Original Paper

Brain, Behavior and Evolution

Brain Behav Evol 2000:56:287–292

Modulatory Effects of the Nucleus of the Basal Optic Root on Rotundal Neurons in Pigeons

Yuan Wang Yong Gu Shu-Rong Wang

Laboratory for Visual Information Processing, Institute of Biophysics, Chinese Academy of Sciences, Beijing, China

Key Words

Electrical stimulation · Lidocaine · Modulation · Nucleus rotundus · Nucleus, basal optic root · Pigeon

Abstract

The present paper reports for the first time in birds the modulatory effects of the nucleus of the basal optic root (nBOR) on visual neurons in the nucleus rotundus in particular and those of the accessory optic system on the tectofugal pathway in general. Pharmacological blockade of the nBOR by lidocaine led to a decrease or increase in visual responsiveness of rotundal cells, suggesting excitatory or inhibitory actions of the nBOR on rotundal cells. These results were confirmed by changes in the excitability of rotundal cells following electrical stimulation of the nBOR. Response latency measurements implied that there might be at least two pathways from the nBOR to the nucleus rotundus, one being a direct excitatory pathway and the other an indirect inhibitory pathway possibly mediated by the subpretectal nucleus and the interstitio-pretecto-subpretectal nucleus, which have been thought to send inhibitory afferents to the nucleus rotundus. Taken together with previous neuroanatomical and immunocytochemical studies, it is suggested that modulatory interactions might exist between the nBOR and the nRt in particular and between the accessory optic system and the tectofugal pathway in general in birds.

Introduction

The visual system in birds consists of the thalamofugal, tectofugal and accessory optic pathways. In the thalamofugal pathway, retinal fibers contralaterally project to the nucleus geniculatus lateralis, pars dorsalis, which bilaterally projects to the visual wulst of the telencephalon [Güntürkün et al., 1993]. In the tectofugal pathway, retinal fibers project to the contralateral optic tectum, whose layer 13 neurons project to the rotundus nucleus (nRt) [Karten et al., 1997; Deng and Rogers, 1998a, b; Hellmann and Güntürkün, 1999]. This nucleus projects ipsilaterally to the telencephalic ectostriatum [Engelage and Bischof, 1993]. In the accessory optic pathway, displaced ganglion cells in the retina project axons to the nucleus of the basal optic root (nBOR) [Karten et al., 1977; Reiner et al., 1979; Fite et al., 1981; Nickla et al., 1994]. It sends diverse projections to various regions in the mesencephalon, diencephalon and cerebellum, including the contralateral nBOR, the nucleus lentiformis mesencephali, reticular formation, central gray, pontine nuclei, vestibulocerebellum and oculomotor complex [Brecha et al., 1980; Gioanni et al., 1984; Telford and Frost, 1989; Wylie and Linkenhoker, 1996]. In addition to these projections, a few terminals also could be seen in the nRt, the subpretectal nucleus (SP) and the interstitio-pretecto-subpretectal nucleus (IPS) after injecting a tracer into the nBOR [Wylie et al., 1997]. The latter two nuclei possibly send inhibitory efferents to the nRt [Deng and Rogers, 1998b; Mpodozis et al., 1996; Tömböl et al., 1994, 1999]. Therefore, a question arises concerning possible actions the acces-

KARGER

Fax + 41 61 306 12 34
E-Mail karger@karger.ch
www.karger.com

0006–8977/00/0565–0287$17.50/0

Accessible online at:
www.karger.com/journals/bbe

Shu-Rong Wang
Laboratory for Visual Information Processing, Institute of Biophysics
Chinese Academy of Sciences, 15 Datun Road, Beijing 100101 (China)
Tel. +86 10 6488 9858 (Office) / 6488 8528 (Lab)
E-Mail wangsr@sun5.ibp.ac.cn

sory optic neurons can exert on rotundal neurons through these pathways.

Physiological studies have shown that the nBOR is involved in generating optokinetic nystagmus [McKenna and Wallman, 1985] which stabilizes an object image on the retina by compensatory eye movements and in detecting translational and rotational optic flow [Wylie and Frost, 1990; Wylie et al., 1998]. The nRt is involved in the analysis of geometric pattern, brightness, color and fine spatial detail [Hodos and Karten, 1966; Hodos et al., 1973] as well as motion in depth [Wang and Frost, 1992; Wang et al., 1993]. It is conceivable that nBOR neurons detecting self-motion and nRt neurons that detect object motion would functionally interact somewhere in the visual system. Therefore, knowledge about the physiological interaction between both nuclei is important not only for understanding the functional significance of these structures, but also for further showing an interaction between the accessory optic system and the tectofugal pathway in general.

The present study was therefore undertaken to show the effects of the accessory optic nucleus on visual responses of rotundal neurons by using lidocaine blockade and electrical stimulation of the pigeon's nBOR.

Materials and Methods

The experiments were performed on 15 adult pigeons *(Columba livia)* of either sex, weighing 300–450 g, under guidelines regarding the use of animals approved by the Society for Neuroscience. The pigeon was anesthetized with urethane (20%, 1 ml/100 g body weight), and then placed in a stereotaxic apparatus. Its body temperature was maintained at 41°C by a heating pad. The caudal forebrain on the left side was surgically exposed and the overlying dura mater 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 180 cm in height and 220 cm in width was positioned 40 cm distant from the viewing eye. For visual stimulation, a black square was generated by a workstation (SiliconGraphics Indigo 2) and rear-projected onto the screen with a three-color projector (Electrohome ECP4). The square measured 1.1–3.2° in dimension and moved at velocities of 10–40°/s. Luminance of the black square and its white background was 0.1 cd/m^2 and 6.6 cd/m^2, respectively.

Action potentials of rotundal neurons were extracellularly recorded with a micropipette filled with 2 *M* sodium acetate and 2% pontamine-skyblue. In the first series of experiments, a two-barrel pipette was used, one of whose channels was filled with 2 *M* sodium acetate and 2% pontamine-skyblue [Hellon, 1971] for both electrophysiological confirmation of the nBOR and marking electrode tip positions, and the other filled with 2% lidocaine hydrochloride and connected to a pneumatic picopump (PV800, Medical Systems Corp.) for drug application. Visual responses and their changes in rotundal cells were superimposed for three sweeps obtained from three injections, whose intervals ranged from 10–20 min. In the second series of experiments on electrically stimulating the nBOR, a bipolar tungsten electrode was used, whose 100 μm-exposed poles were 600 μm apart. In these cases, the nBOR was located first with a single pipette according to its stereotaxic coordinates [Karten and Hodos, 1967] and visual responses, and the pipette was then replaced by the bipolar electrode. Electrical stimulation was delivered by passing rectangular pulses of 0.1 ms in duration, 100–500 μA in intensity and 0.2–0.5 Hz in frequency. Excitatory and inhibitory responses of rotundal cells to the nBOR stimulation were superimposed for three (excitation) or ten (inhibition) sweeps. All the statistical values reported here represent means ± standard deviation.

By the end of experiments, the recording sites of visual neurons within the nRt and application sites of lidocaine within the nBOR were marked with pontamine-skyblue, which was ejected by negative current pulses of 10–20 μA in intensity and 0.5 s in duration at 1 Hz for 10–15 min. In the second series of experiments, the stimulation sites within the nBOR were electrolytically marked by passing positive current of 30–35 μA for 10 s through the active pole. Under deep anesthesia, the brain was removed from the skull and fixed in 4% paraformaldehyde for 6–12 h, 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 microscopic observation of the recording sites marked in the nRt and of lidocaine blockade or electrical stimulation sites marked in the nBOR.

Results

Twenty-nine rotundal cells were recorded and the effects of either lidocaine-blocking or electrically stimulating the nBOR on these cells were examined. The dye-marked recording sites of 27 rotundal cells were all located within the nRt (fig. 1A). The drug application sites marked with the dye and stimulation sites marked with electrolytic lesions in 9 pigeons were all positioned within the nBOR (fig. 1B).

In the first series of experiments, the effects of chemically blocking the nBOR on visual responses were examined in 16 rotundal cells. Following injection of lidocaine (30–100 nl) into the nBOR, visual responses in 9 of 16 cells (56%) examined were enhanced (fig. 2A). Their firing rate in response to visual stimulation was increased to 138.2 ± 17.4% of the pre-drug level after lidocaine injection into the nBOR. The statistics of data obtained by three sweeps for each cells showed a significant increase (t test, $t = 6.59$, $n = 9$, $p < 0.005$) in visual firing rate of rotundal cells following chemical blockade of the nBOR. Judging from the length of a path along which target motion kept a cell firing, receptive fields of three of the cells were expanded in size from 50.7, 61.0, and 85.7° to 67.1, 73.2, and 108.8°, respectively, when the drug effect was maximal. The other cells did not show any changes in the size of their receptive fields in this situation. The visual enhancement started about 0.5–1.5 min after lidocaine application, and returned to

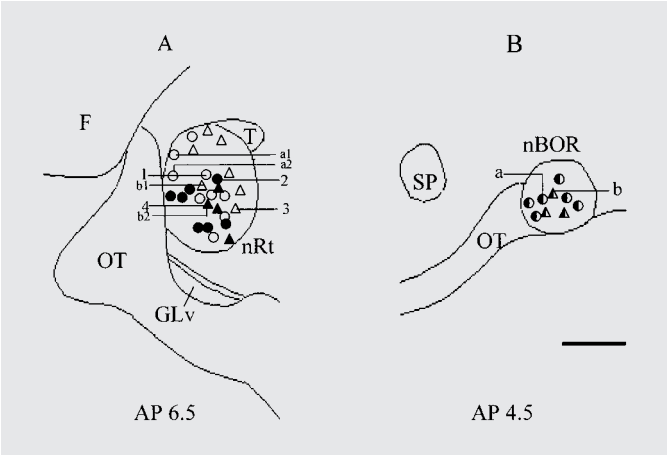

Fig. 1. Cross-section drawings of the pigeon brain show topographic distribution of recording sites of visual cells within the nucleus rotundus (nRt) (**A**) and that of lidocaine application (half-filled circles) and electrical stimulation sites (half-filled triangles) in the nucleus of the basal optic root (nBOR) (**B**). Blockade of nBOR leads to an increase (empty circles) or decrease (filled circles) in visual responsiveness of rotundal cells. Electrical stimulation of nBOR also could increase (empty triangles) or decrease (filled triangles) visual responsiveness of rotundal cells. Cells a1 and a2 (**A**) were examined during lidocaine application at a (**B**). Cells b1 and b2 (**A**) were examined when electrical stimulation was applied at b (**B**). Visual response histograms of rotundal cells 1–4 in **A** are shown in figures 2A, B and 3A, B, respectively. Other abbreviations: F = Forebrain; GLv = nucleus geniculatus lateralis, pars ventralis; OT = optic tectum; SP = nucleus subpretectalis; T = nucleus triangularis; AP = anterior-posterior levels according to the pigeon brain atlas [Karten and Hodos, 1967]. Scale bar = 1 mm.

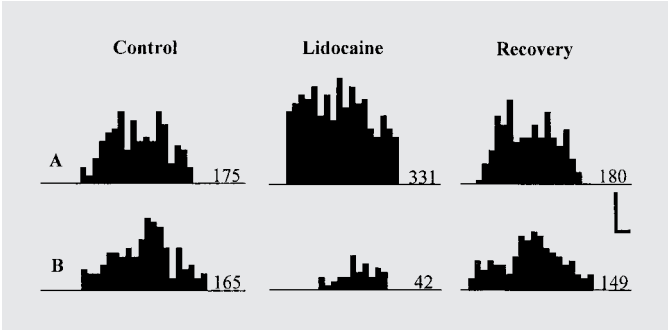

Fig. 2. Histograms showing that visual responses of rotundal cell **A** are increased and those of cell **B** decreased by lidocaine (50 nl) in nBOR. Visual responses are produced by a black square (2.8° in **A** or 1.9° in **B**) which is moved at 14.9°/s through the receptive field (28 × 25°) in the ventrodorsal direction (**A**) or at 40.0°/s through the receptive field (82 × 50°) in the dorsoventral direction (**B**). Effects of lidocaine in nBOR on rotundal cells recovered in 6 (**A**) or 5 (**B**) min after stopping drug application. Numerals at the right of histograms are the number of spikes counted for three sweeps. Note that period of time during which visual firing occurs is prolonged in **A** and shortened in **B**, implying changes in the receptive field size. The recording sites of cells **A** and **B** are labeled with numerals 1 and 2, respectively, in figure 1A. Scales = 10 spikes, 250 ms.

control 2.5–15 min after stopping drug application. Seven of these cells were spontaneously active, and two others were silent. Following lidocaine injection in the nBOR, two spontaneous cells increased their firing rates from 2.5 and 40 to 5.0 and 80 spikes/s, respectively, and one silent cell began firing at 4 spikes/s. Five other spontaneous cells, whose firing rates were 3.0, 3.0, 3.8, 7.0 and 7.5 spikes/s, and one silent cell did not show observable changes in their activity after drug application.

Visual responses in 7 of 16 rotundal cells (44%) were decreased by lidocaine (30–100 nl) blockade of the nBOR (fig. 2B). Their firing rates averaged over three sweeps were reduced to 49.6 ± 14.8% of the pre-drug levels when the blockade effect exerted on rotundal cells reached its maximum. The statistics of data obtained from three sweeps for each cells showed a significant decrease ($t = 7.73$, $n = 7$, $p < 0.005$) in visual firing rates of rotundal cells after chemical blockade of the nBOR. Receptive fields of three of these cells shrank in size from 46.0, 53.3 and 85.6° to 16.5, 32.2 and 78.3°, respectively, when the drug effect was maximal. The other cells did not obviously change their receptive fields in size when visual responses were reduced. The decrease in visual responsiveness started about 0.5 min after onset of lidocaine injection and recovered to its control level in 2.5–8 min after ceasing drug application. However, three spontaneous cells whose firing rates were respectively 2.0, 3.1 and 10 spikes/s, and four silent cells did not significantly change their resting activity after lidocaine application.

The recording sites of these rotundal cells were marked with dye and all of the sites were located within the nRt (fig. 1A), with five being in the rostral, nine in the middle and two in the caudal divisions of this nucleus. Six lidocaine injection sites were all marked within the nBOR (fig. 1B), with two sites in the rostral and four in the middle divisions of the nucleus.

In the second series of experiments, effects of electrically stimulating the nBOR on rotundal activity were examined on 13 rotundal cells. Of these, 8 cells (62%) produced excitatory responses which were mostly characterized by one spike following a single electrical shock (fig. 3A). Their average discharge latency was 9.4 ± 2.9 ms, ranging from 5 to 14 ms. Excitatory effects of the nBOR stimulation on the visual responses of these cells were not clearly observed.

The nBOR stimulation did not affect resting activity in three spontaneous cells that fired 4.1, 7.3 and 42 spikes/s respectively, and in five silent cells.

Visual activity in 5 cells (38%) was inhibited by electrical stimulation of the nBOR (fig. 3B), with an average latency of 47.0 ± 28.0 ms, ranging from 15 to 80 ms. The inhibition lasted for 10–40 ms, with an average value of 23 ± 10 ms. Spontaneous firing in two cells that fired 3.7 and 12.4 spikes/s, respectively, was completely abolished by the nBOR stimulation.

The dye-marked recording sites of 11 rotundal cells responding to electrical stimulation of the nBOR were all located within the nRt (fig. 1A), with one excitatory and two inhibitory cells in the rostral division, five excitatory and two inhibitory cells in the middle division, and one excitatory cell in the caudal division of the nRt. Three stimulation sites marked with electrolytic lesions were located within the nBOR (fig. 1B), with one site in the rostral and two in the middle division of the nBOR.

In each of 8 animals, more than two rotundal cells were examined responding to the same drug application or electrical stimulation site in the nBOR. In four of the pigeons, the same sites produced either excitatory or inhibitory responses in all rotundal cells, and in four others the same sites produced different responses from cell to cell (fig. 1).

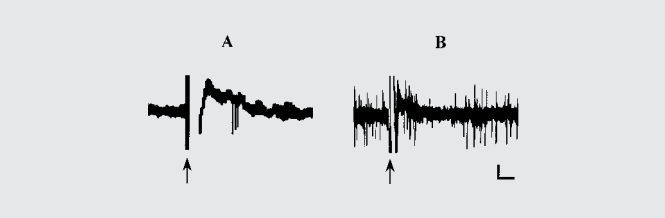

Fig. 3. Extracellular recordings showing excitatory (**A**) and inhibitory (**B**) effects of electrical stimulation of nBOR on rotundal cells. Three (**A**) or ten (**B**) sweeps are superimposed. Note that cell **A** is silent and fires one spike following one stimulation, whereas cell **B** is spontaneously active and clearly shows inhibition of visual and spontaneous firing by shocks. Electrical stimulation was delivered by passing rectangular pulses of 0.1 ms in duration and 500 μA in intensity at 0.2 Hz. Arrows point to electrical stimulation artifacts. Scales = 25 mV, 2.5 ms in **A**, and 50 mV, 10 ms in **B**.

Discussion

In the present study, both pharmacological blockade and electrical stimulation experiments show excitatory and inhibitory actions of the nucleus of the basal optic root on the avian nucleus rotundus in particular and those of the accessory optic system on the tectofugal pathway in general. Blockade of the nBOR by lidocaine can decrease or increase visual firing rates of rotundal cells, indicating that accessory optic cells can excite and inhibit rotundal cells, respectively. Lidocaine application sites marked with dye are all localized within the nBOR, and dye-marked recording sites of visual cells are in the nRt, indicating the existence of an accessory optic-rotundal interaction. Several studies [Ferrera et al., 1994; Wang et al., 1995, 2000; Li et al., 1998] have shown that lidocaine is an excellent tool for investigating interactions between neural structures. First, the specificity and reversibility of lidocaine effects on neuronal activity indicate that these effects are pharmacological but not toxicological [Wang et al., 1995]. Second, the action of lidocaine lasts long enough to examine its effects on visual responses elicited by motion [Wang et al., 1995, 2000; Li et al., 1998].

The results obtained with lidocaine blockade are confirmed by our electrical stimulation experiments, which show that accessory optic stimulation can excite or inhibit rotundal cells. Markings confirm that the recording sites are localized within the nRt, and electrical stimulation sites in the nBOR. These stimulation sites are marked at the negative pole of the bipolar electrode, which has been thought to be effective for electrical stimulation. The excitatory and inhibitory responses of rotundal cells to the nBOR stimulation are unlikely to originate from stimulation of the optic tract, which lies lateral to the nBOR, for three reasons: (1) results obtained using electrical stimulation are consistent with those using lidocaine, which is a local anesthetic to block neuronal transmission [Ferrera et al., 1994; Wang et al., 1995]; (2) latency of excitatory responses evoked in rotundal cells by the nBOR stimulation (average 9.4 ms) is much shorter than that of inhibitory responses (47 ms), which are likely to be produced via the subpretectal nuclei, implying that excitatory responses could occur through a direct route but not the optic tract-tectum-nucleus rotundus pathway, and (3) the active pole of the bipolar stimulating electrode is positioned within the nBOR as shown by electrolytic lesions.

These results are supported by a recent anatomical finding that, in addition to diverse projections to various regions in the mesencephalon, diencephalon and cerebellum [Brecha et al., 1980; Gioanni et al., 1984; Telford and Frost, 1989; Wylie and Linkenhoker, 1996], a few terminals are also seen within the SP, the IPS and nRt on the ipsilateral side after injecting an anterograde tracer into the nBOR [Wylie et al., 1997]. The SP/IPS complex sends inhibitory efferents to the nRt [Deng and Rogers, 1998b; Mpodozis et

al., 1996; Tömböl et al., 1994, 1999]. Therefore, it is suggested that the excitation might be exerted by a direct nBOR-nRt pathway, whereas the inhibition effects are caused by indirect pathways via the SP/IPS complex. In fact, immunohistochemical studies have shown that the nRt possesses homogeneously-distributed GABA-like terminals [Domenici et al., 1988, Ngo et al., 1992; Tömböl et al., 1994], which contain flattened vesicles and make synapses with rotundal cells [Ngo et al., 1992; Tömböl et al., 1994]. Furthermore, binding sites of GABA-benzodiazepine and $GABA_B$ receptors have been observed within the avian nRt [Dietl et al., 1988; Veenman et al., 1994]. On the other hand, glutamate receptor R4 subunits are heterogeneously distributed in the nRt, with R4-positive cell density decreasing from the dorsal to the ventral portion of the nucleus [Theiss et al., 1998]. This dorso-ventral variation might be related to a topographical projection from the tectum [Karten et al., 1997; Hellmann and Güntürkün,1999] and/or to the functional divisions of the nucleus rotundus [Wang and Frost, 1992; Wang et al., 1993]. However, our small sample of rotundal cells made it impossible to determine regional variation, if any, of excitatory and inhibitory responses evoked in the nRt by chemical blockade or electrical stimulation of the nBOR. This limited sampling was necessary to obtain solid data as we marked every recording site in the nRt and most drug-application and electrical stimulation sites in the nBOR.

It has been shown that the nRt receives bilateral projections from the optic tectum [Bischof and Niemann, 1990; Karten et al., 1997; Hellmann and Güntürkün,1999], and the SP/IPS complex receives input from the ipsilateral tectum in pigeons [Karten et al., 1997] and chicks [Tömböl et al., 1999] as well as from the contralateral tectum in the zebra finch [Bischof and Niemann, 1990]. Dual actions of the nBOR on rotundal cells through a direct and an indirect pathway are similar to those of tectal cells on the nRt. Our previous studies [Gao et al., 1995; Huang et al., 1998] have suggested that the direct tecto-rotundal pathway is excitatory and glutamatergic, and the indirect tecto-rotundal pathway via the subpretectal nuclei is inhibitory and GABAergic. Therefore, there might be at least two stages where tectal and accessory optic information could interact: inputs from the tectum and the nBOR interact within the nRt, or tectal and accessory optic information interact within the SP/IPS complex first and then this integrated information is sent to the nRt by inhibitory fibers. In view of the finding that the nRt is organized into several functionally distinct divisions where different types of visual information such as luminance, color and motion in depth are processed [Wang and Frost, 1992; Wang et al., 1993], and that the nBOR can exert excitatory or inhibitory effects on responses of visual neurons throughout the nRt as shown by markings in the present study (fig. 1), it appears that the nBOR could modulate visual responses in several, if not all, functional domains of the nRt. Therefore, the ways in which the nBOR affects the processing of different types of visual information within the nRt should be an object of further study.

Acknowledgments

This work was supported by the National Natural Science Foundation of China and by the Chinese Academy of Sciences.

References

Bischof, H.J., and J. Niemann (1990) Contralateral projections of the optic tectum in the zebra finch *(Taenopygia guttata castanotis).* Cell Tiss. Res., *262:* 307–313.

Brecha, N., H.J. Karten, and S.P. Hunt (1980) Projections of the nucleus of the basal optic root in the pigeon: an autoradiographic and horseradish peroxidase study. J. Comp. Neurol., *189:* 615–670.

Deng, C., and L.J. Rogers (1998a) Bilaterally projecting neurons in the two visual pathways of chicks. Brain Res., *794:* 281–290.

Deng, C., and L.J. Rogers (1998b) Organization of the tectorotundal and SP/IPS-rotundal projections in the chick. J. Comp. Neurol., *394:* 171–185.

Dietl, M.M., R. Cortés, and J.M. Palacios (1988) Neurotransmitter receptors in the avian brain. III. GABA-benzodiazepine receptors. Brain Res., *439:* 366–371.

Domenici, L., H.J. Waldvogel, C. Matute, and P. Streit (1988) Distribution of GABA-like immunoreactivity in the pigeon brain. Neuroscience, *25:* 931–950.

Engelage, J., and H.J. Bischof (1993) The organization of the tectofugal pathway in birds: a comparative review. *In* Vision, Brain, and Behavior in Birds (ed. by H.P. Zeigler and H.J. Bischof), MIT Press, Mass., pp. 137–158.

Ferrera, V.P., T.A. Nealey, and J.H.R. Maunsell (1994) Responses in macaque visual area V4 following inactivation of the parvocellular and magnocellular LGN pathways. J. Neurosci., *14:* 2080–2088.

Fite, K.V., N. Brecha, H.J. Karten, and S.P. Hunt (1981) Displaced ganglion cells and the accessory optic system of pigeon. J. Comp. Neurol., *195:* 279–288.

Gao, H.F., G.Y. Wu, B.J. Frost, and S.R. Wang (1995) Excitatory and inhibitory neurotransmitters in the nucleus rotundus of pigeons. Vis. Neurosci., *12:* 819–825.

Gioanni, H., J. Rey, J. Villalobos, and A. Dalbera (1984) Single unit activity in the nucleus of the basal optic root (nBOR) during optokinetic, vestibular and visuo-vestibular stimulations in the alert pigeon *(Columba livia).* Exp. Brain Res., *57:* 49–60.

Güntürkün, O., D. Miceli, and M. Watanabe (1993) Anatomy of the avian thalamofugal pathway. *In* Vision, Brain, and Behavior in Birds (ed. by H.P. Zeigler and H.J. Bischof), MIT Press, Mass., pp. 115–135.

Hellmann, B., and O. Güntürkün (1999) Visual-field-specific heterogeneity within the tectorotundal projection of the pigeon. Eur. J. Neurosci., *11:* 2635–2650.

Hellon, R.F. (1971) The marking of electrode tip positions in nervous tissue. J. Physiol. (Lond), *214:* 12.

Hodos, W., and H.J. Karten (1966) Brightness and pattern discrimination deficits in the pigeon after lesions of nucleus rotundus. Exp. Brain Res., *2:* 151–167.

Hodos, W., H.J. Karten, and J.C. Bonbright (1973) Visual intensity and pattern discrimination after lesions of the thalamofugal visual pathway in pigeons. J. Comp. Neurol., *148:* 447–467.

Huang, L.H., J.L. Li, and S.R. Wang (1998) Glutamatergic neurotransmission from the optic tectum to the contralateral nucleus rotundus in pigeons. Brain Behav. Evol., *52:* 55–60.

Karten, H.J., and W. Hodos (1967) A Stereotaxic Atlas of the Brain of the Pigeon *(Columba livia)*. Johns Hopkins Press, Baltimore, Md.

Karten, H.J., K.V. Fite, and N. Brecha (1977) Specific projection of displaced ganglion cells upon accessory optic system in the pigeon *(Columba livia).* Proc. Natl. Acad. Sci. USA., *74:* 1753–1756.

Karten, H.J., K. Cox, and J. Mpodozis (1997) Two distinct populations of tectal neurons have unique connections within the retinotectorotundal pathway of the pigeon *(Columba livia).* J. Comp. Neurol., *387:* 449–465.

Li, J.L., Q. Xiao, Y.X. Fu, and S.R. Wang (1998) Centrifugal innervation modulates visual activity of tectal cells in pigeons. Vis. Neurosci., *15:* 411–415.

McKenna, O.C., and J. Wallman (1985) Accessory optic system and pretectum of birds: comparisons with those of other vertebrates. Brain Behav. Evol., *26:* 91–116.

Mpodozis, J., K. Cox, T. Shimizu, H.J. Bischof, W. Woodson, and H.J. Karten (1996) GABAergic inputs to the nucleus rotundus. J. Comp. Neurol., *374:* 204–222.

Ngo, T.D., A. Németh, and T. Tömböl (1992) Some data on GABA-ergic innervation of nucleus rotundus in chicks. J. Hirnforsch., *33:* 335–355.

Nickla, D.L., M.D. Gottlieb, G. Marin, X. Rojas, L.R.G. Britto, and J. Wallman (1994) The retinal targets of centrifugal neurons and the retinal neurons projecting to the accessory optic system. Vis. Neurosci., *11:* 401–409.

Reiner, A., N. Brecha, and H.J. Karten (1979) A specific projection of retinal displaced ganglion cells to the nucleus of the basal optic root in the chicken. Neuroscience, *4:* 1679–1688.

Telford, L., and B.J. Frost (1989) Functional activity in the accessory optic system during visual, vestibular and visual-vestibular stimulation in the pigeon. Exp. Brain Res., *77:* 391–397.

Theiss, C., B. Hellmann, and O. Güntürkün (1998) The differential distribution of AMPA-receptor subunits in the tectofugal system of the pigeon. Brain Res., *785:* 114–128.

Tömböl, T., G. Egedi, and A. Németh (1994) Phaseolus vulgaris lectin labeled and GABA immunogold stained terminals in nucleus rotundus. An EM study. J. Brain Res., *35:* 233–252.

Tömböl, T., A. Németh, T. Sebestény, and A. Alpár (1999) Electron microscopic data on the neurons of nuclei subpretectalis and posterior-ventralis thalami. A combined immunohistochemical study. Anat. Embryol., *199:* 169–183.

Veenman, C.L., R.L. Albin, E.K. Richfield, and A. Reiner (1994) Distributions of $GABA_A$, $GABA_B$, and benzodiazepine receptors in the forebrain and midbrain of pigeons. J. Comp. Neurol., *344:* 161–189.

Wang, Y.C., and B.J. Frost (1992) 'Time to collision' is signalled by neurons in the nucleus rotundus of pigeon. Nature, *356:* 236–238.

Wang, Y.C., S.Y. Jiang, and B.J. Frost (1993) Visual processing in pigeon nucleus rotundus: luminance, color, motion, and looming subdivisions. Vis. Neurosci., *10:* 21–30.

Wang, S.R., Y.C. Wang, and B.J. Frost (1995) Magnocellular and parvocellular divisions of pigeon nucleus isthmi differentially modulate visual responses in the tectum. Exp. Brain Res., *104:* 376–384.

Wang, Y., J. Xiao, and S.R. Wang (2000) Excitatory and inhibitory receptive fields of tectal cells are differentially modulated by magnocellular and parvocellular divisions of the pigeon nucleus isthmi. J. Comp. Physiol. A, *186:* 505–511.

Wylie, D.R., and B.J. Frost (1990) Binocular neurons in the nucleus of the basal optic root (nBOR) of the pigeon are selective for either translational or rotational visual flow. Vis. Neurosci., *5:* 489–495.

Wylie, D.R., and B. Linkenhoker (1996) Mossy fibers from the nucleus of the basal optic root project to the vestibular and cerebellar nuclei in pigeons. Neurosci. Lett., *219:* 83–86.

Wylie, D.R., B. Linkenhoker, and K.L. Lau (1997) Projections of the nucleus of the basal optic root in pigeons *(Columba livia)* revealed with biotinylated dextran amine. J. Comp. Neurol., *384:* 517–536.

Wylie, D.R., W.F. Bischof, and B.J. Frost (1998) Common reference frame for neural coding of translational and rotational optic flow. Nature, *392:* 278–282.