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Research report

Visual wulst influences on flash evoked responses in the ectostriatum of the zebra finch

Jürgen Engelage *, Hans-Joachim Bischof

Universität Bielefeld, Fakultät für Biologie, Lehrstuhl für Verhaltensphysiologie, Postfach 100131, 33501 Bielefeld, Germany

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Abstract

Anatomical data suggest that visual information from the thalamofugal pathway contributes to visual processing in the tectofugal pathway. We addressed the question of the functionality of anatomically described connections to the visual system of a laterally eyed bird, the zebra finch. The study shows the contribution of visual wulst efferents to visual processing in the ectostriatum by recordings of visually evoked slow field potentials. Suppression of visual wulst activity resulted in a selective reduction of distinct potential components in contralaterally evoked slow field potentials. A clear reduction was observed in the maximum amplitude of short latency components in the negative wave. Long latency components of the negative wave and the entire positive wave of the contralaterally flash evoked potentials were almost abolished. Ipsilateral visual evoked potentials (VEPs) were not significantly affected. Cooling and spreading depression of the optic tectum resulted in a uniform amplitude reduction of the negative wave. The positive wave was almost abolished. Ipsilateral VEPs disappeared completely during suppression of optic tectum activity. The results showed that the visual wulst has a significant, most likely facilitatory, influence on the processing of contralateral visual information in the ectostriatum. Ipsilateral stimulus processing was partly independent from visual wulst activity. A model for thalamo- and tectofugal connectivity in the ectostriatum is suggested.

Key words: Zebra finch; Visually evoked potential; Visual wulst; Ectostriatum; Optic tectum; Ipsi- and contralateral stimulus responses; Cooling; Spreading depression

1. Introduction

The visual system processes information in two prominent parallel pathways in each hemisphere in higher vertebrates (e.g. [19]). In birds, the thalamofugal pathway leads from the retina via the contralateral lateral geniculate nucleus pars dorsalis (GLd) to the visual wulst (VW). The tectofugal pathway leads from the retina to the optic tectum of the contralateral side, then to the nucleus rotundus of the thalamus and further on to the ectostriatum of the telencephalon. The thalamo- and tectofugal pathways process contra- and ipsilateral information, although the optic nerve in birds crosses over completely and visual information is carried primarily to the contralateral hemisphere (for reviews see [8,10,13]). Therefore ipsilateral and binocular stimulus processing in birds can only be achieved by secondary recrossing fibres between visual target areas of the left and right hemisphere. This has been shown for the thalamofugal pathway by Wilson [29,30] and Denton [9]. Engelage and Bischof [11,12] showed significant ipsilateral stimulus responses in the ectostriatum, the primary telencephalic target area of the tectofugal pathway. Bischof and Niemann [6] showed that the recrossing connection from the optic tectum to the contralateral nucleus rotundus, which is most likely to mediate most of the ipsilateral information, is much stronger than previously thought.

Based on physiological and anatomical data, in particular from the concatenation pattern of the different nuclei, it has been suggested that the thalamo- and tectofugal pathway in birds are homologue to the geniculocortical and extrageniculocortical pathway in mammals (e.g. [16,24,25]). In both, birds and mammals, it has been shown that the two parallel pathways are interconnected. A direct connection between the visual

* Corresponding author. Fax: (46) (521) 106-2998.

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wulst and the optic tectum has been shown anatomically [3,4] and electrophysiologically [1,2,3] in the pigeon. This connection is predominantly inhibitory [1,2,18]. Using horseradish peroxidase (HRP) injections, connections between the visual wulst and the ectostriatal belt (Ep) have been shown in the pigeon [15,20] and the Japanese Quail [26].

The present study examines the contribution of visual wulst efferents to the processing of visual information in the ectostriatum of a laterally eyed bird. Two questions made these experiments particularly interesting. First: in which way are contra- and ipsilateral stimulus responses in the ectostriatum affected by the visual wulst efferents? Second: which of the two parallel visual pathways is the major source for the ipsilateral stimulus response in the ectostriatum?

2. Material and Methods

The experiments were performed on 15 adult male and female zebra finches. The birds were deeply anaesthetized with urethane (20% w/v, 0.1 ml) and mounted in a specially designed stereotaxic headholder [5]. Evoked potentials were recorded with glass micropipettes filled with Alcian blue in 3 M NaCl (5–15 MΩ). Stereotaxic coordinates for the electrode penetrations were derived from an atlas of the zebra finch brain.

Visual 'Ganzfeld' stimuli ensuring illumination of the entire retina were provided by a stroboscope. Flashed stimuli (200 μs duration) were directed to one or both eyes by a fibre optics system (eye distance 5 mm, relayed by 2 cm of black tubing from the end of the fibre optics system). Shutters were used to close the left, right or both fibre optics system(s). Thus the birds' left and right eyes could be stimulated separately (contra- or ipsilateral monocular stimuli) or simultaneously (binocular stimuli) with the same stroboscope. Recordings of electroretinograms (ERGs) and VEP recordings from enucleated birds [11] showed that under our experimental conditions no light is spreading from one eye to the other. Controls were made by closing shutters in the fibre optics system or in some cases by removing the fibre optics system from the eyes.

The terms ipsilateral and contralateral refer to the position of the recording electrode in the ectostriatum, relative to the stimulated eye. In the same sense they refer to the application site of cooling and spreading depression relative to the recording electrode.

Signals were amplified 10×, filtered through a 4-Hz high-pass and averaged 64 times by a Nicolet Signal Averager. The interstimulus time interval was 5 s. The recording of an averaged evoked response was completed in about 5 min. Data acquisition and processing was accomplished by an HP-86 microcomputer. Peak amplitudes and latencies were estimated on line with a minimum-maximum routine of the microcomputer. In addition, more detailed information on amplitudes and latencies was obtained by processing the evoked potential plots on a graphic tablet.

Reversible suppression of the neuronal activity in the visual wulst ipsilateral to the recording site was achieved by two different techniques. Since the surface of the visual wulst and the optic tectum are easily accessible, activity could be suppressed by cooling it down with small pieces of ice placed on the surface of the entire area during recordings. After every recording the ice water was absorbed with filter paper. In other experiments, neuronal activity was suppressed by application of a KCl solution to the surface, spreading depression [17]. Usually, spreading depression was induced by placing small pieces of filter paper soaked with 5% KCl on the surface of the visual wulst or the optic tectum, ipsilateral to the recording site in the ectostriatum. Concentration dependent effects were tested with 10% and 15% solutions of KCl as well. The effectiveness of the treatments was controlled in some experiments by simultaneous recordings from the suppressed areas. Suppression of visual wulst or optic tectum activity was always performed in the hemisphere ipsilateral to the recording site. All statistical results are given as median values ± 1 standard error of the median (S.E.). Data were tested for significant median differences with a two tailed Mann–Whitney *U*-test [23]. Although cooling experiments could be repeated several times without significant changes in the VEP waveform, we only used the first application of either cooling or spreading depression for the statistical analysis.

3. Results

The recordings presented in this study are from the dorso-rostro-lateral area of the ectostriatal complex. All samples were recorded at similar electrode positions (anterior 3.5 mm, lateral 3.75 mm to 4.0 mm, depth 2750 μm to 3250 μm). A more detailed description of the distribution and characteristics of ectostriatal evoked potentials (VEPs) has been given elsewhere [11,12]. At the recording sites efferent projections from the visual wulst have been shown with anatomical techniques [20,26]. VEPs in this region were mainly characterized by a slow negative-positive wave with at least two clearly distinguishable peaks (short (N′) and long (N″) latency components (Fig. 1, A1). Occasionally, similar subdivisions were observed for the positive wave (P) as well. For the negative peak we measured up to −700 μV (median 500 μV ±73 S.E.), and up to + 500 μV (median 326 μV ±43 S.E.) for the positive wave (P). The latencies for the first negative

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Fig. 1. Changes in the contra- and ipsilateral ectostriatal VEPs during cooling of the visual wulst. A: stimulation contralateral to the recording site during cooling of the visual wulst ipsilateral to the recording site. B: stimulation ipsilateral to the recording site during cooling of the visual wulst ipsilateral to the recording site. (1) before cooling; (2) during cooling; (3) recovery from cooling; (4) computer simulated amplitude enhancement of VEP wave. For the calculation of (4) see text. Superimposed (thin dashed line) is the VEP averaged from control and recovery. The arrows indicate the major differences between the VEPs recorded during and after cooling (recovery) of the visual wulst. Thin horizontal lines mark the zero line and thin vertical lines mark the locations of amplitude measurements. Average 64 ×, bin width 500 μs, stimulus at 50 ms. Note the severe amplitude reduction of N′ and the almost complete diminution of N″ and P due to the suppression of visual wulst activity in A2. As no significant differences in the ipsilateral VEPs before and during cooling were observed, no amplitude corrections were calculated and no superpositions were plotted for these VEPs.

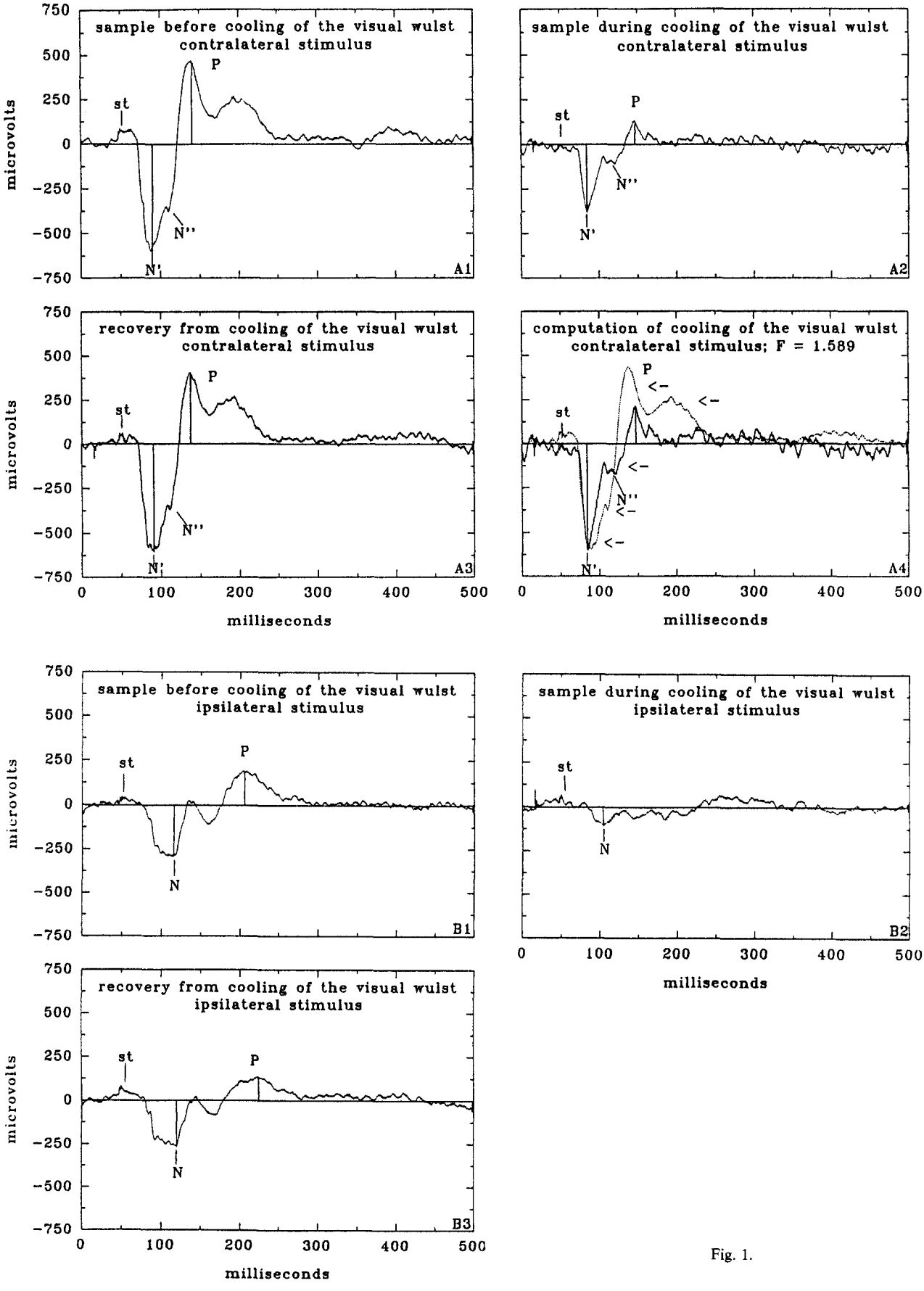
sample before cooling of the visual wulst contralateral stimulus
sample during cooling of the visual wulst contralateral stimulus
recovery from cooling of the visual wulst contralateral stimulus
computation of cooling of the visual wulst contralateral stimulus; F = 1.589
sample before cooling of the visual wulst ipsilateral stimulus
sample during cooling of the visual wulst ipsilateral stimulus
recovery from cooling of the visual wulst ipsilateral stimulus
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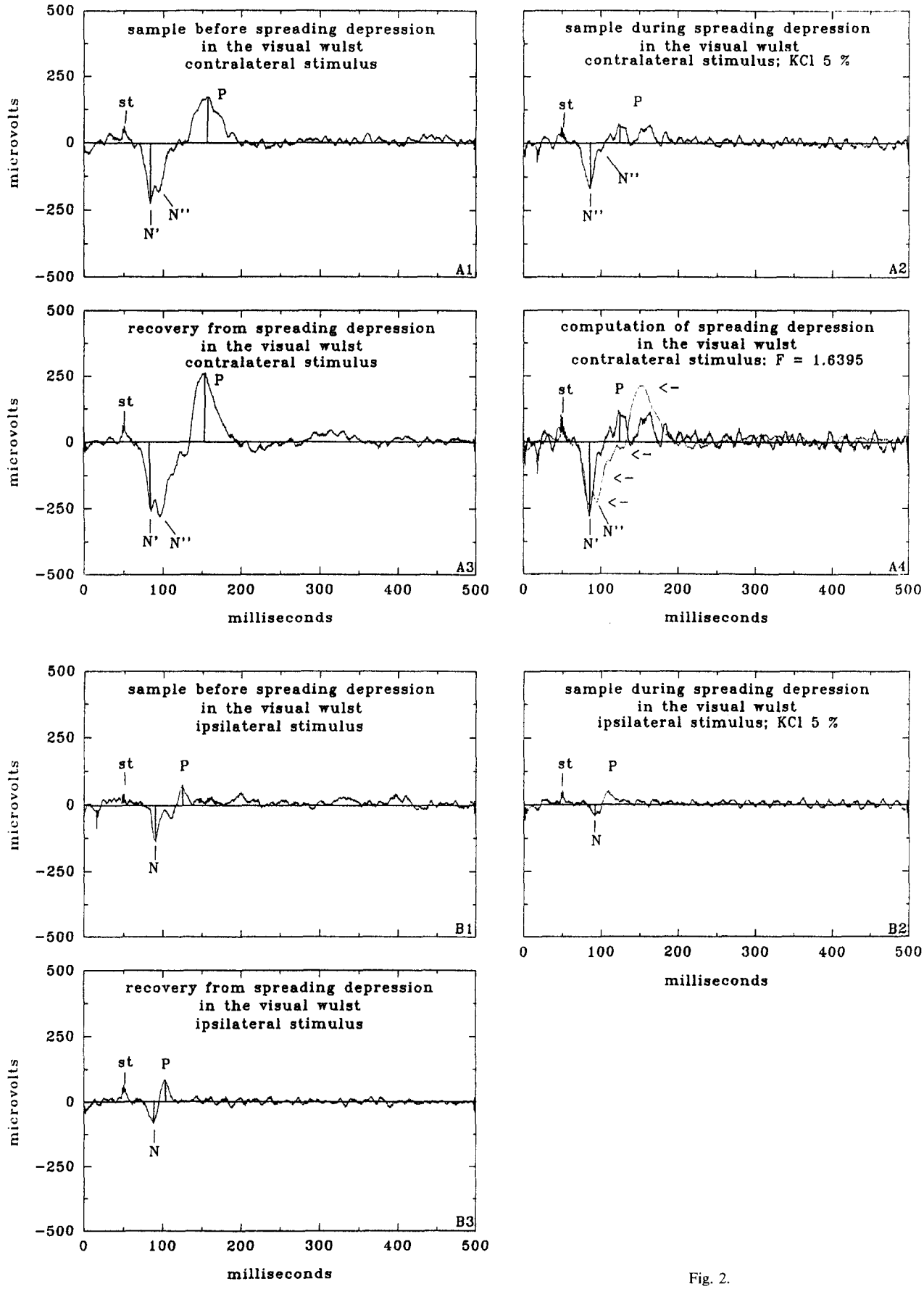
Fig. 1.


sample before spreading depression
in the visual wulst
contralateral stimulus
st
P
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A1
sample during spreading depression
in the visual wulst
contralateral stimulus; KCl 5 %
st
P
N''
A2
recovery from spreading depression
in the visual wulst
contralateral stimulus
st
P
N'
N''
A3
computation of spreading depression
in the visual wulst
contralateral stimulus: F = 1.6395
st
P
<-
N'
N''
A4
sample before spreading depression
in the visual wulst
ipsilateral stimulus
st
P
N
B1
sample during spreading depression
in the visual wulst
ipsilateral stimulus; KCl 5 %
st
P
N
B2
recovery from spreading depression
in the visual wulst
ipsilateral stimulus
st
P
N
B3
microvolts
500
250
0
-250
-500
0
100
200
300
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500
milliseconds

Fig. 2.

wave were between 40 ms and 65 ms, and between 125 ms and 200 ms for the positive wave [11,12]. This waveform was reproducible for a given coordinate and remained stable for several hours at the same electrode position.

Ipsilateral VEPs were mainly characterized by a slow negative wave (193 μV ±66) and a small positive wave (Fig. 1, B1). A separation of two different peaks was possible in some, but not all cases. These ipsilateral VEPs were smaller in amplitude and had longer latencies (50 to 70 ms for the negative wave) than the contralateral VEPs obtained at the same recording sites (see also [11,12]).

3.1. Suppression of visual wulst activity

A comparison of the resulting VEPs during cooling with those obtained before and after cooling (recovery) showed mainly two effects (compare Fig. 1, A1 through to A3). First, the maximum amplitudes of the negative wave (685 μV ±237 vs. 414 μV ±132) and the positive wave (475 μV ±143 vs. 184 μV ±95) were clearly reduced in amplitude. Both amplitudes were significantly different ($P < 0.1$; $z > 1.96$, $n = 3$). Second, the changes in the long latency components (N″) of the negative wave were much more severe than those of the short latency components (N′). The long latency negative wave components (N″) and the positive wave (P) were almost completely abolished. Moreover, the observed change in the potential shape during cooling was not only the result of a uniform constant amplitude reduction in all bins. This could be shown by comparing the average of the VEPs before cooling and recovery, with the VEPs during cooling, corrected for the maximum amplitude in all bins (Fig. 1, A4). This corrected VEP was calculated by a multiplication of the amplitude in each bin of the during cooling average with the ratio from the maximum amplitude of the negative wave (N′), average from pre cooling and the maximum amplitude of the negative wave (N′), average from recovery (Fig. 1, A3) and during cooling, respectively. Therefore not only an unselective uniform amplitude reduction but a selective diminution of distinct potential components was observed (F = [[N′ pre cooling + N′ recovery]/2]/N′ during cooling).

Recordings sampled 10–15 min after absorbing the ice water from the surface of the visual wulst revealed a complete recovery of the ectostriatal VEPs from these effects (Fig. 1, A3).

The amplitudes of the entire ipsilateral VEPs were reduced (399 μV ±95 vs. 203 μV ±95 for the negative wave, and 282 μV ±95 vs. 167 μV ±95 for the positive wave). No selective changes in the shape of the ipsilateral VEPs were observed (Fig. 1, compare B1 and B2). However, the results for the negative and the positive wave were not significantly different. Similar to contralateral stimulation, the recovery from cooling was complete (Fig. 1, B3).

The cooling procedure could be repeated several times in intervals of about 15 to 20 min without any sign of severe permanent alterations of the VEPs. Occasionally, a slight overall increase in amplitude was observed.

Using the spreading depression technique, we obtained results that were very similar to those obtained with cooling. Spreading depression induced with a 5% solution of KCl in the visual wulst led to almost complete reduction of long latency components (N″) in the negative wave and the positive wave (P) and a comparably small reduction of short latencycomponents (N′) in the negative wave (compare Fig. 2, A1 and A2). The amplitude of the negative wave (N′) was reduced about 30% during spreading depression (339 μV ±37 before spreading depression vs. 241 μV ±26 during spreading depression). The amplitude of the positive wave (P) was reduced about 60%, i.e. twice as much (293 μV ±33 vs. 96 μV ±25). Ipsilateral stimulus responses were reduced during inactivation of the visual wulst with 5% KCl (Fig. 2, B1 and B2), but not completely abolished (156 μV ±31 vs. 109 μV ±44 for the negative wave and 107 μV ±13 vs. 98 μV ±21). Again, the recoveries from the KCl treatment were almost complete (compare Fig. 2, A1 and A3 and Fig. 2, B1 and B3). The results for the negative wave of contralaterally evoked VEPs were significantly different ($P < 0.01$; $z > 2.1$; $n = 8$). Consistent with the larger voltage dif-

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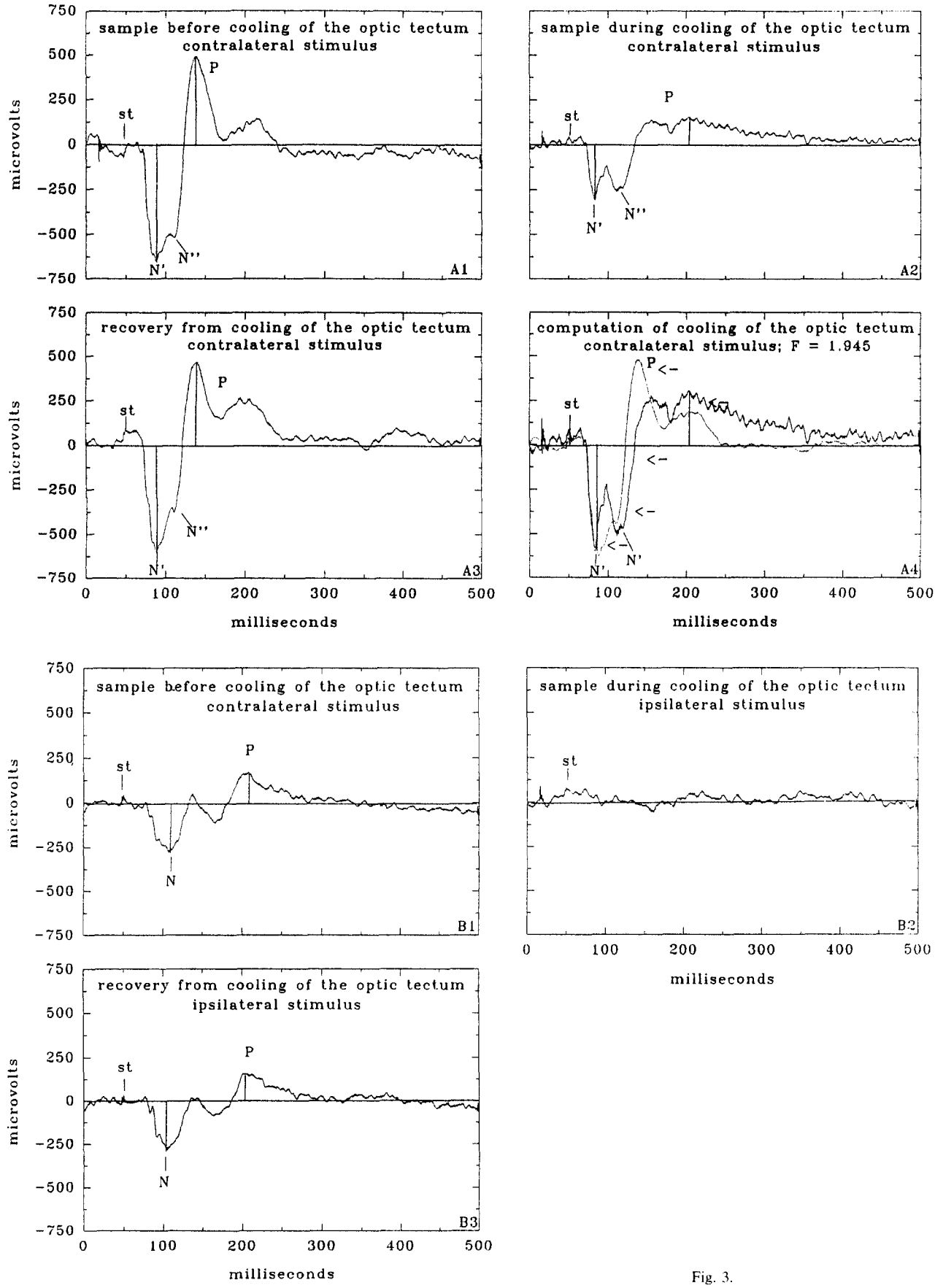
Fig. 2. Changes in the contra- and ipsilateral ectostriatal VEPs during spreading depression in the visual wulst. A: stimulation contralateral to the recording site during spreading depression in the visual wulst ipsilateral to the recording site. B: stimulation ipsilateral to the recording site during spreading depression in the visual wulst ipsilateral to the recording site. (1) before spreading depression; (2) during spreading depression; (3) recovery from spreading depression; (4) computer simulated amplitude enhancement of VEP wave. For the calculation of (4) see text. Superimposed (thin dashed line) is the VEP averaged from control and recovery. The arrows indicate the major differences between the VEPs recorded during and after spreading depression (recovery) of the visual wulst. Thin horizontal lines mark the zero line and thin vertical lines mark the locations of amplitude measurements. Average 64 ×, bin width 500 μs, stimulus at 50 ms. Note the reduction of N″ components in the negative wave and the almost complete diminution of the positive wave due to suppression of visual wulst activity in A2. As no significant differences in the ipsilateral VEPs before and during cooling were observed, no amplitude corrections were calculated and no superpositions were plotted for these VEPs.


sample before cooling of the optic tectum
contralateral stimulus
sample during cooling of the optic tectum
contralateral stimulus
recovery from cooling of the optic tectum
contralateral stimulus
computation of cooling of the optic tectum
contralateral stimulus; F = 1.945
sample before cooling of the optic tectum
contralateral stimulus
sample during cooling of the optic tectum
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recovery from cooling of the optic tectum
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-250
-500
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400
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Fig. 3.

ference between the positive waves before and after cooling, the significance was increased ($P < 0.001$; $z > 3.1$; $n = 8$). No significant differences were obtained for the negative and the positive wave during ipsilateral stimulation (Fig. 5). The non uniform reduction of distinct VEP components could be shown as described above for cooling of the visual wulst.

3.2. Suppression of optic tectum activity

Results obtained during reversible suppression of the optic tectum were different from those in the visual wulst. No obvious selective effects on the negative wave were observed in these experiments. Only a uniform reduction in amplitudes could be shown (Fig. 3A). During cooling of the optic tectum, the amplitude of the negative wave (N′ and N″) of the contralateral VEP was reduced about 45% (658 μV $\pm$218 vs. 354 μV $\pm$116 for N′). The positive wave was reduced by 67% (453 μv $\pm$150 vs. 150 μv $\pm$41, $P < 0.1$, $z > 1.9$; $n = 3$).

The results obtained during spreading depression in the optic tectum, induced by 5% KCl, were very similar to those described for cooling (516 μv $\pm$158 vs. 293 μv $\pm$124 for N′, Fig. 4A). Again the positive wavewas more severely reduced (326 $\pm$94 vs. 156 μv $\pm$42, $P < 0.1$; $z > 1.9$; $n = 3$).

As no significant ipsilateral VEPs could be detected from background noise during cooling or spreading depression in the optic tectum (Fig. 3, B2 and Fig. 4, B3), no amplitude measurements were taken for ipsilateral VEPs, no amplitude correction was calculated and no statistics were carried out.

4. Discussion

Our results clearly show that ectostriatal responses are not exclusively dependent on tectofugal input. Contralateral VEPs are selectively reduced by visual wulst suppression. The suppression effect is most obvious in the long latency components (N″ and the P-wave) of the VEP. Short latency components are less affected. Ipsilateral VEPs were also reduced during suppression of visual wulst activity. However, the reduction was uniform and not significant. Compared to visual wulst suppression, optic tectum suppression also reduces the short latency components (N′) of the contralateral VEP. Ipsilateral VEPs were significantly reduced or abolished.

4.1. Ipsilateral stimulus processing

Our results show that visual wulst efferents into the ectostriatum obviously do not contribute significantly to the processing of ipsilateral stimuli. On the other hand, they confirm that the tectofugal projection makes a significant contribution to ipsilateral stimulus processing [11–13]. However, the present understanding of the wiring pattern of the optic tectum does not allow for a comprehensive interpretation of the data. Significant recrossing projections from the optic tectum to the contralateral nucleus rotundus [6,11] and tectum–tectum connections [21] have been previously described. Our results clearly show that the tectum-tectum projection is very important for the processing of ipsilateral visual stimuli as observed in these experiments. This tectum–tectum interaction has been described as both inhibitory *and* excitatory in the pigeon [22]. Results from experiments with picrotoxin induced disinhibition in the optic tectum also confirm the notion of an inhibitory and excitatory tectum–tectum interaction in the zebra finch [13].

Our own results could be explained by assuming an excitatory tectum-tectum interaction. Inhibitory interaction should result in the reverse effect. We do not yet know the weighting functions for the efficiency of excitatory and inhibitory influences within the tectal wiring pattern that would be needed for a complete understanding and interpretation of our results. However, all our results are in agreement with previous reports.

4.2. Contralateral stimulus processing

With contralateral stimulation, suppression of either thalamofugal or tectofugal afferents results in a clear diminution of ectostriatal VEP components. In con-

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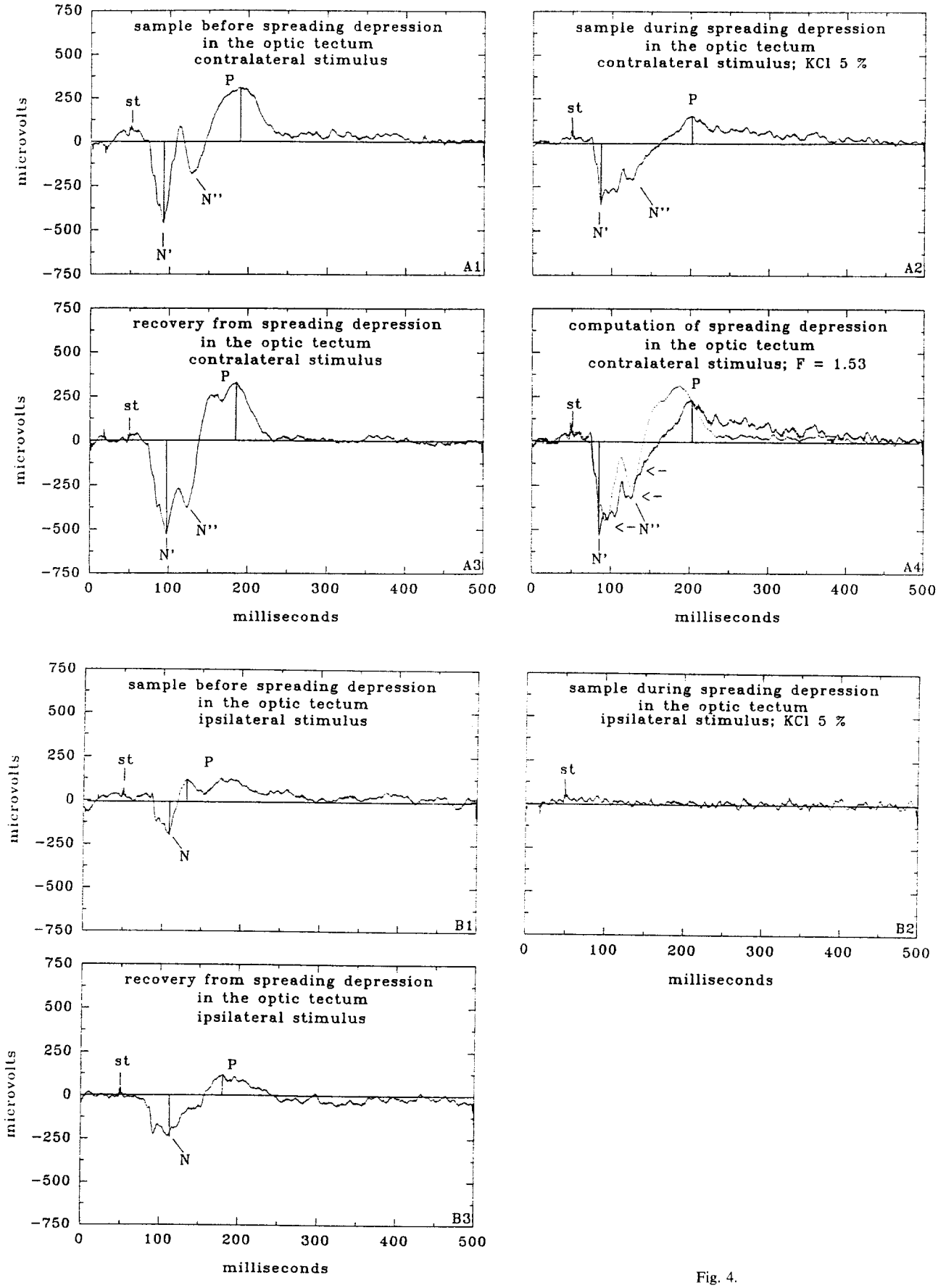
Fig. 3. Changes in the contra- and ipsilateral ectostriatal VEPs during cooling of the optic tectum. A: stimulation contralateral to the recording site during cooling of the optic tectum ipsilateral to the recording site. B: stimulation ipsilateral to the recording site during cooling of the optic tectum ipsilateral to the recording site. (1) before cooling; (2) during cooling; (3) recovery from cooling; (4) computer simulated amplitude enhancement of VEP wave. For the calculation of (4) see text. Superimposed (thin dashed line) is the VEP averaged from control and recovery. The arrows indicate the major differences between the VEPs recorded during and after cooling (recovery) of the optic tectum. Thin horizontal lines mark the zero line and thin vertical lines mark the locations of amplitude measurements. Average 64 ×, bin width 500 μs, stimulus at 50 ms. Note the almost uniform reduction of all VEP components in the negative wave and the severe reduction of the positive wave due to suppression of optic tectum activity in A2. As no ipsilateral VEPs were detectable from background noise, no amplitude corrections were calculated and no superpositions were plotted for these VEPs.


sample before spreading depression
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contralateral stimulus
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contralateral stimulus; KCl 5 %
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computation of spreading depression
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contralateral stimulus; F = 1.53
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Fig. 4.

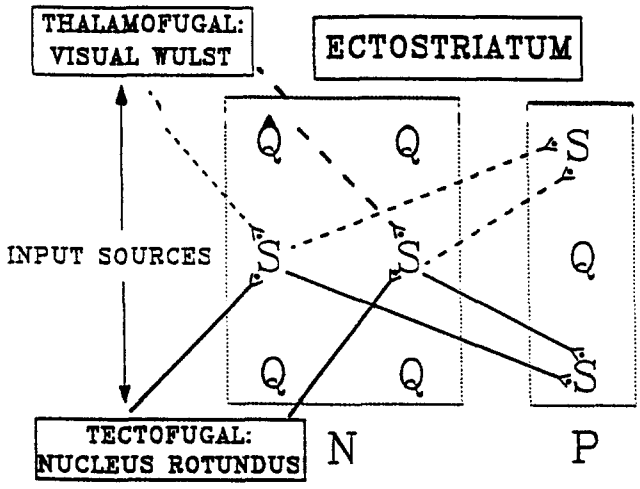

Fig. 5. Suggested connectivity pattern for thalamofugal-tectofugal interaction of input sources in the ectostriatum. Q, current sink; S, current source. Dashed lines, thalamofugal input; solid lines, tectofugal inputs. The line thickness indicates the strength of the respective input sources. The dashed boxes only separate the source-sink-source and the sink-source-sink sequences for the N- and P-wave components and do not represent different parts of the ectostriatum. First N column = N′ second N column = N″. For a detailed description see Discussion.

trast to ipsilateral stimulation, the ectostriatal VEP response is never completely abolished. This shows that both pathways contribute to the contralateral ectostriatal VEP. However, for optic tectum suppression, one has to consider the likelihood that complete suppression of the entire structure was not achieved. This is unlikely for the visual wulst. The visually responsive area is so small [7] that a complete suppression ofthis structure could be achieved. Therefore, in experiments with optic tectum suppression, the ectostriatal VEP was composed of reduced tectofugal and complete wulst inputs. In experiments with visual wulst suppression, the ectostriatal VEP is predominantly generated by tectofugal afferents. However, this does not affect our main conclusion. We may underestimate the tectofugal contribution to the ectostriatal VEP, but there is no doubt that visual wulst efferents facilitate the ectostriatal VEP response.

Besides an intratelencephalic projection from the visual wulst to the ectostriatum [29,26], a bilateral visual wulst–optic tectum projection has been described [1–4]. This projection could potentially modulate the tectofugal processing. However, as this projection has been described as predominantly inhibitory, the facilitatory effect of the visual wulst on the ectostriatal VEP response suggests that the observed visual wulst influence is mediated via the intratelencephalic projection. If the tectal projection from the visual wulst is to be the main channel of visual wulst information transfer to the tectofugal pathway, the ectostriatal VEPs should be enhanced during suppression of visual wulst activity. Our data show that the reverse is the case. However, a contribution of this projection to the observed results cannot be excluded completely yet.

4.3. Connectivity within the ectostriatum

The different patterns in the reduction of contralateral VEPs during either visual wulst or optic tectum suppression can be explained by a model of the basic ectostriatal connectivity pattern. This model is based on data from an earlier paper and the present results. An analysis of current source densities [12] showed three different sink-source patterns for the N′, N″ and the P component. This showed that each of the potential components is generated by a different subset of neurons. The subsets generating the N′ and N″ component receive input from the tecto- and thalamofugal pathway. The tectofugal input is uniform for N′ and N″ (both components are reduced to the same degree during tectal suppression). However, thalamofugal input is dominant for the N″ component (N″ is reduced much more severely than N′ during visual wulst suppression). The N′ and N″ component may represent independent responses of ectostriatal neurons. However, our results as well suggest that the generation of the P-wave is dependent on a concomitant activation of the N′ and N″ component. The P-wave is severely reduced (almost abolished) if only one generator input source (N′ or N″) is reduced (Fig. 5). Moreover, the relation between the N components and the P-wave is not linear. The P-wave is almost abolished, even if a substantial amount of the N wave is preserved. A linear reduction of the N′ and N″ generator source

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Fig. 4. Changes in the contra- and ipsilateral ectostriatal VEPs during spreading depression in the optic tectum. A: stimulation contralateral to the recording site during spreading depression in the optic tectum ipsilateral to the recording site. B: stimulation ipsilateral to the recording site during spreading depression in the optic tectum ipsilateral to the recording site. (1) before spreading depression; (2) during spreading depression; (3) recovery from spreading depression; (4) computer simulated amplitude enhancement of VEP wave. For the calculation of (4) see text. Superimposed (thin dashed line) is the VEP averaged from control and recovery. Thin horizontal lines mark the zero line and thin vertical lines mark the locations of amplitude measurements. Average 64 ×, bin width 500 μs, stimulus at 50 ms. Note the almost uniform reduction of all VEP components in the negative wave and the severe reduction of the positive wave due to suppression of optic tectum activity in A 2. As no ipsilateral VEPs were detectable from background noise, no amplitude corrections were calculated and no superpositions were plotted for these VEPs.

does not account for the severe P-wave reduction during either optic tectum or visual wulst suppression.

5. Conclusions

Our study clearly shows that the ectostriatum integrates information from the thalamofugal pathway. Most probably, the visual wulst influence is mediated to the ectostriatum by the visual wulst–hyperstriatum–ectostriatum pathway [20,26] and modulates the tectofugal processing.

As visual wulst suppression clearly reduces ectostriatal VEP responses, we assume that the visual wulst influence on the ectostriatum is facilitatory. Whether this facilitation is directly, i.e. the excitation of the thalamofugal pathway adds to that of the tectofugal pathway, or whether inhibitory mechanisms are involved, cannot be decided yet.

The assumption of a facilitatory visual wulst influence on the ectostriatum is in full agreement with lesion experiments in the tecto- and thalamofugal pathway of the pigeon [14]. These studies show that the visual wulst as a 'specialist' adds to the overall performance in discrimination tasks, but is not essential for basic visual information processing. Therefore, these studies suggest an integrative role for the ectostriatum in visual processing in the same way as our physiological recordings do. In addition, the described thalamo–tectofugal connectivity may contribute to visual discrimination learning and visual transfer learning [27,28]. If the visual wulst mainly facilitates ectostriatal processing, it is easy to understand that visual wulst lesions are more effective in combined thalamo–tectofugal lesion experiments. The ectostriatum may be capable to compensate for this facilitatory visual wulst influence to a large degree. Therefore our data and the suggested model of thalamo–tectofugal connectivity in the ectostriatum provide a framework in which the behavioural results from lesions studies may be explained and understood much better.

Acknowledgement

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