOR were marked with Pontamine Sky Blue, which was applied by negative current pulses of 10–20 μA intensity and 0.5 s duration at 1 Hz for 10–15 min. Under deep anesthesia, the brain was removed from the skull and fixed in 4% paraformaldehyde for 6–12 h, then soaked in 30% sucrose solution in a refrigerator overnight. Frozen sections were cut at 100 μm thickness and counterstained with Cresyl Violet. Sections were dehydrated and covered for subsequent microscopic observation of the recording sites of pretectal cells and of lidocaine administration sites within the accessory optic nucleus.

RESULTS

Thirty pretectal cells were recorded extracellularly from the nLM and quantitatively examined for effects of lidocaine injections in the nBOR on their visual responses evoked by motion in various directions. The spontaneous and visual activities of pretectal cells were fairly stable in control conditions. Statistics done on 30 pretectal cells showed that the maximal percentage change of visual firing rates was 9%, with an average change of $3.1 \pm 2.2\%$ (mean ± S.D.). Twenty-two of these were spontaneously active, with firing rates ranging from 3 to 85 spikes/s. Among the spontaneous cells, six enhanced spontaneous activity by $89 \pm 40\%$, one reduced resting activity from 7.0 to 2.5 spikes/s and the remainder did not significantly change spontaneity ($t = 0.39$, $P > 0.01$, $n = 15$) during lidocaine administration in the nBOR. According to the directional selectivity of pretectal cells, the 30 cells examined were classified as unidirectional (80%), bidirectional (10%) and omnidirectional (10%) cells. The recording sites of 19 pretectal cells were marked within the nLM and 13 lidocaine administration sites marked within the nBOR (Fig. 1). These histological markings verified the reliability of the recording and drug administration techniques used in the present study.

Twenty-four unidirectional cells were examined for effects of drug administration in the nBOR on their visual responses produced by motion in four orthogonal directions, including the preferred directions. These nLM cells could be categorized into three groups according to changes in directional responses to drug application. The first group included seven pretectal cells that changed firing rates in all directions of motion. Among them, three temporonasal- and one nasotemporal-preferring cells increased firing rates by $47 \pm 21\%$ in the preferred directions and by $72 \pm 39\%$ in the other directions ($t = 6.39$, $P < 0.01$, $n = 12$; Fig. 2A). Three others preferring temporonasal motion reduced firing rates by $30 \pm 13\%$ in the preferred directions and by $39 \pm 22\%$ in the other directions ($t = 5.02$, $P < 0.01$, $n = 9$; Fig. 2B). The second group contained 11 pretectal cells that changed firing rates only in the preferred directions. Within this group, two temporonasal- and six nasotemporal-preferring cells increased firing rates by $31 \pm 11\%$ in the preferred directions ($t = 7.49$, $P < 0.01$, $n = 8$), and they did not show a significant change ($t = 1.06$, $P > 0.01$, $n = 24$) in firing rates in three other directions. One nasotemporal-preferring cell reduced firing rates by 25% in the preferred direction and did not change its firing rates in the other directions. Two vertical-preferring cells increased visual responses by 27% in the preferred directions and did not show a significant change in three other directions ($t = 1.02$, $P > 0.01$, $n = 6$; Fig. 3A). The third group consisted of two horizontal- and four

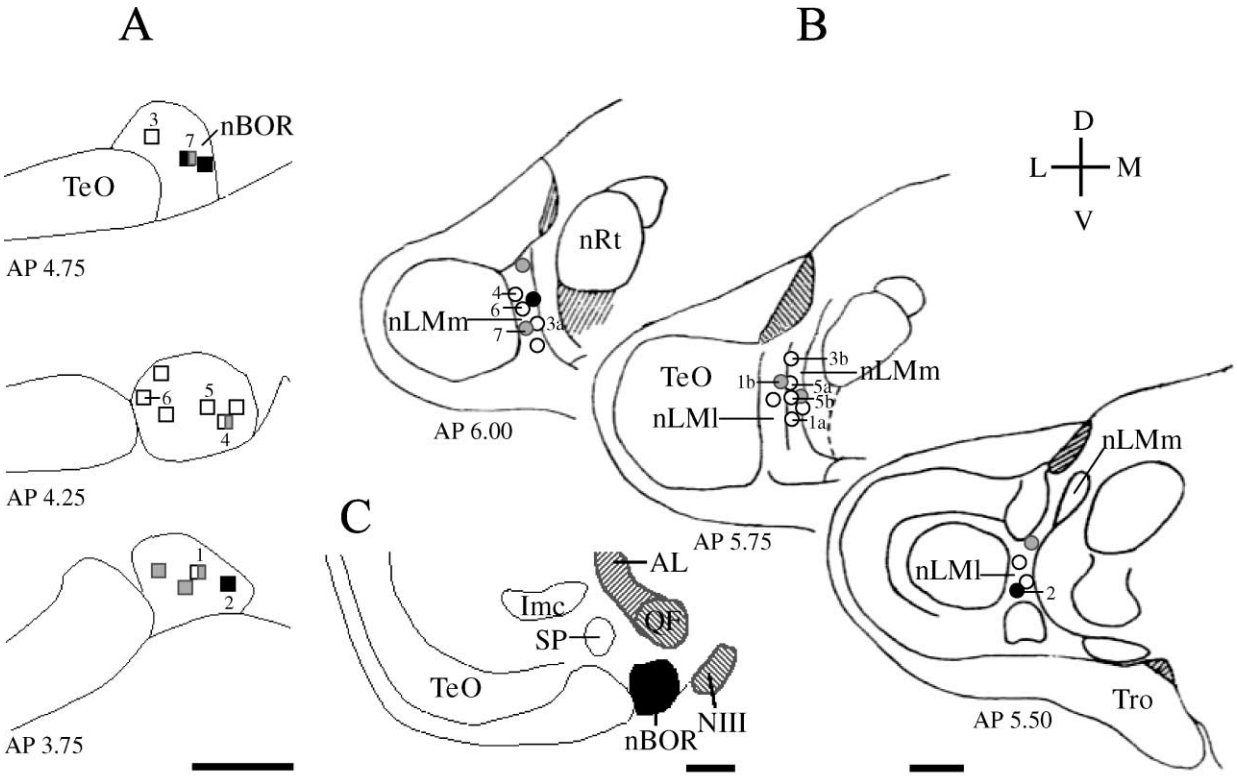

Fig. 1. Topographic distribution of 13 lidocaine administration sites in the nBOR (A), and that of 19 recording sites in the pars medialis (nLMm) and pars lateralis (nLMl) of the nLM (B), in cross-sections of the pigeon's brain. C shows the location of the nBOR proper in a cross-section of the pigeon's brain at AP 4.25. In A, open, filled and stippled squares represent drug sites producing an increase, decrease or no change in visual responsiveness of pretectal neurons, respectively. Half-stippled squares symbolize those drug administration sites which produced an increase or decrease, or no change in pretectal activity. In B, open, filled and stippled circles represent recording sites of pretectal cells whose visual responses were increased, decreased or not affected by blockade of the nBOR, respectively. Numerals labeling drug sites 1–7 in A correspond to those marking recording sites 1–7 in B. Note that some injection sites (1, 3 and 5 in A) each correspond to two pretectal cells a and b in B. AL, ansa lenticularis; Imc, nucleus isthmi pars magnocellularis; NIII, nervus oculomotoris; nRt, nucleus rotundus; QF, tractus quintofrontalis; SP, nucleus subpretectalis; TeO, tectum opticum; Tro, tractus opticus. AP, anterior–posterior levels in the pigeon brain atlas.[18] D, L, M and V represent dorsal, lateral, medial and ventral, respectively. Scale bars = 1 mm.

vertical-preferring cells, all of which did not significantly change their firing rates produced by motion in all directions examined during lidocaine injections into the nBOR ($t = 0.52$, $P > 0.01$, $n = 24$).

Three bidirectional cells all responded to horizontal but not vertical motion. Two of these were more sensitive to nasotemporal motion than temporonasal motion. Their firing rates in the temporonasal direction were 76% and 70% of those in the nasotemporal direction, respectively. During lidocaine administration in the nBOR, their firing rates in the temporonasal direction were increased to 157% and 122% of control values, whereas no obvious changes in firing rates were produced by nasotemporal motion (Fig. 3B). The other cell was more sensitive to temporonasal motion, with firing rates in the temporonasal direction being 174% of those in the nasotemporal direction. It was not affected at all by drug application. In contrast, omnidirectional cells almost equally responded to motion in all directions, and their firing rates in all directions changed in the same fashion during drug administration. Of three omnidirectional cells examined in the present study, one increased firing rates in all directions by 27%, one decreased firing rates in all directions by 28% and one did not change firing rates in any direction during lidocaine administration in the nBOR.

The recording sites of 19 visual cells were all marked within the nLM, including 13 in the pars medialis and six in the pars lateralis, according to the nomenclature of Gamlin and Cohen.[11] No obvious differences in effects of lidocaine blockade on the directional selectivity and visual responsiveness of pretectal cells were observed between the two subdivisions of the nLM. Meanwhile, 13 lidocaine administration sites were all marked within the nBOR proper, although the nBOR complex consists of three distinct subdivisions, i.e. the nBOR proper, the pars dorsalis and the pars lateralis.[4] Generally speaking, lidocaine injected in the dorsal part of the nBOR proper enhanced visual responses only in the preferred directions in horizontal- and vertical-preferring cells. During drug administration in the intermediate nBOR, most horizontal-preferring cells changed firing rates in all directions, but no vertical-preferring cells were affected. Lidocaine administration in the ventral nBOR mainly enhanced visual responses evoked by horizontal motion. However, in some cases where more than two pretectal

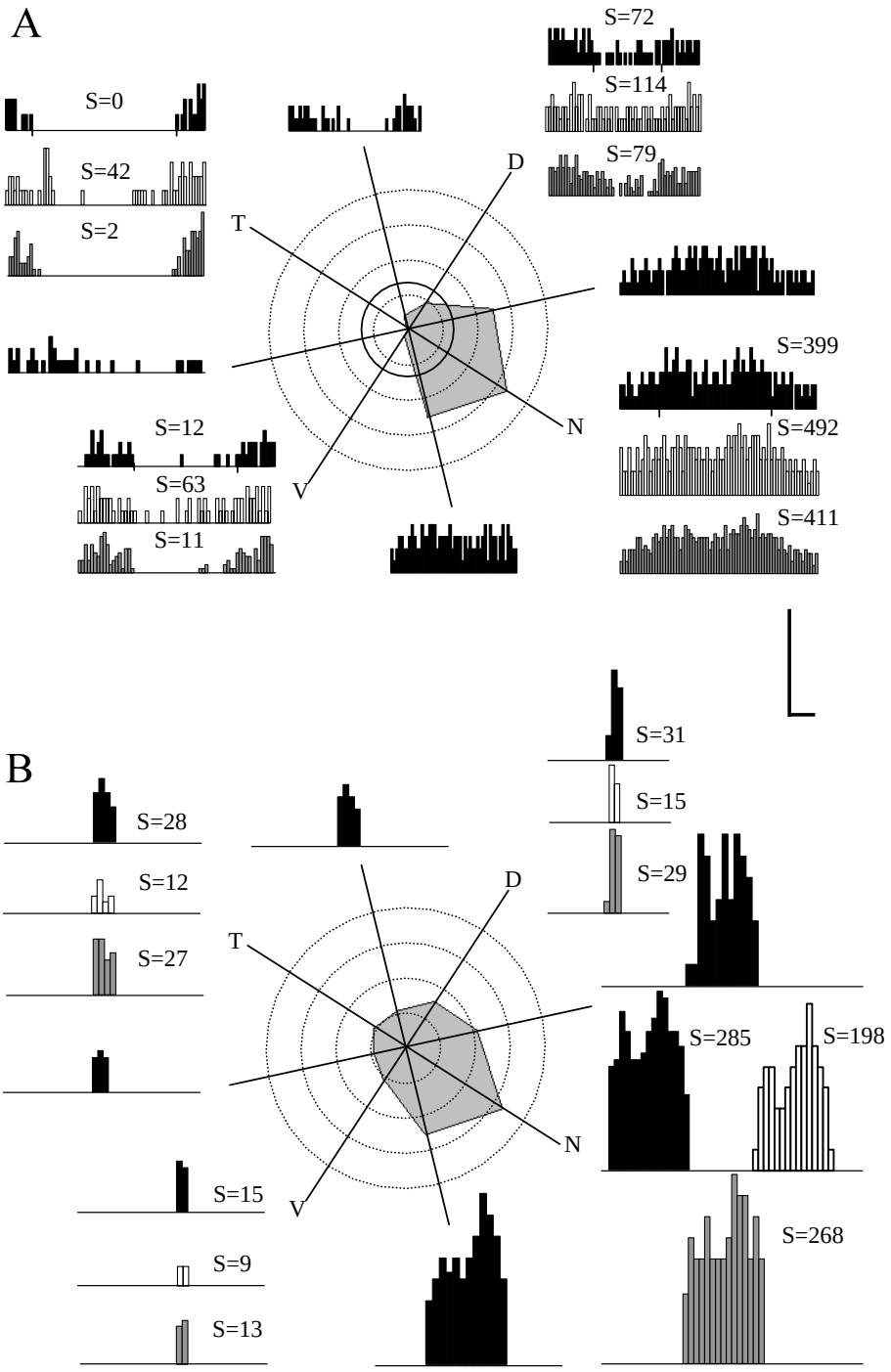

Fig. 2. Histograms showing visual responses of two unidirectional neurons (A, B) before, during and after lidocaine administration in the nBOR. (A) Lidocaine (60 nl) in the nBOR enhanced visual responses produced by motion at 6.4°/s in orthogonal directions including the preferred direction. The spontaneous activity of this cell was 6.5, 9.0 and 6.8 spikes/s before, during and after lidocaine, respectively. (B) Visual responses produced by motion at 43°/s in orthogonal directions including the preferred direction were decreased by lidocaine (80 nl) in the nBOR. Stippled polygons are response vector diagram profiles. Equal firing rate circles are spaced by 5 spikes/s in A and 10 spikes/s in B, and the solid-line circle in A represents the spontaneous activity level. Filled, open and stippled histograms were obtained before, during and after lidocaine administration, respectively. Visual responses were usually measured in eight directions, whereas the effects of lidocaine were only examined in four orthogonal directions, including the preferred direction. S, the total number of spikes accumulated for three sweeps. Short vertical lines beneath filled histograms mark the start and end of visual responses elicited by motion. The recording sites of cells A and B are shown with numerals 1a and 2 in Fig. 1B, and their drug sites are labeled with numerals 1 and 2 in Fig. 1A. N, D, T and V represent nasal, dorsal, temporal and ventral, respectively. Note that the horizontal meridian of the visual field was rotated by 38°[9] to meet the pigeon's normal conditions for flying, walking, standing and perching.[7] Scales: 30 spikes/200 ms and 2 s (A); 15 spikes/200 ms and 1 s (B).

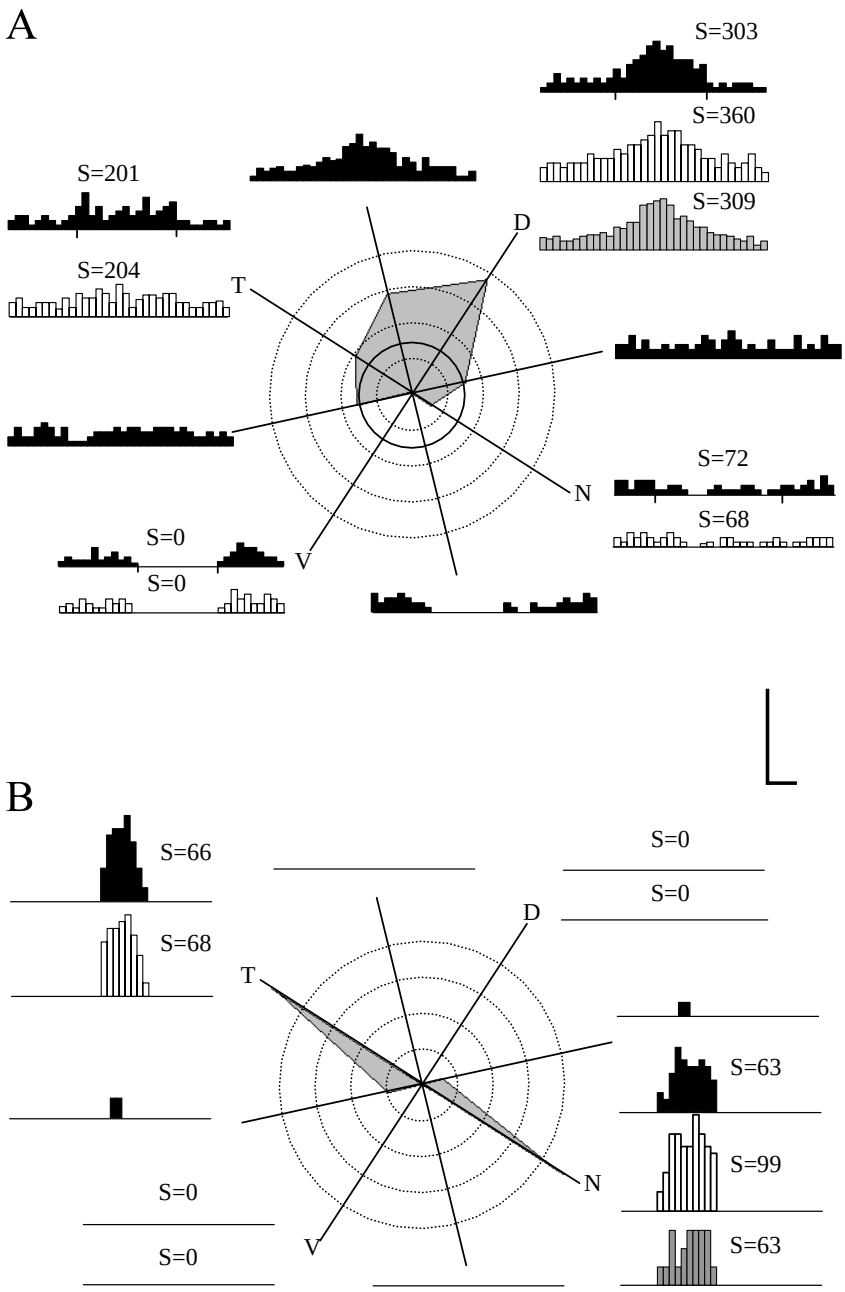

Fig. 3. Histograms showing direction-dependent changes in visual responses of unidirectional (A) and bidirectional (B) pretectal neurons following blockade of the nBOR by lidocaine. In A, visual responses evoked by motion at 8.9°/s in the ventrodorsal direction were increased and those in other directions were not affected by lidocaine (60 nl). The spontaneous firing rate of this cell was 14.4, 25.2 and 16.8 spikes/s before, during and after lidocaine, respectively. In B, visual responses elicited by motion at 12°/s in the temporonasal direction but not those in the nasotemporal direction were increased by lidocaine (60 nl). Drug administration sites for cells A and B are shown with numerals 3 and 4 in Fig. 1A, and their recording sites correspond to 3a and 4 in Fig. 1B, respectively. Equal firing rate circles are spaced by 10 spikes/s in A and 2.5 spikes/s in B. Other notation is as in Fig. 2. Scales: 60 spikes/200 ms and 1 s (A); 15 spikes/200 ms and 1 s.

cells were examined during lidocaine injections in the same site, visual responses of these cells were all increased, partially increased or decreased and unchanged (Fig. 1).

DISCUSSION

Although extensive studies have reported visual response properties of neurons in the avian nLM and nBOR,[9,10,28,30–34,37] as well as neuronal connections between both nuclei,[1,4,35] very little is known about their functional interactions.[2,15,22] The present study using lidocaine blockade has provided electrophysiological evidence that the nBOR could strongly modulate visual responsiveness of pretectal neurons in a direction-dependent manner. Several studies[8,19,27,29] have shown that lidocaine is a useful tool for investigating functional interactions between neural structures. First, the

specificity and reversibility of the effects of lidocaine on neuronal activity indicate that these effects are pharmacological but not toxicological.[8,19,27,29] Second, blockade of lidocaine once injected could last long enough to examine its effects on visual responses elicited by motion in several directions.[19,27] It appears that pharmacological blockade by lidocaine is more advantageous over electrical stimulation and brain lesions in studies on functional interactions between neural structures. However, it is worth noting that an increase in firing rates of a pretectal cell after lidocaine administration in the nBOR actually implies inhibition of the pretectal cell by the accessory optic nucleus, whereas a decrease in firing activity of a pretectal cell by lidocaine shows excitation of the cell by the accessory optic–pretectal pathway.

The present study not only confirms the finding that spontaneous activity in pretectal cells could be inhibited by electrical stimulation of the nBOR,[2] but also provides a quantitative analysis of the influence of the nBOR on visual responsiveness of pretectal neurons. Our results indicate for the first time that the pigeon nBOR can modulate visual responses of pretectal neurons in a direction-dependent manner. Unidirectional cells could be categorized into three groups according to their directional responses. The first group of cells change firing rates in all directions of motion, the second group of cells change firing rates only in the preferred directions, and cells in the third group do not significantly change firing rates in any direction. Bidirectional cells appear to change firing rates mainly in the temporonasal direction. In omnidirectional cells, changes in firing rates, if any, would occur in all directions in the same fashion. Taken together with the existence of a functional map showing that ventrodorsal-, dorsoventral- and nasotemporal-preferring cells are topographically distributed from the dorsal to the ventral portion of the nBOR,[31] this suggests that the directional selectivity of nBOR cells at lidocaine administration sites and effects of the chemical blockade on directional responses of pretectal cells appear to be related to some extent. For example, lidocaine injected into the dorsal part of the nBOR proper enhanced visual responses only in the preferred directions of pretectal cells, whereas drug administration in the ventral nBOR mainly enhanced visual responses evoked by horizontal motion. However, it is difficult to figure out a clear topographic correlation, probably due to the small sample of pretectal cells and nBOR injection sites in the present study. It appears that the nBOR modulates directional selectivity and visual responsiveness of pretectal neurons in three modes: (i) to enhance or reduce visual responsiveness in all directions of motion; (ii) to reduce visual responsiveness only in the preferred directions, broadening direction tuning in most directional cells; and (iii) to enhance visual responsiveness only in the preferred directions, sharpening direction tuning in some directional cells. Therefore, it is likely that nBOR–nLM modulation is more diverse than nLM–nBOR modulation, during which the nLM mainly excites nBOR cells with temporonasal preference and inhibits those preferring nasotemporal motion.[22] This suggests that reciprocal modulations between both nuclei may function in different ways.

Inhibitory modulation of pretectal cells by the nBOR is also supported by the finding that the nLM is rich in GABA receptors and GABAergic fibers,[6,26] and GABA inhibits spontaneous and visual activities of pretectal cells,[36] indicating that GABA-mediated inhibition may play an important role in optokinetic nystagmus.[3,36] In mammals, projections from the accessory optic nuclei to the nucleus of the optic tract and the dorsal terminal nucleus have been thought to be GABAergic.[13,14,23–25] The present finding that visual responses of pretectal cells could be inhibited by the nBOR is also supported by electrophysiological studies on rats.[24,25] In view of the fact that nLM neurons mainly prefer horizontal motion, whereas nBOR cells predominantly prefer vertical and backward motion, this suggests that the nBOR–nLM pathway may play an important role in modulating directional selectivity and visual responsiveness of pretectal cells. Therefore, both the nBOR and nLM may work together in coordination and in competition in generating optokinetic nystagmus.

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