

# Electrical interaction between neurons in the pigeon isthmo-optic nucleus

Jing Hu, Wen-Chang Li, Quan Xiao and Shu-Rong Wang

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

[Received 1 June 1999; Accepted 25 August 1999]

**ABSTRACT:** The present study used brain slices to investigate interneuronal communication in the isthmo-optic nucleus in pigeons. Electrical stimulation of the isthmo-optic tract generated a transmembrane potential in isthmo-optic cells that was obtained by subtracting the extracellular potential from the intracellular potential. This transmembrane potential resulted in enhancement of excitability and/or in production of spikes in 42 (63%) cells. In most cases, proximal axons marked in brain slices by Lucifer yellow were too short to reach the stimulation site, indicating that spikes were evoked by electrical field effect or ephaptic interaction produced by nearby cells whose axons were activated by stimulation. Eleven (16%) cells discharged a spikelet, or spike that was abolished by hyperpolarizing current injection leaving a spikelet. Markings of five of these cells all indicated the presence of dye-couplings, each of which consisted of a pair of cells. Fourteen (21%) cells only produced antidromic spikes with a short and constant latency. Four of these cells were marked and their axons passed through the stimulation site, implying that their nearby cells' axons might be cut too short to be electrically stimulated or they were in a sparse-cell area. The present results provide electrophysiological and neuroanatomical evidence that both electrical field effect and electrical coupling may play important roles in interneuronal communication within the pigeon isthmo-optic nucleus. These findings are supported by anatomical arrangement of densely packed cells and their oriented dendrites in this centrifugal nucleus. © 2000 Elsevier Science Inc.

**KEY WORDS:** Brain slice, Electrical field effect, Gap junction, Isthmo-optic nucleus, Pigeon, Transmembrane potential.

## INTRODUCTION

It has been generally accepted that communication between neurons is mediated either by chemical synapses via transmitters and receptors, or by electrical synapses through gap junctions. Several lines of evidence have also shown that transient electrical field produced by population spikes can change the excitability of synaptically unconnected neurons [9,17,18,30,33,34,40]. This type of interneuronal communication has been referred to as electrical field effect or ephaptic interaction [19]. Though early studies were performed on interaction between axons positioned side by side, increasing number of studies have been devoted to electrical field action in the mammalian hippocampus [17,23,30,33,34,40], be-

cause it may be involved in synchronous or epileptiform activity of cortical neurons. Moreover, the fact that pyramidal cells in the hippocampus are densely packed and their primary dendrites in parallel meets an anatomical criterion for generating electrical field effect [34].

These anatomical features also exist in the isthmo-optic nucleus in ground-feeding birds such as pigeons. It receives input primarily from the ipsilateral tectum [6,27,38,43] and projects to the contralateral retina [41,42,44], terminating in either “convergent” or “divergent” modes on amacrine cells and/or displaced ganglion cells [5,10,25,29,44]. This retinopetal nucleus consists of two parallel layers of densely packed cells, whose dendrites orient into and intermingle in neuropil between the opposing layers [3,13,16, 21,26]. Previous studies have indicated the existence of the attachment plaques [2] and gap junctions [21,22] between some isthmo-optic cells. Therefore, the present study was undertaken to reveal the possible roles of electrical coupling and electrical field effect in interneuronal communication within the pigeon isthmo-optic nucleus.

## MATERIALS AND METHODS

The experiments were performed on brain slices of 22 adult pigeons (*Columba livia*) of either sex and 310–480 g body weight (b.wt.) following guidelines regarding the use of animals in neuroscience research approved by the Society for Neuroscience. The animal was anesthetized with ketamine hydrochloride (40 mg/100 g b.wt.), and then decapitated. The brain was immediately removed from the skull, and washed in ice-cold Krebs-Ringer solution containing (in mM) NaCl, 124; KCl, 5; CaCl<sub>2</sub>, 2; MgSO<sub>4</sub>, 2; KH<sub>2</sub>PO<sub>4</sub>, 1.25; NaHCO<sub>3</sub>, 26; glucose 10 [14], oxygenated with a mixture of 95% O<sub>2</sub> and 5% CO<sub>2</sub>. A midbrain tissue block including the isthmo-optic nucleus (ION) and the isthmo-optic tract was trimmed and glued on the stage of a Vibroslice (Campden Instruments Ltd., 752M; LE12 7IZ, UK). Slices containing the ION and the tract were sectioned sagittally at 350 μm in thickness, and then transferred from a storage container into the recording chamber (BSC-HT; Medical System Corp., Greenvale, NY, USA) perfused at a rate of 2 ml/min with Krebs-Ringer solution bubbled with a mixture of 95% O<sub>2</sub> and 5% CO<sub>2</sub>. The slices were incubated at 30°C for 60 min.

\* Address for correspondence: Shu-Rong Wang, Institute of Biophysics, Chinese Academy of Sciences, 15 Datun, Beijing 100101, China. E-mail: wangsr@mimi.cnc.ac.cn

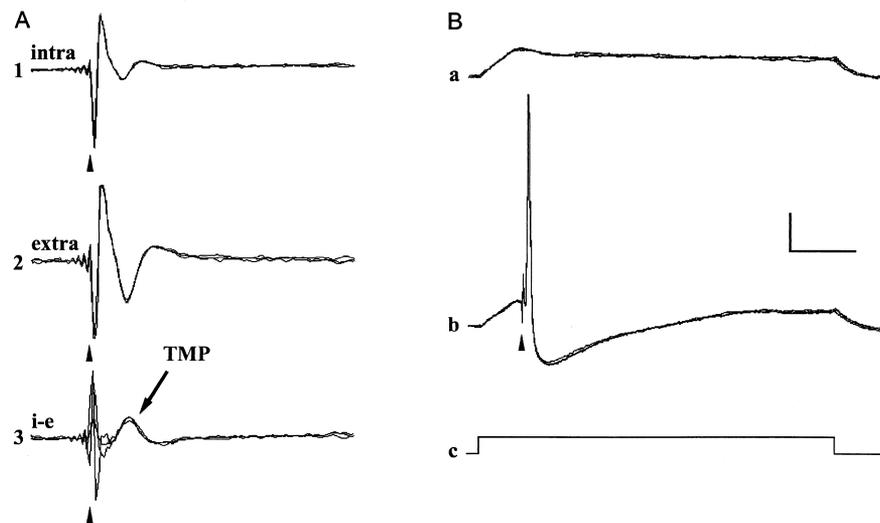


FIG. 1. (A) Transmembrane potential (TMP, 3) of an isthmo-optic cell was obtained by computer subtraction of the extracellular potential 2 from the intracellular potential 1 following electrical stimulation (0.1 mA in intensity, 30  $\mu$ s in duration) to the isthmo-optic tract. (B) A combination of depolarization (a) produced by subthreshold depolarizing current injection (0.2 nA, c) and TMP induced by electrical stimulation to the tract (b) elicited a spike from the cell at threshold of 6.9 mV (b). Three sweeps were superimposed. Arrowheads point to electrical artifacts. Scales: (A) 2 mV, 0.5 ms; (B) 10 mV, 10 ms.

Under an operating microscope, the isthmo-optic fibers in sagittal sections gather around the ION, bundling into the isthmo-optic tract rostrally and traveling rostradorsally to the nucleus. A bipolar tungsten electrode with poles 400  $\mu$ m apart was placed across the tract and 1000–1500  $\mu$ m distance from the nucleus. Rectangular pulses of 10–30  $\mu$ s in duration and 0.01–0.8 mA in intensity were delivered for electrical stimulation. For intracellular recording and staining, a micropipette filled with either 3 M potassium acetate or a 5% solution of Lucifer yellow (dilithium salt; Sigma Chemical Co., St. Louis, MO, USA) in 1 M LiCl [30] was used. The electrode tip was broken to about 0.5  $\mu$ m by touching it against the edge of a glass slide with a micromanipulator under a microscope. The micropipette was advanced into the nucleus in slices under microscopic control. Isthmo-optic neurons were impaled by applying brief positive current pulses (4 nA in intensity, 0.3 s in duration). Intracellular impalement was signaled by a sudden direct current (d.c.) drop of 40–80 mV. Following electrical stimulation to the tract, intracellular potential was recorded and then extracellular field potential recorded after the electrode was just pulled outside the cell. These potentials were amplified, stored on magnetic tapes (TEAC RD-135T Data Recorder; TEAC Corp., Tokyo), and then analyzed. Transmembrane potential (TMP) was obtained by subtracting the field potential from the intracellular potential [18,34] with a computer program. In some experiments, the dye was injected by passing negative current of 2–4 nA through the electrode for 1–10 min. One injection of Lucifer yellow was made in each slice. After 0.5–2 h survival, the slices were removed from the recording chamber, fixed in 8% paraformaldehyde and kept in a refrigerator overnight. The slices were rinsed with physiological saline and then placed in 100% dimethylsulfoxide (DMSO) for 20 min [12]. The DMSO-mounted slices containing Lucifer yellow-marked ION cells were coverslipped and observed or photographed with a fluorescence microscope.

## RESULTS

Sixty-seven isthmo-optic cells were intracellularly impaled and examined for actions of electrical field and electrical coupling on the cells' excitability. Their resting membrane potential was negative  $56.2 \pm 9.5$  mV ( $n = 67$ ). Following antidromic stimulation to the isthmo-optic tract, intracellular potential and extracellular field potential were recorded from these cells. Transmembrane potential was obtained by subtracting the field potential from the intracellular potential [18,34], showing that it was a depolarizing potential in nature (Fig. 1A). Therefore, in cases that subthreshold depolarization was reached by injecting positive current (0.01–1 nA,  $n = 19$ ), superimposed TMP that was caused by subthreshold current stimulation to the tract (0.05–0.2 nA,  $n = 19$ ) could elicit a spike (Fig. 1B). In 42 of the 67 (63%) cells, TMP did not occur when stimulation current intensities ranged from 0.01–0.02 mA, but it occurred and increased in amplitude from 2.0–9.8 mV, as current intensities increased from 0.03–0.1 mA. Further increase in stimulation current intensities resulted in generating TMP that was large enough to produce spikes. Their latencies were shortened from 1.2–0.5 ms ( $n = 42$ ) as current intensities increased from 0.05–0.5 mA. Increasing stimulation intensities to 0.2–0.8 mA produced antidromic spikes characterized by constant short latencies ( $0.25 \pm 0.05$  ms,  $n = 8$ ) (Fig. 2). The spikes induced by TMP were abolished by hyperpolarizing current injection ( $0.20 \pm 0.12$  nA,  $n = 19$ ), whereas the antidromic spikes by larger hyperpolarizing current ( $0.93 \pm 0.49$  nA,  $n = 7$ ). Five of seven cells marked by the dye possessed axons of 100–300  $\mu$ m long and their perikarya were 1230–1930  $\mu$ m distant from the stimulation site, convincingly indicating that the spikes (latencies = 0.5–1.0 ms, current intensities = 0.1–0.5 mA) in these cells were induced by electrical field effect or ephaptic interaction generated by nearby cells whose axons were antidromically activated (Fig. 4C, Bc). Two others' axons were long enough to reach the stimulating electrode. They not only produced TMP-induced spikes (laten-

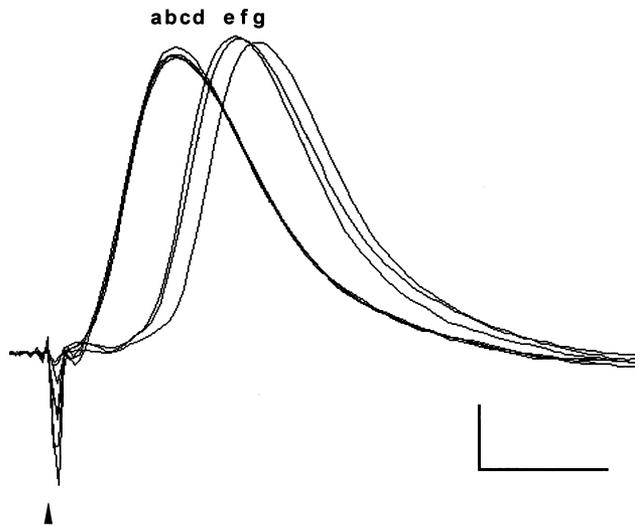


FIG. 2. Latencies of spikes of an isthmo-optic cell excited by the electrical field were shortened as current intensities of electrical stimulation increased from 0.025 to 0.1 mA (g–e). Further increase of current intensities to 0.1–0.5 mA produced antidromic spikes with a short and constant latency (a–d). Arrowhead points to electrical artifact. Scales: 10 mV, 1.0 ms.

cies = 0.5–0.8 ms, intensities = 0.05–0.1 mA), but also antidromic potentials (0.25–0.32 ms, 0.2–0.5 mA) following electrical stimulation to the tract.

Eleven of the 67 (16%) cells fired spikes and/or spikelets following electrical stimulation to the tract. Among these, seven cells produced spikes ( $50.0 \pm 27.6$  mV,  $n = 7$ ) which were abolished by hyperpolarizing current injection ( $0.50 \pm 0.27$  nA,  $n = 7$ ), with occurring spikelets ( $25.6 \pm 6.8$  mV,  $n = 7$ ). These spikelets were abolished by larger hyperpolarizing current injection ( $0.99 \pm 0.88$  nA,  $n = 7$ ). Four others only produced spikelets ( $18.0 \pm 2.4$  mV,  $n = 4$ ), which were abolished by hyperpolarizing current injection ( $0.79 \pm 0.24$  nA,  $n = 4$ ). As shown in Fig. 3, electrical stimulation of 0.1–0.3 mA produced a spike which was abolished by hyperpolarizing current injection, with occurring a spikelet. The spikelet was eliminated by larger hyperpolarizing current. This cell was marked by the dye, showing that it was a twin-cells [21]. One of the cells had an axon of 700  $\mu\text{m}$  long and the other's axon was 1700  $\mu\text{m}$  long, passing through the stimulation site (Figs. 4A,Ba,b). Three cells producing both spikes and spikelets and two cells with spikelets only were marked by the dye, and they all showed dye-couplings, each of which was composed of a pair of cells. One of the double cells had a short axon (250–700  $\mu\text{m}$ ) and the other had a long axon (1300–2000  $\mu\text{m}$ ), with perikarya being 1300–1600  $\mu\text{m}$  distant from the stimulation site.

In the remaining 14 (21%) cells, only antidromic potentials were recorded following electrical stimulation to the tract. Their latencies were short ( $0.29 \pm 0.11$  ms,  $n = 14$ ) and did not change as current intensities increased from 0.01 to 0.8 mA. A combination of subthreshold stimulation (0.01–0.1 mA) to the tract with subthreshold depolarizing current injection (0.1–4 nA) did not evoke spikes other than antidromic potentials. These potentials were abolished by hyperpolarizing current injection ( $0.93 \pm 0.49$  nA,  $n = 7$ ), but no spikelets occurred. Dye-markings of four of these cells showed that they had axons reaching the stimulation site. These cells might have nearby cells whose axons were cut too

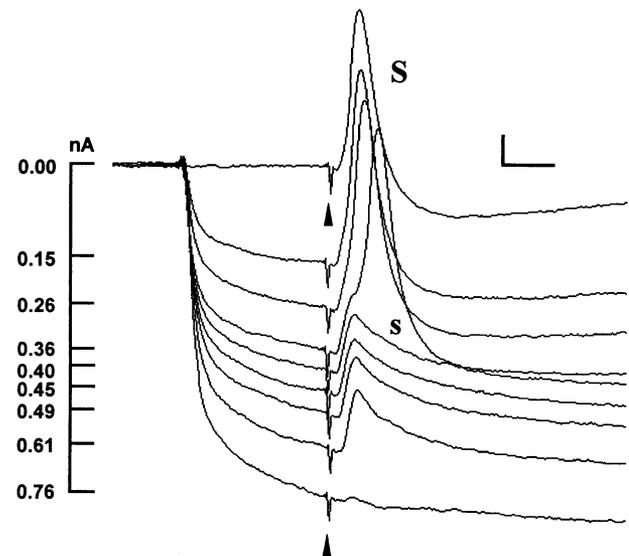


FIG. 3. Effect of hyperpolarizing current injections (intensities in nA on the left scale) on spikes (S) and spikelets (s) evoked by electrical stimulation (0.5 mA, 10  $\mu\text{s}$ ) to the isthmo-optic tract. At 0.40 nA, a spike abolished and spikelet occurred. The spikelet was eliminated by a larger hyperpolarizing current injection. These responses were recorded from an isthmo-optic cell that was dye-coupled with another cell shown in Fig. 4. Arrowhead points to electrical artifact. Scales: 10 mV, 2 ms.

short to reach the stimulation site or be in a sparse-cell area of the nucleus.

## DISCUSSION

The present study indicates that electrical field effect and electrical coupling via gap junction play important roles in communication between isthmo-optic neurons in pigeons. These interactions are of fundamental significance to the centrifugal or retinopetal system, because of a 1:1:1 relationship between tectal cells projecting to the ION, retinopetal cells and their retinal target cells [22,36,38,43,44]. Orthodromic [1,31,33] and antidromic [31,33, 34] activation of neuronal pathways can produce intracellular potentials in the recorded neurons and electrical field potentials in the extracellular space, thereby resulting in transmembrane potentials that are obtained by subtracting the extracellular potential from the intracellular potential [18,34]. The present study shows that TMP produced by electrical stimulation of the isthmo-optic tract can enhance the excitability of isthmo-optic neurons or discharge spikes. These ephaptic spikes are characterized by the following properties: (1) They could be produced by a combination of TMP with subthreshold current depolarization, or by suprathreshold electrical stimulation to the tract; (2) their latencies are reduced as current intensities increase. Following larger current stimulation, isthmo-optic cells produce antidromic spikes with a short and constant latency. Antidromic and ephaptic spikes can be eliminated by hyperpolarizing current injection. However, in some cases, spikelets occur after ephaptic spikes are eliminated by hyperpolarizing current. These spikelets also can be abolished by further increase in hyperpolarizing current intensities.

Dye-markings show that proximal axons of cells firing spikes to the tract stimulation are usually shorter than the distance from the perikarya to the stimulation site. This implies that electrical

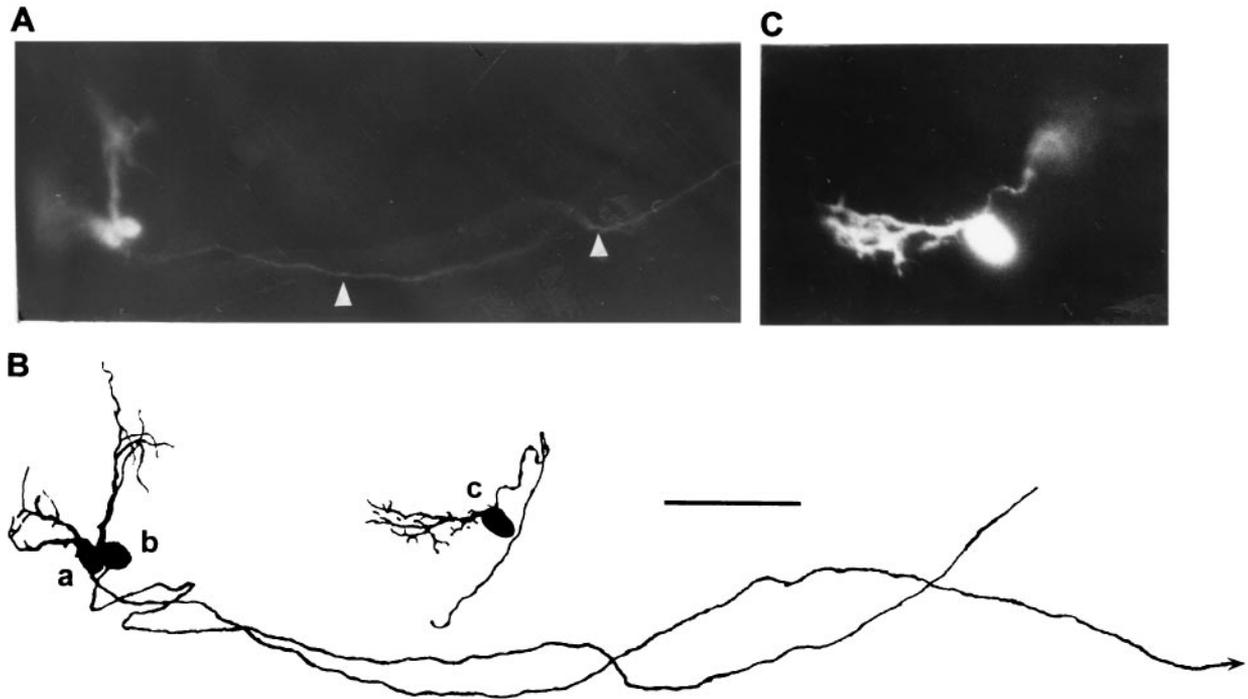


FIG. 4. Microphotographs (A,C) and their drawings (B) of three isthmo-optic cells marked with Lucifer yellow. Dye-coupled cells in A were drawn in Ba,b and single cell in C corresponded to Bc. Cell a had a long axon passing through the electrode site (arrow) but cell b had a short proximal axon. The axon of cell c took a hairpin-loop way to the isthmo-optic tract. Electrophysiological recordings of the dye-coupled cells (a, b) are shown in Fig. 3. Arrowheads point to axons. Scale bars: 100  $\mu\text{m}$ .

field effect and/or electrical coupling via gap junction play important roles in activating isthmo-optic cells. The cell that discharges both spike and spikelet or spikelet alone is always dye-coupled with another cell, as shown in the present study. It is, therefore, most likely that the spikelet may originate from the dye-coupled cell [23,24] and induce a spike in the recorded cell by itself [24] or with co-action of depolarizing TMP. The spike can be delayed by hyperpolarizing current, revealing a small depolarization that is suggested to be coupling potential [24]. It, of course, can not exclude the possibility that these full-sized spikes are antidromic ones and spikelets may be axon hillock potentials. Small potentials or spikelets are also present in various brain regions of adult and developing mammals, and they may result from the existence of gap junction [8,11,23,32]. Gap junction can be modulated by transmitters [32,39]. Nitric oxide is likely to be a transmitter in the centrifugal pathway [28] and isthmo-optic cells contain nitric oxide synthase [Xiao, Lázár, Xu, and Wang, unpublished data]. Therefore, it appears that nitric oxide would modulate interneuronal communication within the nucleus. Recent studies have shown that electrical coupling via gap junction, but not electrical field effect or ephaptic interaction, underlies synchronizing high frequency oscillations in the rat hippocampus slices [8], though the field effect may play a role in epileptiform discharge propagation in the rat piriform cortex [7]. Some isthmo-optic cells only produce antidromic spikes characterized by short and constant latencies. This phenomenon may be explained by suggesting that proximal axons of these cells are long enough to reach the stimulation site, but their neighboring cells are not excited because of their short proximal axons, or these cells are actually in a cell-sparse region without significant electrical field effect or ephaptic interaction.

Anatomically, like the mammalian hippocampus [9,18], the

pigeon ION is also a favorable model for studying electrical field effect, because it consists of two parallel layers of densely packed cells, whose dendrites orient into and intermingle in neuropil between the opposing layers [3,13,16,26]. This organization is feasible for the current to flow inward at one point and outward at another of the same cell [34]. The present results show that TMP created within the nucleus by electrical stimulation to the tract could be large enough to fire isthmo-optic cells. Furthermore, there exist the attachment plaques [2], which are probably dye-couplings between the ION cells [21]. These suggest that isthmo-optic neurons may be synchronously excited through electrical field effect and electrical synapses. Taken together with the previous studies showing that the optic tectum directly excites the ION [22], which in turn enhances visual responses of retinal ganglion cells [4,15,33] and modulates tectal activity [20], it is postulated that birds such as pigeons can promptly provide, through this centrifugal system, early warning of predators [15,35], or rapidly switch its visual attention to threatening objects [4,20,37].

#### ACKNOWLEDGEMENTS

This work was supported by the National Natural Science Foundation of China and by the Chinese Academy of Sciences. We are grateful to Mrs. Shun-Yi Wei for her excellent secretarial assistance.

#### REFERENCES

1. Andersen, P.; Bliss, T. V. P.; Skrede, K. K. Unit analysis of hippocampal population spikes. *Exp. Brain Res.* 13:208–221; 1971.
2. Angaut, P.; Repérant, J. A light and electron microscopic study of the nucleus isthmo-opticus in the pigeon. *Arch. Anat. Microsc.* 67:63–78; 1978.

3. Clarke, P. G. H.; Caranzano, F. Dendritic development in the isthmo-optic nucleus of chick embryos. *Dev. Neurosci.* 7:161–163; 1985.
4. Clarke, P. G. H.; Gyger, M.; Catsicas, S. A centrifugally controlled circuit in the avian retina and its possible role in visual attention switching. *Vis. Neurosci.* 13:1043–1048; 1996.
5. Cowan, W. M. Centrifugal fibers to the avian retina. *Br. Med. Bull.* 26:112–118; 1970.
6. Crossland, W. J.; Hughes, C. P. Observations on the afferent and efferent connections of the avian isthmo-optic nucleus. *Brain Res.* 145:239–256; 1978.
7. Demir, R.; Haberly, L. B.; Jackson, M. B. Voltage imaging of epileptiform activity in slices from rat piriform cortex: Onset and propagation. *J. Neurophysiol.* 80:2727–2742; 1998.
8. Draguhn, A.; Traub, R. D.; Schmitz, D.; Jefferys, J. G. R. Electrical coupling underlies high-frequency oscillations in the hippocampus *in vitro*. *Nature* 394:189–192; 1998.
9. Faber, D. S.; Korn, H. Electrical field effects: Their relevance in central neural networks. *Physiol. Rev.* 63:821–863; 1989.
10. Fritzsche, B.; Crajon de Caprona, M. D.; Clarke, P. G. H. Development of two morphological types of retinapetal fibers in chick embryos, as shown by the diffusion along axons of a carbocyanine dye in the constant retina. *J. Comp. Neurol.* 300:405–421; 1990.
11. Grace, A. A.; Bunney, B. S. Intracellular and extracellular electrophysiology of nigral dopaminergic neurons. Evidence for electrotonic coupling. *Neuroscience* 10:333–348; 1983.
12. Grace, A. A.; Llinàs, R. Dehydration-induced morphological artifacts in intracellularly stained neurons: Circumvention using rapid DMSO clearing. *Neuroscience* 16:461–475; 1985.
13. Güntürkün, O. A Golgi study of the isthmic nuclei in the pigeon (*Columba livia*). *Cell Tissue Res.* 248:439–448; 1987.
14. Hardy, O.; Audinat, E.; Jassik-Gerschenfeld, D. Electrophysiological properties of neurons recorded intracellularly in slices of the pigeon optic tectum. *Neuroscience* 23:305–318; 1987.
15. Holden, A. L. Centrifugal pathways to the retina: Which way does the “searchlight” point? *Vis. Neurosci.* 4:493–495; 1990.
16. Holden, A. L.; Powell, T. P. S. The functional organization of the isthmo-optic nucleus in the pigeon. *J. Physiol.* 223:419–447; 1972.
17. Jefferys, J. G. R. Influence of electric fields on the excitability of granule cells in guinea-pig hippocampal slices. *J. Physiol.* 319:143–152; 1981.
18. Jefferys, J. G. R. Nonsynaptic modulation of neuronal activity in the brain: Electric currents and extracellular ions. *Physiol. Rev.* 75:689–723; 1995.
19. Korn, H.; Faber, D. S. Electrical field effect interactions in the vertebrate brain. *Trends Neurosci.* 3:6–9; 1980.
20. Li, J. L.; Xiao, Q.; Fu, Y. X.; Wang, S. R. Centrifugal innervation modulates visual activity of tectal cells in pigeons. *Vis. Neurosci.* 15:411–415; 1998.
21. Li, W. C.; Wang, S. R. Morphology and dye-coupling of cells in the pigeon isthmo-optic nucleus. *Brain Behav. Evol.* 53:67–74; 1999.
22. Li, W. C.; Hu, J.; Wang, S. R. Tectal afferents monosynaptically activate neurons in the pigeon isthmo-optic nucleus. *Brain Res. Bull.* 49:203–208; 1999.
23. MacVicar, B. A.; Dudek, F. E. Electrotonic coupling between pyramidal cells: A direct demonstration in rat hippocampal slices. *Science* 213:782–785; 1981.
24. Martin, A. R.; Pilar, G. Dual mode of synaptic transmission in the avian ciliary ganglion. *J. Physiol.* 168:443–463; 1963.
25. Maturana, H. R.; Frenk, S. Synaptic connections of the centrifugal fibers in the pigeon retina. *Science* 150:359–362; 1965.
26. McGill, J. I.; Powell, T. P. S.; Cowan, W. M. The organization of the projection of the centrifugal fibers to the retina in the pigeon. *J. Anat.* 100:35–49; 1966.
27. Miceli, D.; Repérant, J.; Marchand, L.; Rio, J. P. Retrograde transneuronal transport of the fluorescent dye rhodamine  $\beta$ -isothiocyanate from the primary and centrifugal visual systems in the pigeon. *Brain Res.* 601:289–298; 1993.
28. Morgan, I. G.; Miethke, P.; Li, Z. K. Is nitric oxide a transmitter of the centrifugal projection to the avian retina? *Neurosci. Lett.* 168:5–7; 1994.
29. Nickla, D. L.; Gottlieb, M. D.; Marin, G.; Rojas, X.; Britto, L. R. G.; Wallman, J. The retinal targets of centrifugal neurons and the retinal neurons projecting to the accessory optic system. *Vis. Neurosci.* 11:401–409; 1994.
30. Onn, S. P.; Grace, A. A. Dye-coupling between rat striatal neurons recorded *in vivo*: Compartmental organization and modulation by dopamine. *J. Neurophysiol.* 71:1917–1934; 1994.
31. Richardson, T. L.; Turner, R. W.; Miller, J. J. Extracellular fields influence transmembrane potentials and synchronization of hippocampal neuronal activity. *Brain Res.* 294:255–262; 1984.
32. Strata, F.; Atzori, M.; Molnar, M.; Ugolini, G.; Berretta, N.; Cherubini, E. Nitric oxide sensitive depolarization-induced hyperpolarization: A possible role for gap junctions during development. *Eur. J. Neurosci.* 10:397–403; 1998.
33. Taylor, C. P.; Dudek, F. E. Synchronous neural afterdischarges in rat hippocampal slices without active chemical synapses. *Science* 218:810–812; 1982.
34. Taylor, C. P.; Dudek, F. E. Excitation of hippocampal pyramidal cells by an electrical field effect. *J. Neurophysiol.* 52:126–142; 1984.
35. Uchiyama, H. Centrifugal pathways to the retina: Influence of the optic tectum. *Vis. Neurosci.* 3:183–206; 1989.
36. Uchiyama, H.; Ito, H. Target cells for the isthmo-optic fibers in the retina of the Japanese quail. *Neurosci. Lett.* 154:35–38; 1993.
37. Uchiyama, H.; Nakamura, S.; Imazono, T. Long-range competition among the neurons projecting centrifugally to the quail retina. *Vis. Neurosci.* 15:417–423; 1998.
38. Uchiyama, H.; Yamamoto, N.; Ito, H. Tectal neurons that participate in centrifugal control of the quail retina: A morphological study by means of retrograde labeling with biocytin. *Vis. Neurosci.* 13:1119–1127; 1996.
39. Velazquez, J. L.; Han, D.; Carlen, P. L. Neurotransmitter modulation of gap junctional communication in the rat hippocampus. *Eur. J. Neurosci.* 9:2522–2531; 1997.
40. Vigmond, E. J.; Velazquez, J. L. P.; Valiante, T. A.; Bardakjian, B. L.; Carlen, P. L. Mechanisms of electrical coupling between pyramidal cells. *J. Neurophysiol.* 78:3107–3116; 1997.
41. Weidner, C.; Repérant, J.; Desroches, A. M.; Miceli, D.; Vesselkin, N. P. Nuclear origin of the centrifugal visual pathway in birds of prey. *Brain Res.* 436:153–160; 1987.
42. Wolf-Oberhollenzer, F. A study of the centrifugal projections to the pigeon retina using two fluorescent markers. *Neurosci. Lett.* 73:16–20; 1987.
43. Woodson, W.; Reiner, A.; Anderson, K.; Karten, H. J. Distribution, laminar location, and morphology of tectal neurons projecting to the isthmo-optic nucleus and the nucleus isthmi, pars parvocellularis in the pigeon (*Columba livia*) and chick (*Gallus domesticus*): A retrograde labeling study. *J. Comp. Neurol.* 305:470–488; 1991.
44. Woodson, W.; Shimizu, T.; Wild, J. M.; Schimke, J.; Cox, K.; Karten, H. J. Centrifugal projections upon the retina: An anterograde tracing study in the pigeon (*Columba livia*). *J. Comp. Neurol.* 362:489–509; 1995.