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Differences between ipsilaterally and contralaterally evoked potentials in the visual wulst of the zebra finch

MANFRED BREDENKÖTTER AND HANS-JOACHIM BISCHOF
Lehrstuhl für Verhaltensphysiologie, Universität Bielefeld, Federal Republic of Germany

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Abstract

The telencephalic target of the thalamofugal visual pathway in birds, the visual wulst, is part of the hyperstriatum accessorium/dorsale in the bird's brain. In this study, we tried to determine the exact location of the visually responsive area in the zebra finch by recording visually evoked potentials (VEPs) from different sites throughout the hyperstriatum and calculating current source densities (CSDs). In addition, we examined the influence of ipsilateral and contralateral stimuli on stimulus processing within this area, and tried to get insight into the neuronal machinery of the thalamofugal pathway by application of drugs such as tetrodotoxin (TTX) and picrotoxin.

About two-thirds of the hyperstriatum is responsive to contralateral stimuli but only a small portion responds to ipsilateral stimuli. Contralateral visual information arrives in the hyperstriatum dorsale (HD) and is processed further to the hyperstriatum accessorium (HA).

The small influence of ipsilaterally evoked potentials is not due to inhibition by the activity of the contralateral eye, as could be demonstrated previously for the ectostriatum. Instead, our results show that ipsilaterally evoked potentials are inhibited at least in part by a projection from the contralateral visual wulst.

Keywords: Birds, Visual wulst, Visually evoked responses, Tetrodotoxin, Picrotoxin, Current source-density analysis

Introduction

On the basis of experiments in barn owls, Pettigrew and Konishi (1976) reported that the telencephalic target of the thalamofugal visual pathway of birds, the visual wulst (so-called because it forms an elevation on the forebrain), processes visual information in a very similar way to the visual cortex of mammals. Most of the neurons of this area were binocularly driven and the visual space was topographically represented in the wulst area. On the other hand, studies on pigeons (Parker & Delius, 1972) and chicks (Wilson, 1980; Denton, 1981) demonstrated that in these species the visual wulst processes mainly information from the contralateral eye. Binocular neurons or responses to ipsilateral stimuli were seldom detected.

On the basis of the studies of Stingelin (1958), who compared the architecture of the forebrain in 51 bird species, Henke (1983) supposed that this difference in function of the visual wulst may be due to the amount of binocular overlap of a given bird species. In owls, which have a large binocular overlap, the wulst is highly differentiated into several distinct layers. Pigeons or chicks, which have a much smaller binocular overlap, possess a visual wulst that is differentiated in only three distinct layers, the hyperstriatum accessorium (HA), the hyperstriatum intercalatus superior (HIS), and the hyperstriatum dorsale (HD). Zebra finches, which show a binocular overlap comparable to the pigeon, but are worse than pigeons in binocular performance (Bischof, 1988), lack the intermediate layer HIS.

In contrast to this difference in binocularity and architecture of the visual wulst, recrossing fibers from the thalamic station of the thalamofugal pathway, the nucleus opticus principalis thalami (OPT), to the visual wulst have been demonstrated in owls (Karten et al., 1973) as well as in pigeons (Perisic et al., 1971; Hunt & Webster, 1972; Mihailovic et al., 1974) and in zebra finches (Nixdorf & Bischof, 1982). These projections may be capable of mediating ipsilateral responses to the visual wulst. Therefore, a discrepancy exists between physiological and anatomical findings concerning the processing of ipsilateral stimuli in laterally eyed birds: although the recrossing projection exists, responses to ipsilateral stimuli are weak or almost absent.

Besides examining the exact location of the visually responsive areas of the hyperstriatum, we wanted to evaluate whether the discrepancy between physiological and anatomical findings also exists in zebra finches. Since the visual wulst of zebra finches responds only weakly to ipsilateral stimuli, we attempted to find the first hints towards an explanation for this discrepancy.

Reprint requests to: Hans-Joachim Bischof, Lehrstuhl für Verhaltensphysiologie, Universität Bielefeld, Postfach 8640, D-4800 Bielefeld 1, Federal Republic of Germany.

Material and methods

Forty fully mature zebra finches (*Taeniopygia guttata castanotis* Gould) of either sex from the institute's stock were subjects of this study, weighing between 10–12 g and between 150–250 days old. The birds were anesthetized with 0.1 ml urethane (20% w/v), producing an anesthesia deep enough to prevent reactions to painful stimuli. The birds were placed in a stereotaxic headholder (Bischof, 1981), the skull was opened, and the dura removed. In most preparations, no brain movements were detectable. Glass micropipettes (outer tip diameter 10 μm, 10–20 MΩ) filled with 0.5 M sodium citrate were used in all experiments. As the pia mater is very fragile in zebra finches, it was not necessary to dissect it before the electrode was lowered slowly into the brain by a stepping motor attached to the z-axis of the micromanipulator (1-μm steps, stepping rate 2 μm/s). No deflection of the brain surface could be observed when the pia was penetrated. After reaching the first recording position at 100-μm depth, the brain surface was covered with warm mineral oil to prevent drying and 30 min were allowed for tissue stabilization.

Abbreviations

HA	hyperstriatum accessorium
HD	hyperstriatum dorsale
HV	hyperstriatum ventrale
LFM	lamina frontalis mediale
LFS	lamina frontalis superior
LH	lamina hyperstriatica
N	neostriatum
TFM	tractus frontomesencephalicus
V	ventricle
X	area X

Electrophysiological activity was recorded with standard electrophysiological methods. Very low frequencies were eliminated by a high-pass filter with a cutoff frequency of 4 Hz. Visual stimuli (flashes, duration 1 ms) were produced by a stroboscope and led to the eyes by fiber optics. With electronic shutters, it was possible to direct the stimulus to either eye or to stimulate both eyes simultaneously. Control recordings were made at irregular intervals by closing both shutters or removing the fiber optics from the eyes.

For all visually evoked potential (VEP) plots shown in this study, 64 signals were recorded and averaged by a signal averager (Nicolet Instruments, Frankfurt, FRG). Bin width was 500 μs, total recording time 500 ms, and the interstimulus time interval was 5 s. This interval was sufficient to minimize habituation effects.

The averaged signals were transferred to an HP 86B microcomputer. Amplitudes and latencies were estimated by a maximum–minimum routine of the computer. Additional information about latencies and amplitudes was evaluated by processing the evoked-potential plots on a graphics tablet.

For the estimation of the visually responsive area of the wulst, electrode penetrations were made at randomized coordinates in a 2 × 3-mm grid covering the rostral part of the telencephalon. In each bird, only part of the coordinates of the grids could be recorded (between 1 and 8 penetrations). Spacing between the tracks was 500 μm (Figs. 1 and 2). In the central part of the visually responsive area, spacing between the penetrations was reduced to 250 μm. For the three-dimensional current-source-density (CSD) analysis, spacing between the penetrations was 100 μm. Measurements were made every 100 μm along the electrode track, starting at 100-μm depth after penetration of the pia mater.

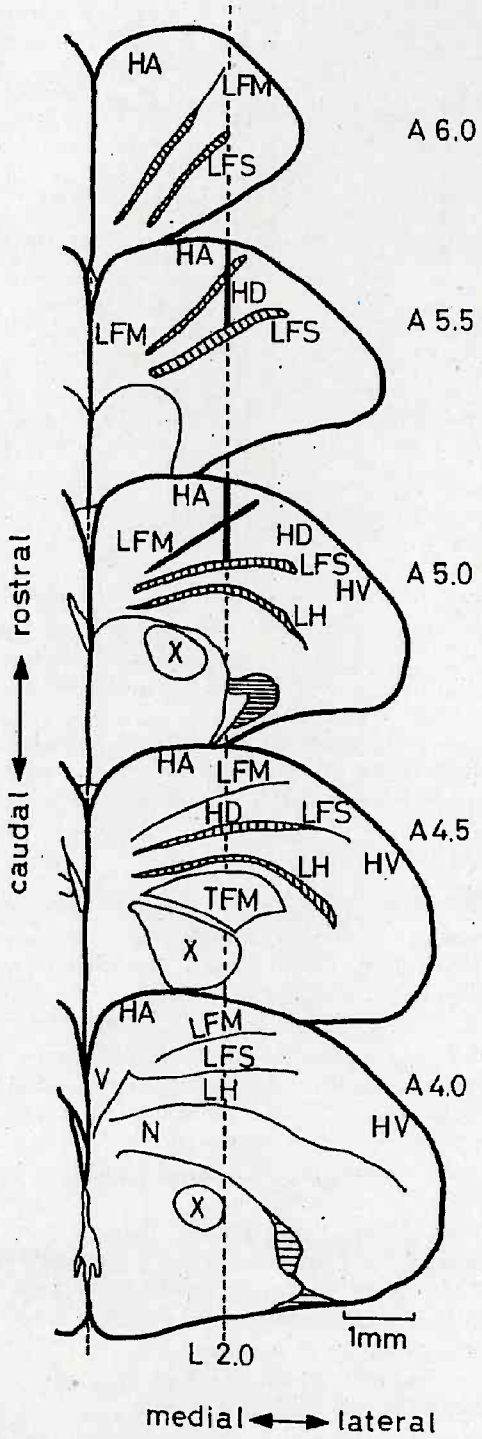

Fig. 1. Coronal sections of the zebra finch forebrain. Dark bars represent electrode tracks at the center of the visually responsive area (see Fig. 2). For terminology, see Abbreviations.

The coordinates for the electrode penetrations were derived from a stereotaxic atlas of the zebra finch brain (Nixdorf & Bischof, unpublished). Although tissue damage cannot be excluded (see below), we did not find any distortions of the

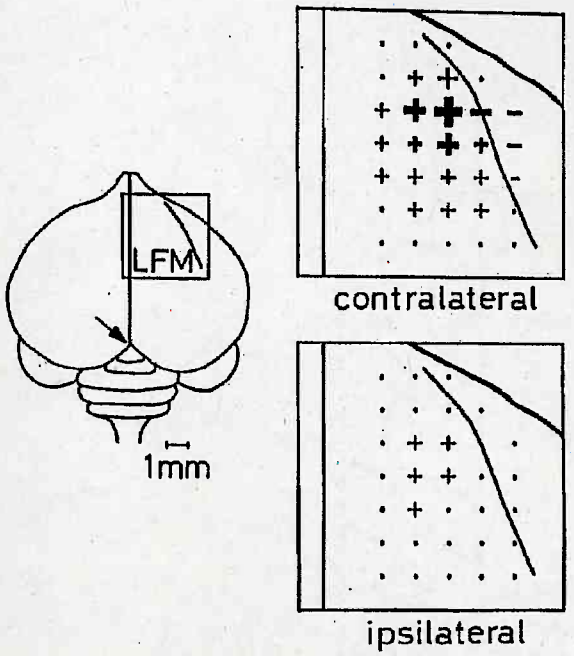

Fig. 2. Visual responsive area of the wulst. "+" signs show where the main response in 100-μm depth is positive, "–" signs depict a negative response. The size of the signs reflects the magnitude of the responses. A point means no response. The arrow indicates the stereotaxic reference point (Anterior 0.0, Lateral 0.0). LFM: lamina frontalis mediale.

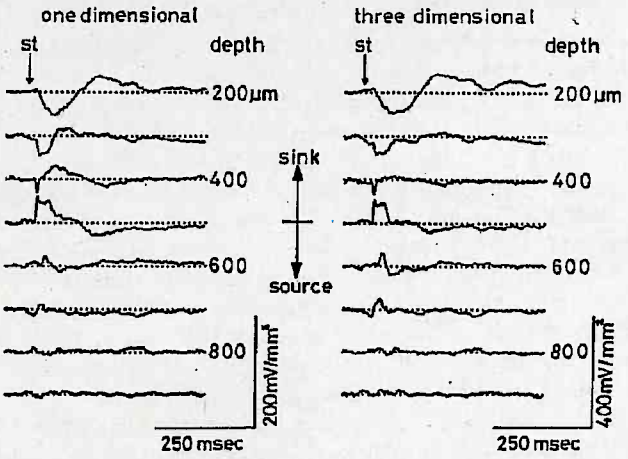

Fig. 3. Comparison of one- and three-dimensional current-source-density (CSD) analysis. The similarity of both graphs demonstrates that a one-dimensional CSD analysis is applicable to recordings from the visual wulst. Note the different scalings of amplitudes; positive deflections upwards; st: stimulus.

evoked potentials even in multiple penetrations with 100-μm spacing. This was confirmed by careful comparison of the responses of these multiple recordings with those of single penetrations at the same coordinates.

Spacing of penetrations and lack of horizontal deflection was controlled in some cases by examination of cresyl-violet-stained coronal sections of the fixed brain. The depth could be estimated physiologically, as a characteristic response occurred each time when the electrode reached the lamina frontalis mediale (LFM); namely, a very high frequency of spikes that could be detected with altered filter settings.

0.3 μl tetrodotoxin (TTX 0.25 mM in distilled water), which selectively blocks the sodium channels and therefore prevents the production of spikes (Reiter et al., 1986), was injected into one eye in order to investigate the influence of the other eye on ipsilaterally evoked responses. In other experiments, TTX was applied around the point of electrode penetration using small pieces of paper infiltrated with the substance.

The influence of intrinsic and extrinsic inhibition on VEPs of the visual wulst was examined by applying drops of picrotoxin (0.5 μl, saturated solution in 0.165 M NaCl), which blocks inhibitory synapses (Galindo, 1969; Bisti et al., 1971), directly to the wulst ipsilateral or contralateral to the recording site after removing the mineral oil with small pieces of filter paper. The oil remaining at the border of the preparation area prevented the solution from running off the site of application.

Further information concerning the location of the current sources and sinks within the visual wulst were obtained by calculation of one-dimensional current-source-density (CSD) profiles. The application of a one-dimensional CSD analysis instead of the three-dimensional analyses deserves translational symmetry in two dimensions in the neural tissue it was recorded from. Applicability of the one-dimensional analysis was tested by comparing the results of a three-dimensional CSD calculation with those of a one-dimensional calculation for identical coordinates in two birds (Fig. 3). Calculations were made according to Mitzdorf and Singer (1977) and Engelage and Bischof (1989) with a differentiation grid of 100 μm.

Results

Visually responsive area and response characteristics

The hyperstriatum of zebra finches, which includes the visual wulst, is composed of several different layers (Fig. 1). The most dorsal layer is the HA which is separated from the underlying HD by the lamina frontalis medialis (LFM). Figure 1 shows different coronal sections of the zebra finch forebrain. The dark bars represent the positions of the electrode tracks that gave rise to the largest responses with shortest latencies. Figure 2 shows the points of penetration (spacing 500 μm) in a dorsal view. Whereas medial tracks first penetrate the HA, pass the LFM, and then enter the HD, rostro-lateral tracks (e.g. plane Anterior 4.5 – 6.0, Lateral 3.0; compare Figs. 1 and 2) enter the HD directly. This is reflected by the potentials we obtained 100 μm below the brain surface in each track.

In Fig. 2, the optimal responses that we obtained by contralateral or ipsilateral stimulation at each location are depicted by symbols. Each symbol represents one electrode track. A point means that at this site no clear response to visual stimulation could be obtained in the entire track. A "plus" means that the main component of the potential was positive in a depth of 100 μm below the brain surface, and a "minus" that it was negative. Negative potentials occur only at regions outside HA, limited by the LFM which reaches the surface at the indicated position. The brain portion lateral to the LFM surface line belongs to the HD. The amplitudes of the evoked potentials were larger in the center than in the periphery of the visually responsive area, as indicated by the size of the signs in Fig. 2. A comparison of the two plots in Fig. 2 demonstrates that responses to ipsilateral stimuli, which were recorded alternating with the

contralateral ones during the same session, are limited to a much smaller area.

The changes of potentials over depth are depicted in Fig. 4a. It shows typical responses to contralateral stimulation, recorded at the center of the visually responsive area (Anterior 5.5, Lateral 2.0), together with the CSD analyses (right column). Directly after penetration of the brain surface, a strong positive response occurs (latency of first deflection 20 ms, 60 ms main peak). In deeper layers, sometimes detectable only as a shoulder of the large peak, an early peak occurs with a latency of 33 ms. This potential becomes smaller when the electrode proceeds to deeper layers, and is reversed between 400 and 500 μm,

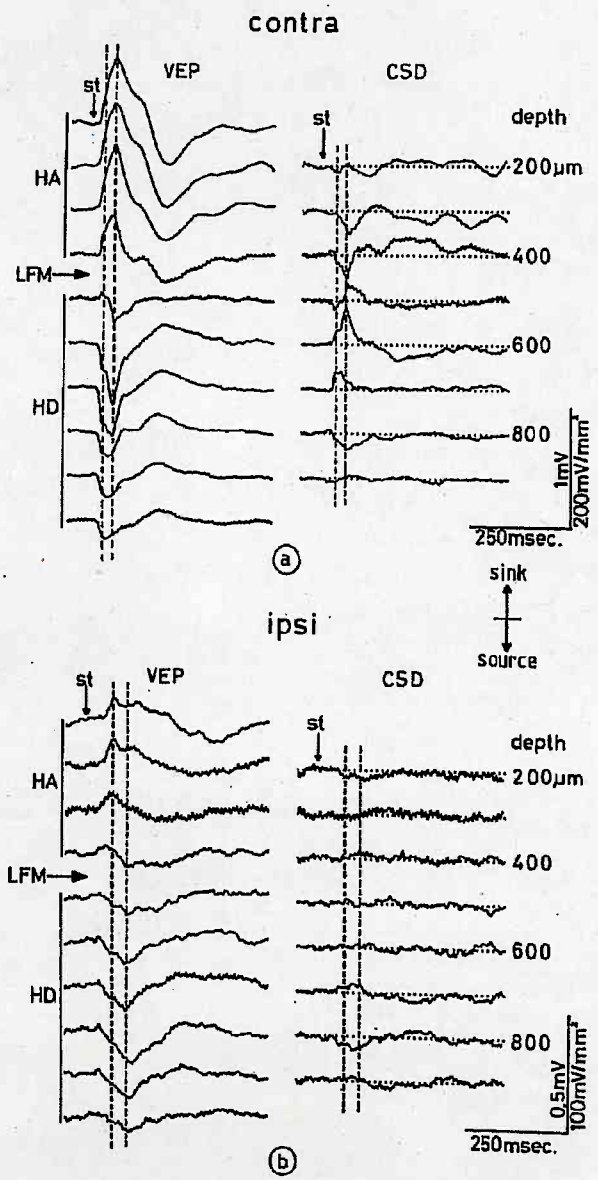

Fig. 4. Example of electrode tracks in the center of the visually responsive area: visually evoked potentials (VEP) and current-source-density (CSD) calculation. The ipsilateral track is the most reliable one of all our recordings. Contra, ipsi: stimulated eye; st: stimulus; positive deflections upwards.

where the LFM separates the HA from the HD. This reversed potential increases in amplitude to the central part of HD and again becomes smaller when the electrode proceeds to deeper regions.

The CSD analysis of contralaterally evoked potentials shows that the origin of the main component of the evoked potential is located in the HD, where a sink with a short latency (33 ms) can be detected in 600- and 700-μm depth, which has corresponding sources at about 500- and 800-μm depth. A second sink with a latency about 60 ms can be detected within HD at 600 μm. This distribution of sinks and sources suggests that the input from the OPT reaches the wulst in ventral layers of the hyperstriatum. From HD, the information may be transmitted to upper layers of the visual wulst, as indicated by the late sink at 300 μm (latency of about 130 ms). Sources corresponding to this late sink appear at 200- and 400-μm depth.

Ipsilaterally evoked responses are much smaller and more irregular than contralateral ones. In contrast to the contralateral ones, they cannot be obtained in each experiment and show irregular changes in amplitude. This means that they often can be detected against the background only in the averages, which are of smaller amplitude and are not as clear-cut as the contralateral ones (Fig. 4b). Figure 4 depicts one of the best recordings with ipsilateral stimulation. Again, positive potentials are obtained directly after penetrating the brain surface. In general, ipsilateral responses are characterized by a smaller positive deflection (amplitudes up to 0.1 mV), with latencies of about 60 ms. At least in upper layers, the potential has a second, not very sharp, peak with a latency of about 120 ms. As with the contralateral VEPs, the ipsilateral responses reverse at the depth of the LFM. The peak latency of the resulting negative wave is 100–120 ms and amplitudes between 0.1–0.13 mV. This means that only the late component of the positive wave is reversed, whereas the early component is not detectable at each depth.

CSDs of ipsilaterally evoked potentials are not described here, as they were not very clear-cut due to the irregularity of the VEPs. The prominent sink at 700 μm shown in Fig. 4b has only one corresponding source at 800 μm, the upper source is lacking. Most probably, this CSD profile is an artificial result of the quite large response within HD.

Tetrodotoxin injection into the contralateral eye

Although anatomical studies (Nixdorf & Bischof, 1982) show that the visual wulst of zebra finches receives input from both eyes, our results show that the influence of the ipsilateral eye is rather low. Therefore, it is conceivable that processing of ipsilateral information is inhibited somewhere in the thalamofugal pathway. In the ectostriatum, ipsilateral responses are inhibited by spontaneous activity of the contralateral eye (Engelage & Bischof, 1988). To test this mechanism for the visual wulst, we injected TTX into the eye contralateral to the recording side.

As can be seen from the evoked responses in Fig. 5, recorded from the central area of the wulst (see Fig. 2) at 100-μm depth, there is a weak inhibitory influence of this treatment on the ipsilaterally evoked potential (compare Fig. 5b and 5d). The response from the contralateral, TTX-injected eye is totally abolished (compare Fig. 5a and 5c).

In contrast, recordings from the ectostriatum contralateral to the injected eye during the same experiment (simultaneous recording from both areas) show that in this brain area the ip-

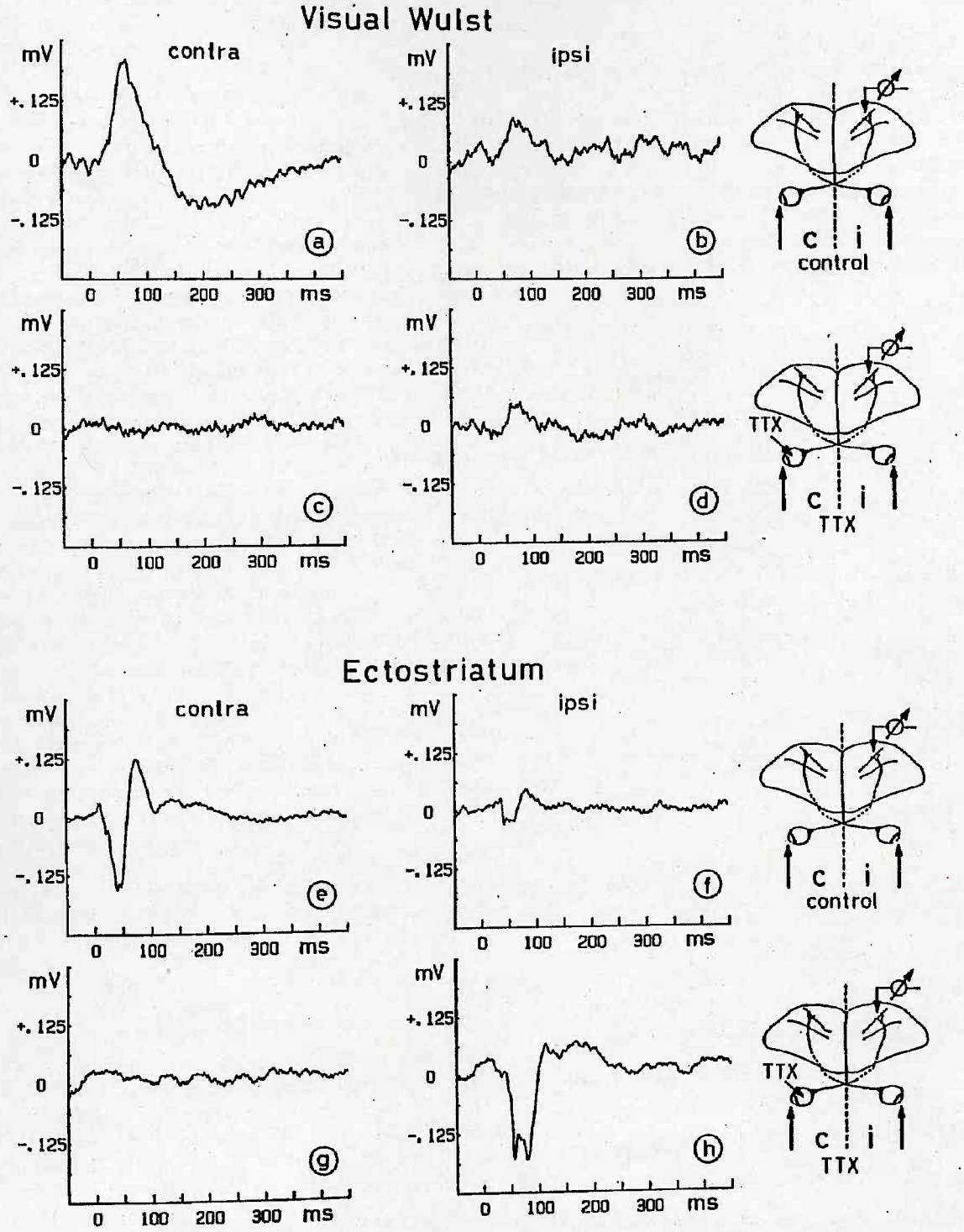

Fig. 5. Tetrodotoxin (TTX) injection into the eye contralateral to the recording site (see insets). The ipsilateral response of the visual wulst is unaffected, whereas the ipsilateral response of the ectostriatum is drastically enhanced. Contra (c), ipsi (i): stimulated eye; stimulus at 0 ms.

silateral response is drastically enhanced (compare Fig. 5f and 5h), as has been shown earlier with enucleation of the contralateral eye (Engelage & Bischof, 1988). As in the visual wulst, the contralateral response of the ectostriatum is totally abolished by TTX injection into the eye (Fig. 5e and 5g).

Tetrodotoxin application to the recording site

Application of TTX to the visual wulst, from which we recorded, results in a depression of the contralateral response (compare Fig. 6a and 6d). In contrast, the ipsilateral response

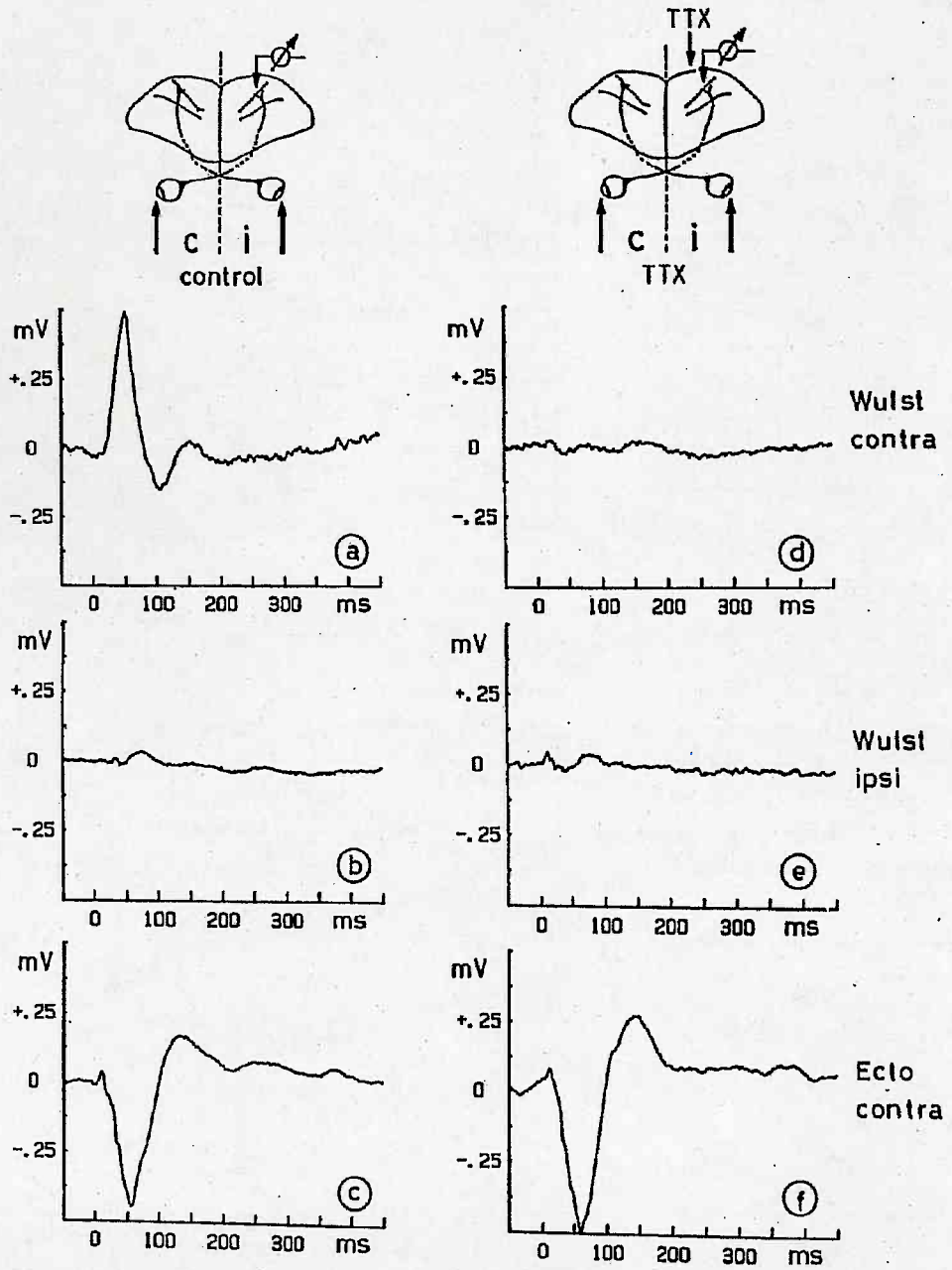

Fig. 6. TTX application to the recording site (see insets). The results are similar to those after injecting TTX in contralateral eye (compare Fig. 5). The recording from the ectostriatum shows that the effect of TTX is local. Contra (c), ipsi (i): stimulated eye; stimulus at 0 ms.

is altered but not suppressed by the TTX treatment (compare Fig. 6b and 6e). Simultaneous recordings from the ectostriatum (Fig. 6c and 6f) of the same side show that the effect of TTX application is local, as no reduction of the evoked potential could be observed here.

Picrotoxin application to the recording site

Application of picrotoxin to the recording site reveals large alterations with both kinds of stimulation. In the contralaterally evoked potentials, a new negative peak appears at the border HA/HD. The latency of this peak is 60 ms, with amplitudes between 2–3 mV (compare Fig. 7a and 7c). Obviously, this negative peak masks the second positive peak that can be detected in normal birds.

The CSD analysis of these contralaterally evoked potentials demonstrates that the distribution of sinks and sources is not essentially altered compared to control birds, in spite of the appearance of the new negative component of the VEP. The amplitudes are much larger (see the difference amplitude scales in Fig. 7a and 7c), and the late component at 300-μm (Fig. 7a) is concealed by the enlargement of the earlier components

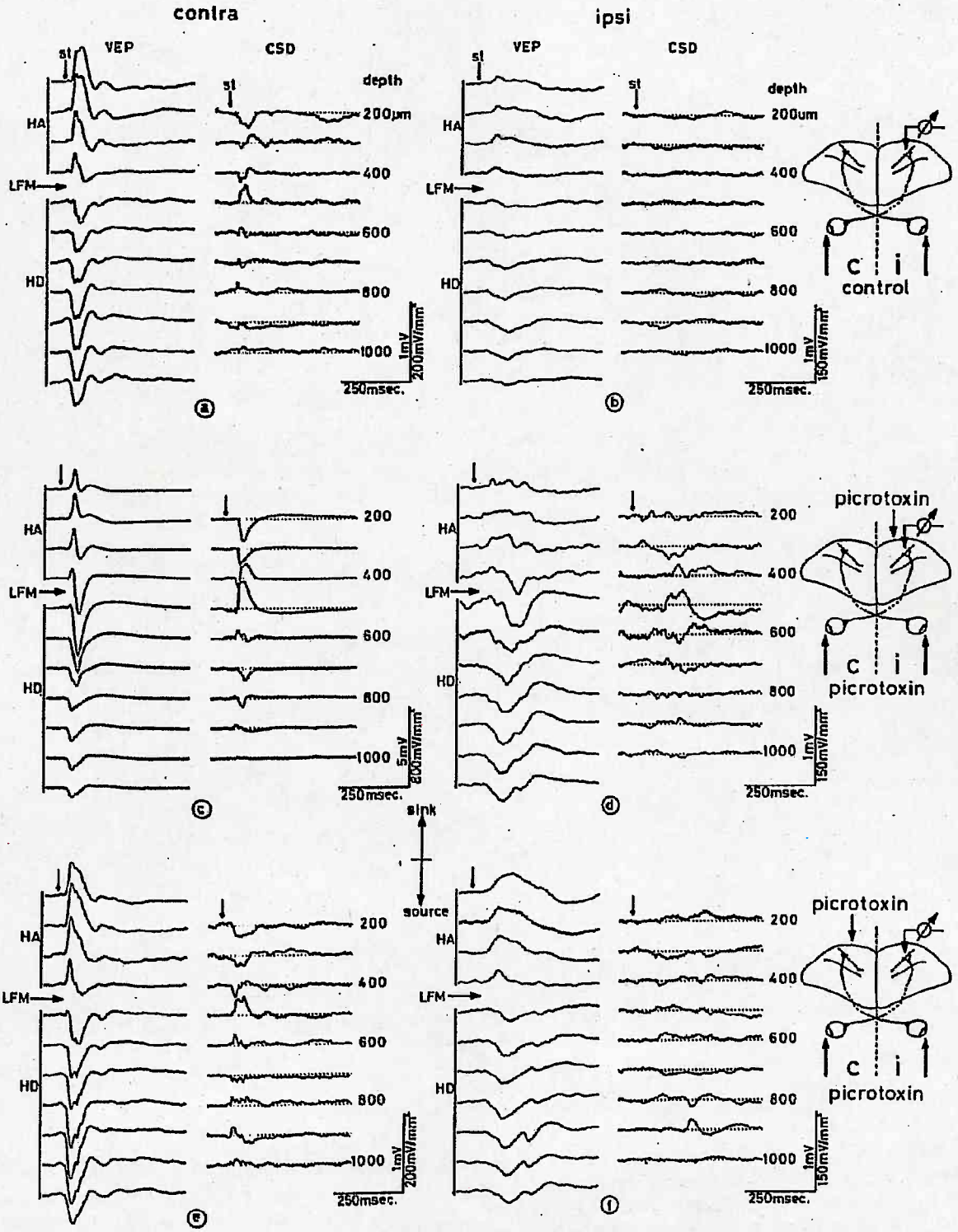

Fig. 7. Application of picrotoxin to the wulst, ipsilateral and contralateral to the recording site (see inset). The contralateral VEPs are only affected by ipsilateral application [compare (c) and (e)]. The ipsilateral VEPs are affected by both kinds of treatment [compare (d) and (f)]. The CSD analysis (right columns) of contralaterally VEPs shows no new components, but a more clear distribution of sinks and sources [compare (a) and (c)]. In contrast to normal birds, ipsilateral CSDs show a clearer distribution of sinks and sources within HA, after applying picrotoxin to the contralateral wulst [compare (b) and (f)]. Contra (c), ipsi (i); stimulated eye; st: stimulus; positive deflections upward.

(Fig. 7c). In addition, the zone of reversal seems to shift dorsally for about 100 μm. However, this is most probably not a real shift, but a consequence of the enhancement of the negative peak that obscures the preceding positive one.

Ipsilaterally evoked responses are also enhanced substantially by picrotoxin application. The distribution of sinks and sources obtained from the CSD analysis is much clearer than in control birds, where almost no reliable patterns exist (compare Fig. 7b and 7d).

Picrotoxin application to the wulst contralateral to the recording site

Application of picrotoxin to the wulst contralateral to the recording site had no significant effect on evoked responses to stimulation of the contralateral eye. Surprisingly however, the ipsilateral response was affected by this treatment. Figure 7f depicts recordings of ipsilateral VEPs of picrotoxin-treated birds (compare the VEPs of a control bird in Fig. 7b). Within HD, two negative components are detectable: a first one with an amplitude of about 0.25 mV (latency 110 ms), and a second component that appears with a latency of about 200 ms and amplitudes of about 0.12 mV.

The CSDs in Fig. 7e also show that reactions to contralateral stimuli are not affected by application of picrotoxin to the wulst contralateral to the recording site. However, CSDs resulting from ipsilateral stimulation again show much clearer sinks and sources compared with control birds. Two sinks can be observed at 200-μm depth with corresponding sources at 300 μm (Fig. 7f). The sinks most probably correspond to the large positive potential within HA, as the latencies are similar (120–150 ms). Another sink–source–sink sequence is observed between 600 and 800 μm within the HD, probably corresponding to the first negative peak of the ipsilaterally VEPs at the same depth. At 900 μm, another sink is observed corresponding in latency to the second peak of the VEP in the same depth.

Discussion

Our results demonstrate that ipsilateral stimulus responses of the visual wulst of zebra finches are indeed very weak, as was supposed by the theory put forward by Henke (1983). The area from which ipsilateral VEPs can be recorded is small when compared with the region where contralateral stimuli elicit responses. Moreover, ipsilateral responses are less reliable and smaller in amplitude. This is underscored by the results of the CSD analysis, that reveals reliable sinks and sources for contralateral, but not for ipsilateral stimuli. It is therefore reasonable to suppose that concomitant processing of stimuli from both eyes cannot be the primary task of the visual wulst in zebra finches. In birds with laterally placed eyes as in the zebra finch, this task may well be performed by the tectofugal pathway. Reliable ipsilateral responses can be obtained in the ectostriatum, the telencephalic target of the tectofugal pathway (Engelage & Bischof, 1988). Nonetheless, the discrepancy remains between the demonstration of a rather prominent recrossing projection to the visual wulst, which would allow the flow of information from the ipsilateral eye (Nixdorf & Bischof, 1982), and the fact that the observed reactions to ipsilateral stimuli are rather weak, as revealed by this study. Our experiments did not solve this discrepancy, but they provide the first hints that the lack of reliable ipsilateral responses might be due at least in part to several inhibitory mechanisms.

In the tectofugal pathway, ipsilateral responses are inhibited by the activity of the unstimulated contralateral eye (Engelage & Bischof, 1988). This is not the case for the thalamofugal pathway, as demonstrated by our experiments with TTX injection to the eye contralateral to the recording site. As we recorded simultaneously from the ectostriatum and the visual wulst, this lack of influence of the contralateral eye cannot be due to different experimental conditions: there is no apparent alteration in the ipsilateral responses within the wulst at the same time when a drastic enhancement of the ipsilateral response of the ectostriatum occurred (Fig. 5).

The picrotoxin application to the recording site shows that inhibition is a substantial part of the signal-processing machinery of the visual wulst. However, intrinsic inhibition does not affect the ipsilateral response preferentially, as application of picrotoxin enhances both the ipsilateral and contralateral response.

Application of TTX at the recording site provides the first hint that ipsilateral stimulus processing may be different from contralateral stimulus processing. There are three possible explanations for the result that TTX application to the recording site fully suppresses contralaterally, but not ipsilaterally evoked responses: first, inhibition may not be mediated by neurons that use sodium channels; second, the ipsilateral input may be located in deeper regions of the wulst and may therefore not have been reached by diffusion of TTX; and third, the ipsilateral responses are passively transferred from outside the wulst. All three explanations are unlikely.

We do not know of investigations showing that mechanisms for spike generation other than sodium channels exist. Our CSD analysis, at least from the picrotoxin experiments, show that the source of the ipsilaterally evoked responses is located within the HD and TTX application leads to a total depletion of responses to contralateral stimuli throughout the whole depth of the HA/HD. The reason why ipsilateral responses are affected in a different way from contralateral ones remains obscure. Perhaps, studies with different TTX concentrations and better control of drug diffusion may resolve this question.

The most surprising finding was that the application of picrotoxin to the wulst contralateral to the recording site results in a change in the ipsilaterally evoked VEPs of the wulst we recorded from. Obviously, this cannot be an effect of the spreading of picrotoxin from one hemisphere to the other because the response to contralateral stimuli is unaffected (compare Fig. 7e and 7c). The result demonstrates that the wulst areas of both sides interact. It seems most probable that this interaction is not mediated by a direct projection, as no direct wulst–wulst connection can be demonstrated anatomically. A re-examination of our own data, which we had erroneously interpreted as a wulst–wulst connection (Nixdorf & Bischof, 1982), suggests that what we showed was a connection from the wulst to the contralateral hippocampus (HP). This error was due to the fact that in the zebra finch there is no clear-cut difference between neuron morphology of visual wulst and HP. In the pigeon, Casini et al. (1986) showed that the HP in turn projects to the visual wulst. Thus, the anatomical correlate of the wulst–wulst connection as postulated by our findings is probably a wulst–hippocampus–contralateral wulst projection.

Our results do not explain why the influence of the contralateral wulst does not affect the evoked responses of the ipsilat-

eral wulst under control conditions. Developmental studies in our laboratory (Bredenkötter & Bischof, 1990) indicate that this projection from the contralateral wulst is much more effective in young zebra finches, and that the suppression of this influence develops in the course of ontogeny. At present, we cannot provide a functional explanation for this finding. It is interesting that during the time of development when the influence of ipsilateral stimuli diminishes in the visual wulst, there is an enhanced reaction to ipsilateral stimuli in the ectostriatum, the target of the tectofugal pathway in birds (Engelage & Bischof, in preparation). Probably, this means that processing of stimuli from both eyes was possible for both visual systems at earlier stages of evolution, and that in birds with laterally placed eyes this task was taken over mainly by the tectofugal system and was suppressed in the thalamofugal system.

In summary, our data show that the visual wulst of zebra finches is less involved in processing of ipsilateral information than that of pigeons or owls, confirming the theory put forward by Henke (1983). Our results further demonstrate that a second source of ipsilateral potentials may exist which is normally inhibited. Although this does not solve the problem of how the ipsilateral input is regulated in birds with lateral eyes, it does show that inhibition from other areas of the brain may play a role.

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