Temporal Patterning of Song Production: Participation of Nucleus Uvaeformis of the Thalamus

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SUMMARY

Birdsong is a learned vocal behavior used in intraspecific communication. The motor pathway serving learned vocalizations includes the forebrain nuclei NIf, HVC, and RA; RA projects to midbrain and brain stem areas that control the temporal and acoustic features of song. Nucleus Uvaeformis of the thalamus (Uva) sends input to two of these forebrain nuclei (NIf and HVC) but has not been thought to be important for song production. We used three experimental approaches to reexamine Uva's function in adult male zebra finches. (1) Electrical stimulation applied to Uva activated HVC and the vocal motor pathway, including tracheosyringeal motor neurons that innervate the bird's vocal organ. (2) Bilateral lesions of Uva including the dorso-medial portion of the nucleus affected the normal temporal organization of song. (3) Chronic multiunit recordings from Uva during normal song and calls show bursts of premotor activity that lead the onset of some song components, and also larger bursts that mark the end of complete song motifs. These results implicate Uva in the production of learned vocalizations, and further suggest that Uva contributes more to the temporal structure than to the acoustic characteristics of song.

Keywords: **Uvaeformis, chronic recording, microstimulation, zebra finch, vocal.**

INTRODUCTION

Bird song is a learned behavior important for intraspecific communication. The basic organization of the song circuitry underlying this behavior has been studied extensively in both canaries and zebra finches (Nottebohm et al., 1976, 1982; Okuhata and Saito, 1987; Bottjer et al., 1988). It includes an efferent pathway that leads through the forebrain nuclei NIf, HVC, and RA to motor neurons that control the vocal musculature (Fig. 1). Lesion studies have shown that this pathway is necessary for song production (Nottebohm, Stokes, and Leonard, 1976; McCasland, 1987). A second branch of the song circuit leads indirectly from HVC to RA (via area X, DLM of the thalamus, and lateral MAN). Lesions of these three nuclei affect song only if the lesions are made during song development; similar lesions in adult zebra finches do not affect song production (Bottjer, Miesner, and Arnold, 1984; Sohrabji, Nordeen, and Nordeen, 1990; Scharff and Nottebohm, 1991).

Zebra finch song consists of a stereotyped sequence of modulated sounds, called *syllables,* and nuclei of the efferent pathway show activity during song production that precedes the production of each syllable in the sequence suggested by connectivity (McCasland, 1987). While the importance of the efferent pathway for song production is thus well established, the source (or sources) of timing signals that drive the efferent nuclei to initiate song and guide its production remains unknown.

Nucleus Uvaeformis of the thalamus (Uva) sends input to two forebrain nuclei in the efferent pathway, HVC and NIf, but heretofore has been thought not to be important for song production

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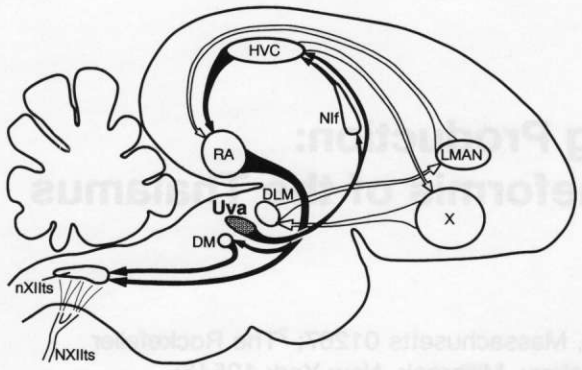

Figure 1 Connectivity of passerine song system nuclei. The brain is shown schematically in sagittal section. The nuclei of the efferent pathway are connected by black arrows, and other song circuit connections are shown with open arrows. DLM = nucleus dorso-lateralis thalamicus, pars medialis; DM of ICo = nucleus intercollicularis, pars dorso-medialis; HVC = (part of the neostriatum; formerly hyperstriatum ventralis pars caudalis); LMAN = nucleus magnocellularis anterior neostriatalis, pars lateralis; NIf = nucleus interfacialis neostriatalis; nXIIts = nucleus nervi tracheosyringealis; ts nerve = Nervus tracheosyringealis; RA = nucleus robustus archistriatalis; Uva = nucleus Uvaeformis thalami; X = area X.

(McCasland, 1987). We now find that (1) stimulation of Uva in zebra finches activates neurons in nuclei of the efferent pathway (including the motor neurons that innervate the vocal organ), (2) lesioning Uva affects song production, and (3) neural activity in Uva leads song production. Together, these results clearly implicate Uva in song production, and our lesion and recording data further suggest that Uva plays a greater role in coordinating the temporal structure than in producing the acoustic characteristics of song.

METHODS

Subjects

Male zebra finches were bred inhouse or obtained from commercial suppliers, and were caged singly or in pairs. Seven adult males were used in stimulation studies of Uva; four of these also received lesions. In addition to the adults, two pairs of juvenile brothers were also lesioned. Song development is completed at approximately 90 days in zebra finches (Immelmann, 1969). In each of the two pairs of brothers, one bird received a lesion during song development (50, 54 days). The brothers were sham-operated at the same age. Three adult males were implanted with chronic recording electrodes.

Surgery, Stimulation, and Lesion Placement

Birds were anesthetized with ketamine/xylazine (25 and 50 mg/kg, respectively), and placed in a stereotaxic device (Kopf) using a custom bill bar that held the upper mandible at 45° below horizontal. After an incision was made at the midline of the scalp, the junction of the cerebral hemispheres and the cerebellum was used to define the locations of apertures made in the skull over Uva and HVC using stereotaxic coordinates derived from the canary atlas (Stokes, Leonard, and Nottebohm, 1974) and experience (for Uva, 0.8 mm anterior, 1.7 mm lateral, and 4.5 mm deep). Glass-insulated tungsten electrodes (Asanuma, 1981) were used to pass stimulating current (single 0.3 ms, 5- to 50-μA bipolar pulses) in and near Uva. The same type of electrode was used to record extracellularly from multiple units within HVC. Recordings of activity in the tracheosyringeal (ts) nerves were made by dissecting them free of connective tissue and muscles along the trachea and suspending each nerve on a hook electrode in an oil pool. The placement of electrodes within HVC was confirmed by stimulating at the recording site and recording activity from the ts nerve (Paton and Manogue, 1982). Small electrolytic lesions (10 μA, 15 s) were placed along electrode tracks to help define locations where stimulation activated other song centers.

The procedure for placing larger lesions was identical to that outlined above, except that after the nucleus had been mapped, unilateral (one adult) or bilateral (three adults and two young birds) lesions were placed within Uva by passing cathodal current (25–50 μA, 1–2 min). The procedure for sham-operated birds was identical, except that current was not passed. The sham-operated birds served as a control, since preoperative song recordings could not be obtained when the lesions were placed during development. Pairs of brothers (lesioned and sham-operated) were housed in the same cage before and after surgery.

Song Recording and Analysis

Females were placed in or near a male's cage, and the male's courtship song was recorded using a dynamic microphone and a Tandberg reel-to-reel tape recorder. The bird was then prepared for surgery and the boundaries of Uva defined by mapping low-threshold stimulation sites for producing activity in the ts nerves. This map was used as a guide to place an electrolytic lesion (15–25 μA, 2–3 min) made with the same electrode used for stimulation. The lesioned birds' songs were then recorded weekly for up to 2 months postoperatively. Sonograms

of songs obtained before and after lesioning were produced with a Kay Sonagraph (300 Hz filter) and compared visually to match pre- and postoperative syllables. Changes in song structure were quantified by generating and comparing amplitude envelopes of five preoperative and five postoperative songs (Williams et al., 1992). A song was defined as beginning with a string of introductory notes (Sossinka and Böhner, 1980) and ending with a silence of at least 500 ms. For each condition, the five songs were drawn from two separate recording sessions. Amplitude envelopes were generated as 100-point moving averages in Signalyze (Infosignal, Neuchâtel, Switzerland); information about the order, spacing, relative amplitude, and amplitude modulation of song syllables, but not frequency information, are reflected in these amplitude envelopes. The five envelopes from each condition for each bird were then compared with Canary (Chris Clark, Cornell Laboratory of Ornithology, Ithaca, NY), a program that uses an algorithm to slide two waveforms over each other and generate a correlation coefficient for each possible comparison. We used the maximal correlation coefficients of the 15 comparisons calculated for each condition to assess relative song stereotypy.

Chronic Recording

Birds were anesthetized, prepared for surgery, and Uva was located as described above. Then a chronic electrode assembly, consisting of an insulated 22-gauge Pt-Ir wire glued to an insulated tungsten microelectrode and appropriate connectors, was lowered into the brain to target Uva; its position was confirmed by stimulating the tungsten electrode and recording the volley on the ts nerve. A silver ground lead was inserted between the skull and the dura, and the entire chronic assembly was attached to the skull with dental acrylic. The skin margins were sutured in a tight purse around the implant, topical antibiotics and local anesthetic were applied to the wound margins, and the bird was allowed to recover for 48 h.

For chronic recording, a miniature headstage connector was attached to the electrode assembly, and signals were led off through a flexible cable to a commutator on the roof of the bird's cage. Recording sessions lasted 1–2 h, during which time the bird could move about, eat, drink, and perch. The bird was induced to vocalize by playing back the songs and calls of other birds, and presenting a female in an adjacent cage. Vocalizations and multiunit recordings from the implanted electrodes were recorded with an instrumentation tape recorder (Racal) and later digitized on an 11/73 microcomputer (Digital Instrument Corporation). The multiunit recordings were half-wave rectified and integrated in 4-ms bins to allow for direct comparison with the amplitude waveform of each vocalization.

Histology

After recording was completed, birds used in stimulation, lesion, and chronic recording studies were perfused with 10% formol saline. The brains were removed, and sectioned at 50 μm on a vibratome. The sections were stained with cresyl violet and examined for electrode tracks and/or lesions. Sections were traced using an imaging system (Dage MTI/Data Translation). The volume of Uva, and the extent of lesions measured using a drafting program (Microstation), and the extent of the lesion was calculated as a percent of each subdivision (Williams, Ball, and Faris, 1989) as well as of the total volume of Uva.

RESULTS

Stimulation of Uva Activates the Efferent Song Pathway

Stimulation of Uva elicited activity, both ipsilaterally and contralaterally, in the ts nerve (Fig. 2). The latency of the activity elicited in the ipsilateral HVC by left Uva stimulation in the bird shown in Figure 2(a) was 7 ms, and the volley in the ipsilateral ts nerve began at 26 ms after Uva stimulation. Stimulation of the left HVC in the same bird elicited activity in the ts nerve with a 19 ms latency [Fig. 2(b)]; this matches the calculated transmission time between HVC and the ts nerve resulting from Uva stimulation. A similar sequential activation of components of the efferent pathway was seen in all birds tested, and is consistent with the hypothesis suggested by connectivity: that Uva stimulation activates neurons in HVC which, in turn, results in firing of hypoglossal motor neurons. Contralateral activation is presumably due to crossing pathways between the two Uvas, and will be addressed in a future paper.

We were able to elicit activity in HVC and the ts nerve with stimulus currents as low as 5 μA from locations throughout Uva as defined by Nissl staining. We also stimulated within the region called the *horn* of Uva, which surrounds Uva dorso-medially and is largest at the posterior end of the nucleus (Williams et al., 1989), and obtained comparable activation of the efferent song pathway [Fig. 2(c)]. Neurons in the horn of Uva are also labeled by tracer injections into HVC (Williams et al., 1989, and in preparation). However, these results cannot

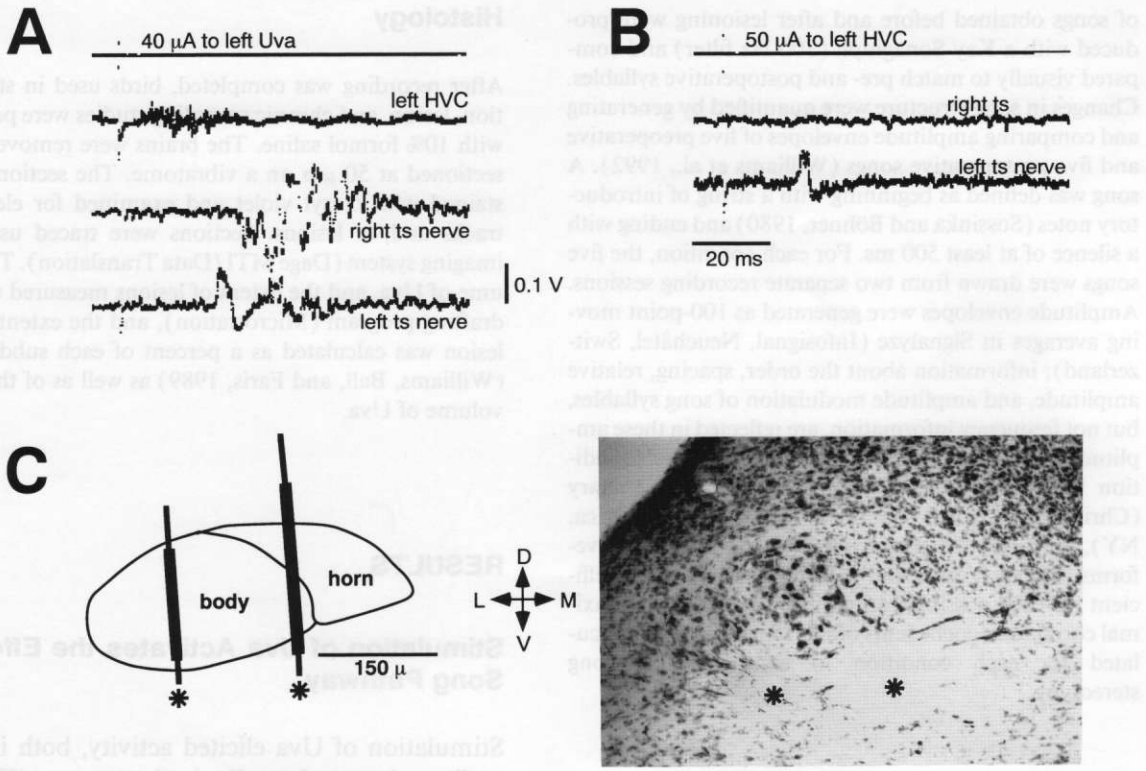

Figure 2 Stimulating Uva elicits activity in HVC and the ts nerve. (A) Activity recorded from the ipsilateral HVC and both ts nerves after stimulating the left Uva. (B) Stimulating the left HVC, through the same electrode used to record in (A), elicits activity only in the ipsilateral ts nerve, and the strength of the volley is decreased relative to that recorded after weaker Uva stimulation. This difference in strength was consistent across preparations and recording sites, and is presumably due to differences in recruitment; Uva is smaller than HVC, so stimuli of different strength would be required to activate the same numbers of neurons that ultimately project to the ts nerve. (C) Stimulation sites within a 150-μm anterior–posterior segment of Uva (shown in transverse section). A 25-μA stimulus pulse was effective in driving the ts nerve along the portion of electrode tracks defined by the black bars; the portion of the track where a 50-μA stimulus pulse (but not a 25-μA pulse) was effective in eliciting activity is shown with open bars. A micrograph of the corresponding section is shown at right. Asterisks indicate the center of marker lesions.

be taken as proof that neurons in the horn of Uva project to HVC: current spread may account for the ability to activate HVC from sites in the horn of Uva (although the dorsolateral extent of current spread within an electrode track suggests that this mechanism cannot account for the low thresholds observed within the horn of Uva), and it is possible that transynaptic spread of tracer from neurons in the body of Uva to neurons in the horn of Uva can account for retrograde tracer accumulation in the horn of Uva. Nevertheless, the results shown here and described more extensively elsewhere (Williams et al., in preparation) argue that the horn as well as the body of Uva should be considered when assessing the role of Uva.

Lesioning Uva Affects Song Production

The most striking effect of partial unilateral or bilateral Uva lesions was that the order of the syllables within the song was altered and the normal stereotypy of this order was impaired (Figs. 3, 4). Intact zebra finches sing courtship song in bouts that consist of a series of repeated introductory notes followed by several song motifs delivered in succession (Sossinka and Böhner, 1980). Since each song unit begins with the same syllable sequence and individual syllables are not repeated, song units are easy to define within the bout. Uva-lesioned birds often repeated one syllable or group of syllables several times before proceeding to the next and did

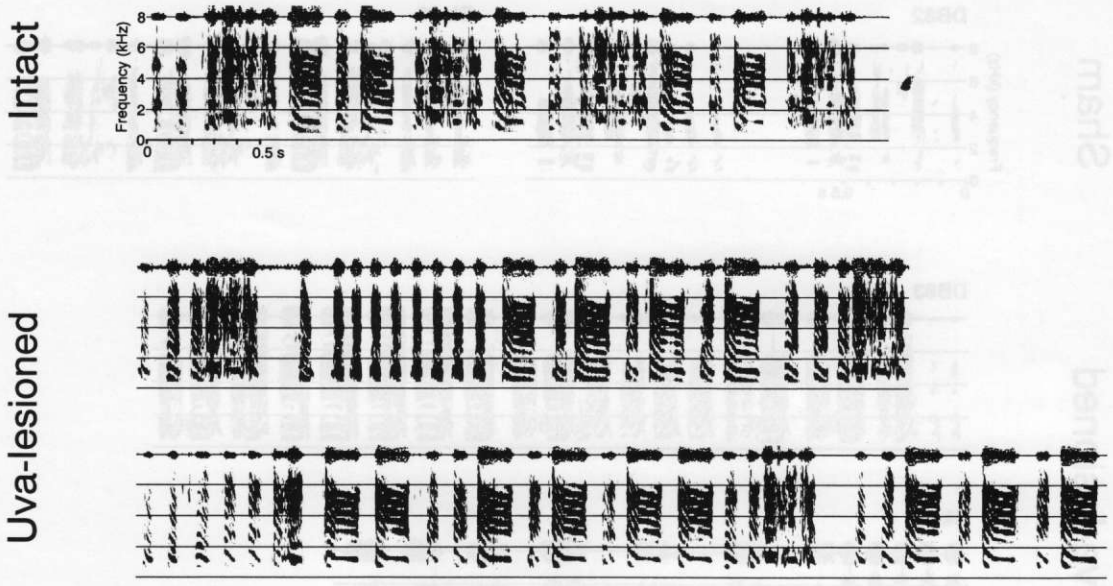

Figure 3 Songs of an adult male before and after Uva lesion. Sonograms of songs from a bird (W78) that received partial lesions of both Uvas as an adult (see Fig. 5). The preoperative song shows the characteristic features of zebra finch song: introductory notes followed by a repeated song motif. Two segments of song bouts with the characteristic highly abnormal temporal patterning seen after Uva lesion are also shown. The amplitude profile of each song is shown along the 8-kHz line.

not always initiate the song with the same syllables during each rendition, making it difficult to define a song unit. Some clusters of syllables remained intact in order within the cluster as well as in acoustic structure, but in such cases the order with which the subgroups of syllables were combined into song became variable (Fig. 3).

Loss of song syllables and/or deficits in song syllable phonology occurred in three of the four adult birds that received Uva lesions (seven of 29 or 24% of all syllables were lost). However, these syllable deficits and losses did not appear to be a direct consequence of the Uva lesion. All song syllables were recorded immediately after the lesion, and losses were noted in later recordings, and the phonology deficits in the remaining syllables disappeared by the 3- to 4-week postoperative recording. This timing is strongly reminiscent of the changes seen after ts nerve injury (Williams and McKibben, 1992), and the syllable phonology deficits and syllable loss after Uva lesion are probably best seen as a consequence of injury to the ts nerves incurred during the physiological recording from the nerves. In contrast, the deficits in temporal patterning of song persisted for as long as we followed the birds postoperatively (up to 2 months).

The syllables of birds that received partial Uva lesions before song development was completed had acoustic structure within the normal range, but again the syllables were delivered in a variable order, making it difficult to define a song unit (Fig. 4). The degree of deficit in song patterning was quantified by comparing the amplitude envelopes of songs in intact and Uva-lesioned birds. The songs of the two adults with lesions that most closely matched the juveniles' lesions, in size and placement, were analyzed and compared to the songs of the lesioned juveniles and their sham-operated brothers. All four birds showed a significant drop in song stereotypy when compared to controls (from an average correlation of 0.807 $\pm$ 0.012 for intact birds to an average correlation of 0.656 $\pm$ 0.011 for Uva lesioned birds; $t = 9.28$, df $= 320$, $p < 0.001$). There were no age-related differences in the effect of Uva lesion upon song structure: intact adults (average song correlation of 0.753 $\pm$ 0.014) had song stereotypy similar to birds that were sham-operated as juveniles (0.821 $\pm$ 0.016; $t = 1.60$, df $= 140$, $p > 0.1$), and Uva lesions resulted in a decrease in song stereotypy both for males that were lesioned as adults (0.653 $\pm$ 0.019) and those that were lesioned as juveniles (0.659 $\pm$ 0.013; there was no difference between the groups, $t = 0.26$, df $= 178$, $p > 0.7$).

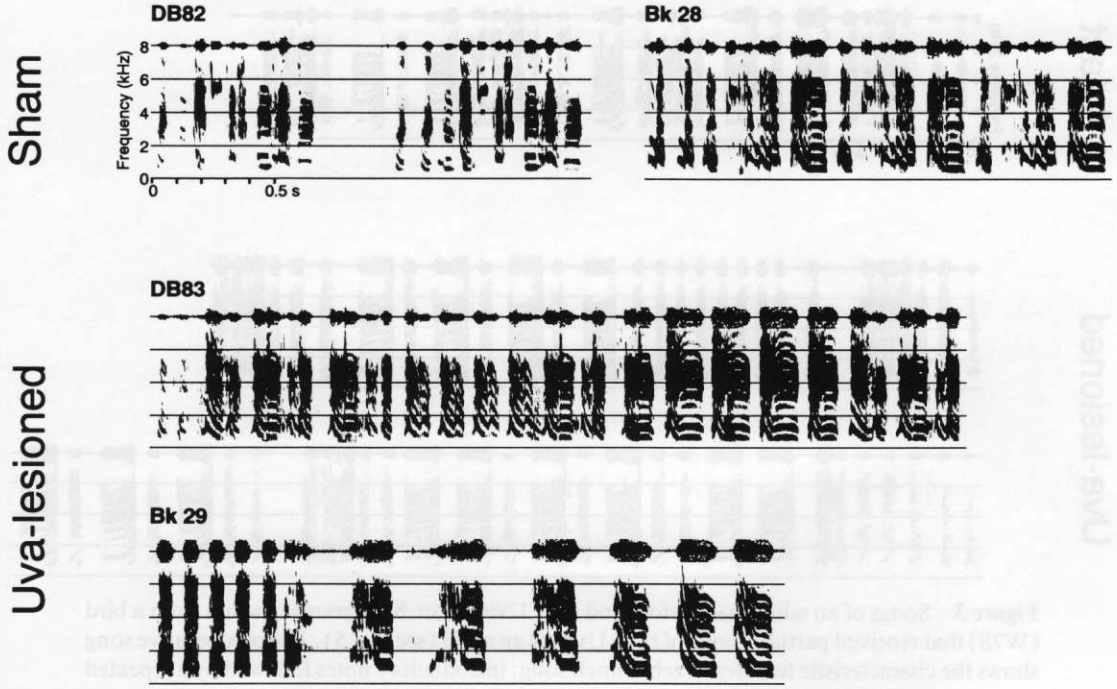

Figure 4 Uva lesions placed during development affect song. Partial bilateral lesions were placed in the Uvas of DB83 (at 54 days) and Bk29 (at 50 days) during song development. Their brothers (DB82 and Bk28, respectively), received sham lesions at the same age to serve as controls (at this age, it is not possible to reliably record preoperative song, which is developing). DB83 shows deficits similar to those of W78, the adult shown in Figure 3; although Bk29 delivers song syllables in a consistent order, the repeated syllables and long intersyllable intervals are abnormal. The amplitude profile of each song is shown along the 8-kHz line.

Although the sample size was small and variation in lesion size and placement was large, some general observations can be made. Lesions that affected song stereotypy varied in size from 2% to 34% of the entire volume, including the horn and body, of both Uvas (for examples of large and small lesions see Fig. 5). Two of the birds that had song structure deficits after surgery received lesions that affected less than 1% of the body of one Uva. In contrast, all effective lesions were bilateral and included at least 6% of the horn of each Uva. Some of the lesions that affected song structure impinged on areas outside Uva, including the posterior commissure which runs ventral to the nucleus [Fig. 5(a)], but lesions restricted completely to Uva and those that showed a small amount of additional tissue damage along the electrode track dorsal to the nucleus were equally effective [Fig. 5(b)].

Song-Related Activity in Uva

Multiunit activity accompanied song and call production in chronic recordings made from Uva in three birds. This activity consisted of premotor bursts that preceded calls and the introductory notes of song by 50–90 ms (Fig. 6). Bursts continued throughout the song, but could not be definitively associated with individual syllables due to the rapid syllable delivery rate in zebra finch song. An elevated rate of activity continued for 1–2 s after the end of the song. In addition, very large bursts, "superbursts," occurred that were precisely timed to the end of song (other large bursts could also occur within motifs but did not do so consistently). In one bird, a superburst occurred at the end of each song motif in the nine singing bouts recorded [see arrows in Fig. 6(a)], and in another it occurred reliably at the end of each song bout of the seven bouts recorded and at the end of some motifs [Fig. 6(b)]. Superbursts also occurred in the third bird, but the data were not sufficient to allow conclusions about timing.

While the activity preceding introductory notes is clearly premotor, the activity during song could have also reflected corollary signals or sensory activation, since Uva has been recently shown to receive auditory input (Okuhata and Nottebohm,

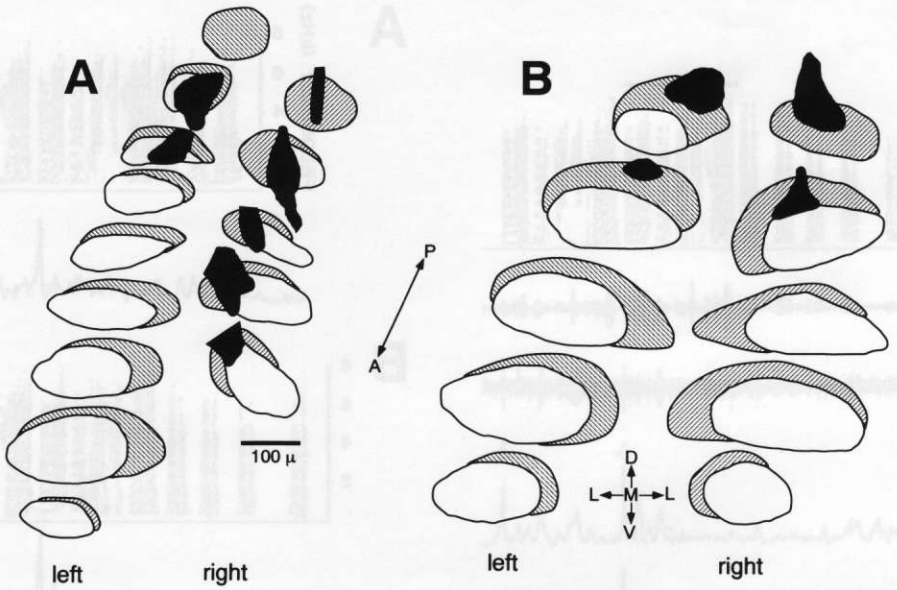

Figure 5 Reconstruction of lesions that affected song. The Uva lesions that affected the songs of W78 [as an adult (A)] and DB83 [as a juvenile (B)] are shown here in black, and the outline of the body of Uva (the region that includes large, round, darkly staining somata in Nissl stains) by a black line, and the horn of Uva (which lies around the dorso-medial portion of the body and is more extensive in posterior sections) by a hatched area. In neither bird were the lesions complete, and that in the Uva of DB83 was almost completely restricted to the horn of Uva on both sides. Although the two birds showed variation in anterior–posterior extent and cross-sectional area of Uva, this was not due to the effects of lesioning a juvenile; the Uva of DB83 is of normal size and extent, and the size of nuclei as mapped by microstimulation did not differ between the four juveniles and four adults used in this study.

1992). In particular, the superburst, reliably timed to the end of the final song syllable, might be due to auditory activation. However, we believe that the final syllable does not act as an auditory stimulus for the following reason. One bird terminated two of his song bouts early, without singing the final syllable (Fig. 7). The superburst occurred on those songs too, but was not timed to the end of the last syllable sung; rather, it occurred at its normal time measured with respect to the onset of the song motif.

Histological examination of the site of implantation of the chronic electrodes revealed an electrode track terminating in the region of Uva. However, as is typically the case for long-term implants, tissue disruption along the electrode track made it impossible to determine whether the electrode tip lay in the body or horn of Uva.

DISCUSSION

The thalamic song nucleus Uva satisfies three criteria used to define a brain region's involvement in a behavior: stimulation of the nucleus elicits activity in the motor system that controls song production, lesion of the nucleus affects the production of song, and Uva neurons show song-related activity.

We have considered the possibility that our data do not reflect a role for Uva, but rather for commissural fibers from DM of ICo that course ventral to Uva. However, several lines of evidence argue against the possibility that DM of ICo (which projects to the ts nucleus) can account for the results reported here. Stimulating Uva activated the HVCs and then the ts nerves sequentially, consistent with the known anatomical connections of Uva. Responses elicited in the ts nerve by Uva stimulation had longer latencies than did responses elicited by stimulating HVC, which is afferent to DM of ICo; had we been stimulating DM efferents, which project directly to nXIIts, we would have expected latencies shorter than those obtained after stimulating HVC, which is afferent to DM. In addition, some of the Uva lesions that affected song did not impinge upon the posterior commissure, where fibers connecting the two DMs of ICo travel,

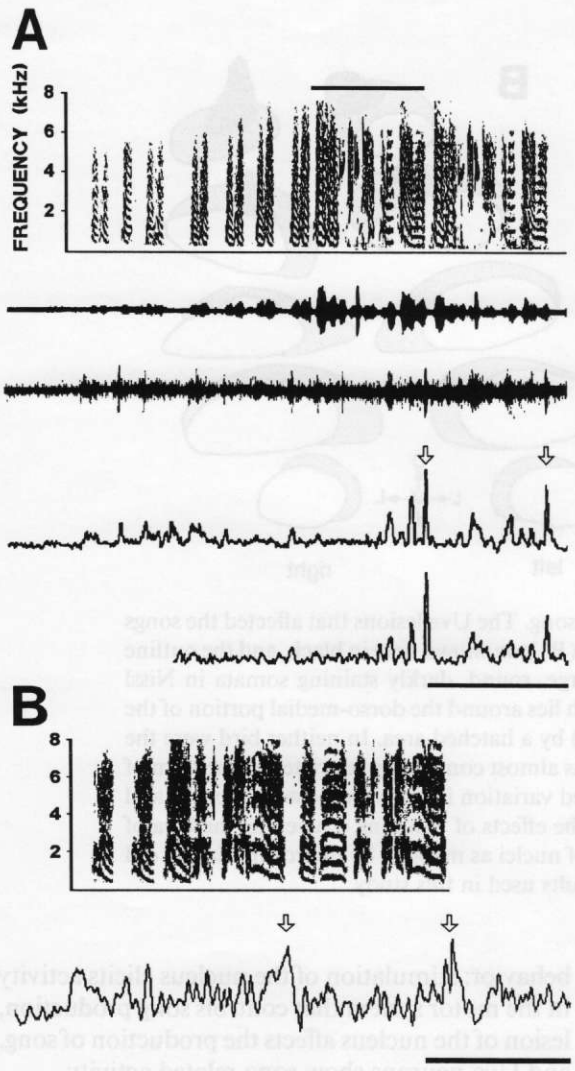

Figure 6 Activity in Uva associated with song production. (A) From top to bottom, traces show the spectrogram of a song bout, the amplitude waveform of the song produced, the raw multiunit recording, the rectified, integrated multiunit recording, and an ensemble average of recordings taken during five song bouts aligned on the end of the final syllable of the first song motif. Because the number and timing of the sequence of introductory notes that leads a song bout is variable, the trace showing the average begins immediately prior to the introductory note shared by all bouts included in the average. Open arrows indicate the occurrence of superbursts at the end of each motif (the first motif in the bout is indicated by the horizontal bar). Time bar = 1 s. (B) The spectrogram and rectified, integrated multiunit activity for a song bout in another bird. Time bar = 0.5 s.

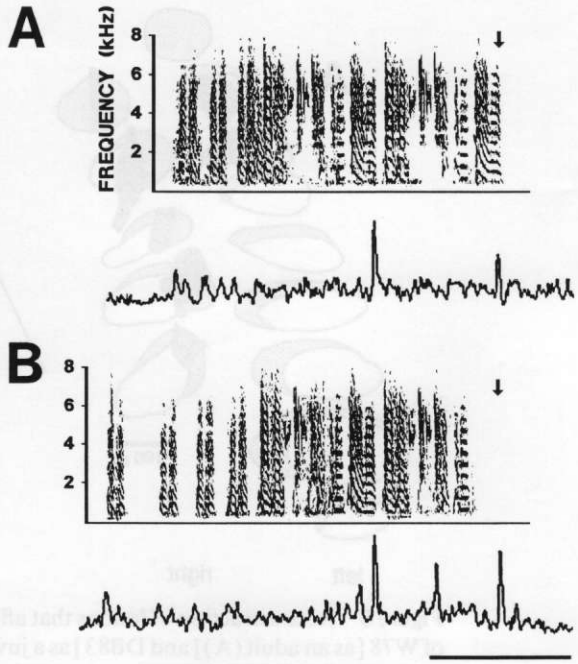

Figure 7 Timing of superburst activity in Uva. (A) Sonogram and rectified, integrated multiunit recording for a song bout which terminated on the final song syllable of the second motif (indicated by arrow). (B) Sonogram and rectified, integrated multiunit recording for a song bout which terminated on the penultimate song syllable of the second motif (timing of normal motif end shown by arrow). Time bar = 1 s.

and lesions to DM of ICo do not produce song deficits like those noted after Uva lesion (B. Simpson, personal communication).

McCasland (1983 and 1987) reported that Uva lesions did not affect song. Our results and his can be reconciled by a careful definition of the boundaries of Uva. The extent of Uva as defined by a group of large, round, darkly staining somata in Nissl material is incomplete (Williams et al., 1989); the nucleus was originally defined as the neurons that project to HVC, and the Nissl-defined Uva is a ventro-lateral subset of both (1) the neurons that are filled by injections of retrograde tracer into HVC and (2) the region for which microstimulation is effective in driving the ts nerve. Lesions of Uva may need to include portions of the dorso-medial division or "horn" of Uva if they are to affect song production.

Our lesion data suggest that Uva's role in song production may be related specifically to the timing and ordering of song syllables and motifs. Songs of birds that received Uva lesions as juve-

niles had syllable phonology within the normal range, and birds that received lesions as adults did not have permanently impaired syllable morphology. However, the normal pattern of syllable delivery was not maintained after Uva lesion; birds repeated some syllables and groups of syllables, varied the order of syllables within the song, and failed to start the song with a constant initial syllable or syllables. Lesions of Uva also affected song in adults that had crystallized their songs. The information provided by Uva appears to be important for both song development and song production after learning has been completed; our data do not allow us to determine whether Uva's role is constant or whether it differs during song learning.

Multiunit activity in Uva increased prior to and during song production. The premotor lead of activity in Uva relative to the onset of sound production was similar to or longer than the lead reported elsewhere for Nif and HVC in zebra finches (McCasland, 1983, Fig. 18; 1987, Fig. 6). Although it is always difficult to assess the relative timing between structures, especially when experimental conditions are different, this timing suggests that Uva may contribute to the initiation and/or control of vocal production. In addition to the activity in Uva that leads song syllables, the termination of song was reliably accompanied by "superbursts" in Uva. Interestingly, in one case the superbursts were timed relative to the beginning of the song motif, and occurred at the same point whether or not all of the song syllables were delivered. This pattern of activity within Uva is consistent with a role in controlling the timing of song motifs.

We suggest that Uva may provide or integrate timing signals that define the train of song motifs within a bout; such timing information may also define the rhythm and sequence of song syllables. In this model, the acoustic characteristics of individual syllables are shaped by the action of the centers downstream from Uva (such as the forebrain nuclei NIf, HVC, and RA). Although its afferents are not well described, we do know that Uva, unlike other forebrain song nuclei, projects contralaterally (Nottebohm et al., 1982; Williams, 1985), placing it in a unique position to serve the coordinating role between the two hemispheres which must be an essential part of song production (Nottebohm, 1971; Nottebohm and Nottebohm, 1976; Suthers, 1990, 1992). Uva's placement at the beginning of the known chain of efferent pathway nuclei as well as the presence of activity recorded from Uva during song production and the motor activation that follows Uva stimulation make Uva plausible as an initiator and coordinator of control signals for song production.

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REFERENCES

ASANUMA, H. (1981). Microstimulation technique. In: *Electrical Stimulation Research Techniques.* M. M. Patterson and R. P. Kesner, eds. Academic Press, New York, pp. 61–70.

BOTTJER, S. W., HALSEMA, K. A., BROWN, S. A., and MIESNER, E. A. (1988). Axonal connections of a forebrain nucleus involved with vocal learning in zebra finches. *J. Comp. Neurol.* **279**:312–326.

BOTTJER, S. W., MIESNER, E. A., and ARNOLD, A. P. (1984). Forebrain lesions disrupt development but not maintenance of song in passerine birds. *Science* **224**:901–903.

IMMELMANN, K. (1969). Song development in the zebra finch and other estrildid finches. In: *Bird Vocalizations.* R. A. Hinde, ed. Cambridge University Press, Cambridge, pp. 61–74.

MCCASLAND, J. S. (1983). Neuronal control of song production. Ph.D. Dissertation. California Institute of Technology, Pasadena, California.

MCCASLAND, J. S. (1987). Neuronal control of bird song production. *J. Neurosci.* **7**:23–39.

NOTTEBOHM, F. (1971). Neural lateralization of vocal control in a passerine bird. I. Song. *J. Exp. Zool.* **177**:229–262.

NOTTEBOHM, F., KELLEY, D. B., and PATON, J. A. (1982). Connections of vocal control nuclei in the canary telencephalon. *J. Comp. Neurol.* **207**:344–357.

NOTTEBOHM, F. and NOTTEBOHM, M. (1976). Left hypoglossal dominance in the control of canary and white-crowned sparrow song. *J. Comp. Physiol.* [*A*] **108**:171–192.

NOTTEBOHM, F., STOKES, T. M., and LEONARD, C. M. (1976). Central control of song in the canary, *Serinus canarius. J. Comp. Neurol.* **165**:457–486.

OKUHATA, S. and NOTTEBOHM, F. (1992). Nucleus Uva might be part of a feedback circuit for song processing. *Soc. Neurosci. Abstr.* **18**:527.

OKUHATA, S. and SAITO, N. (1987). Synaptic connections of thalamo-cerebral vocal nuclei of the canary. *Br. Res. Bull.* **18**:35–44.

PATON, J. A. and MANOGUE, K. R. (1982). Bilateral interactions within the vocal control pathway of birds: two evolutionary alternatives. *J. Comp. Neurol.* **212**:329–335.

SCHARFF, C. and NOTTEBOHM, F. (1991). Behavioral deficits following lesions of various pars of the zebra

finch song system: implications for vocal learning. *J. Neurosci.* **11**:2896–2913.

SOHRABJI, F., NORDEEN, E. J., and NORDEEN, K. W. (1990). Selective impairment of song learning following lesions of a forebrain nucleus in the juvenile zebra finch. *Behav. Neural. Biol.* **53**:51–63.

SOSSINKA, R. and BÖHNER, J. (1980). Song types in the zebra finch (*Poephila guttata castanotis*). *Z. Tierpsychol.* **53**:123–132.

STOKES, T. M., LEONARD, C. M., and NOTTEBOHM, F. (1974). The telencephalon, diencephalon, and mesencephalon of the canary, *Serinus canaria,* in stereotaxic coordinates. *J. Comp. Neurol.* **156**:337–374.

SUTHERS, R. A. (1990). Contributions to birdsong from the left and right sides of the intact syrinx. *Nature* **347**:473–477.

SUTHERS, R. A. (1992). Lateralization of song production and respiration in canaries. *Proc. 3rd Int. Cong. Neuroethol.*, 294.

WILLIAMS, H. (1985). Interhemispheric coordination of bird song. *Soc. Neurosci. Abstr.* **11**:871.

WILLIAMS, H., BALL, G. F., and FARIS, P. L. (1989). Evidence for subdivisions within Uvaeformis, an avian song nucleus. *Soc. Neurosci. Abstr.* **15**:618.

WILLIAMS, H., CRANE, L., HALE, T. K., ESPOSITO, M. A., and NOTTEBOHM, F. (1992). Right-side dominance for song control in the zebra finch. *J. Neurobiol.* **23**:1006–1020.

WILLIAMS, H. and MCKIBBEN, J. R. (1992). Changes in stereotyped central motor patterns controlling vocalization are induced by peripheral nerve injury. *Behav. Neural. Biol.* **57**:67–78.