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ANTIDROMIC ACTIVATION OF THE ISTHMO-OPTIC NUCLEUS

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SUMMARY

1. This paper describes experiments carried out to record from output cells in the isthmo-optic nucleus.

2. One-hundred and twenty-seven axonal responses were fired at fixed latency from the optic nerve-head.

3. Ninety-nine cell responses were fired trans-synaptically from the optic nerve-head.

4. Ninety-four cells were activated antidromically from the optic nerve-head.

5. Tectal tracks could be recognized by the field potential profile of the N-wave, R-wave and P-wave, and by the occurrence of fixed latency axonal responses and trans-synaptically fired cells.

6. Tectal tracks were verified histologically.

7. Tracks yielding antidromically activated cells were traced histologically to the isthmo-optic nucleus.

8. The antidromic A-wave could be recorded from the nucleus, corresponding in timing to the invasion of cell bodies.

9. Somatic records in the nucleus could be recognized by their duration, conformation, and A–B blocking.

10. When antidromic discharge was interacted with orthodromic firing, collision evidence could be provided, showing that the orthodromic impulse travels centrifugally to the retina.

INTRODUCTION

The anatomical identification of the centrifugal pathway to the retina (Cowan, Adamson & Powell, 1961) gives the avian brain a special usefulness for physiological studies of centrifugal control in the visual system. This paper describes observations made with single unit recordings from cells in the isthmo-optic nucleus, the origin of centrifugal fibres running to the retina. It was intended to identify output cells in the nucleus by antidromically activating their axons at the optic nerve-head, and to use the

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'collision' technique to establish that the direction of conduction of their action potentials is centrifugal.

The closeness of the isthmo-optic nucleus to the medial border of the optic tectum means that evidence must be provided for three distinct issues. Electrical stimulation of the optic nerve-head will activate the isthmo-optic system antidromically, and the retinotectal system orthodromically. Therefore histological evidence must be provided that tracks aimed at the isthmo-optic nucleus have actually penetrated the nucleus. Secondly, it must be established that the response properties of tracks made into the nucleus can be distinguished from those of tectal tracks. The third issue concerns the identification of output cells. Electrical stimulation of the optic nerve-head will activate three types of unitary response at fixed latency. Retinotectal axons will be excited orthodromically; isthmo-optic axons will be excited antidromically; and output cells of the nucleus will be excited antidromically. Therefore a distinction must be made between the two classes of axonal spike and records from somata in the nucleus.

A preliminary account of these experiments has been published (Holden, 1966).

METHODS

The experiments were carried out on thirty-eight feral pigeons. Anaesthesia was produced by intraperitoneal injection of 4–6 ml. of urethane (20 g/100 ml.). The body was kept heated throughout dissection and recording.

The trachea was cannulated with a straight tube. The feathers were cut from the head and neck, and the animal was fixed in a rigid head-holder. The first part of the dissection consisted in exposure of the right-hand side of the cerebellum. This was carried out between the boundaries formed by the longitudinal cerebellar sinus medially, the forebrain anteriorly, and the angle between the cerebellum and tectum or brain-stem laterally. The skull and spongy bone were removed with a small dental burr. A sharp 18-gauge needle was inserted tangentially through the bone over the first or second visible cerebellar folium. This operation was carried out under the high power of a Zeiss binocular microscope. The opening was enlarged cautiously with fine forceps and a dura hook. The bone was removed from the first three exposed folia. No attempt was made to strip the bone from the longitudinal sinus. In most pigeons this large vein runs medially down the cerebellum, though variants were noted. The dura was removed from the cerebellum. An 18-gauge needle was carefully inserted, and the opening was widened with a dura hook. The field then lay exposed in pia-arachnoid, ready for insertion of the recording electrodes. At this stage it was covered with Ringer-soaked gel-foam.

The right-hand tectum was then exposed, for electrical stimulation. The dissection was carried out exactly as described in a previous paper (Holden, 1968*a*) and the exposed lateral tectum was covered in Ringer-soaked gel-foam. The left eye was prepared for the insertion of bifocal stimulating electrodes through a hole in the limbus. This too has been described in detail elsewhere (Holden, 1968*a*).

The tectal stimulating electrodes were set in position using a Prior manipulator which could drive them radially into the lateral tectum. They consisted of fine steel needles, insulated to the tips with Araldite coating compound, with the tips ground bare before each

experiment. The tips were thrust lightly into the tectum for about 0·25 mm, the tectum was flooded with paraffin, and just molten low-melting point paraffin wax was poured on to provide mechanical stabilization. The electrodes used to stimulate the optic nerve-head were bifocal silver balls, each carried in a pipette shaft. They were aimed under ophthalmoscopic control at the upper end of the pecten. They too were carried on a Prior micromanipulator.

The recording pipettes were filled with 3 M-KCl, and showed impedances of 3–7 MΩ at 50 c/s. They were carried on an oil-filled Wright microdrive which was itself carried on a Prior micromanipulator. This was arranged so that the electrodes could be inserted into the cerebellum 2 mm lateral to the mid line, in a sagittal plane, inclined at 40° to the horizontal plane fixed by the head-holder. The head was held in the position of normal walking. Electrode insertion was made under the control of a Zeiss binocular microscope. The tips were inserted into a region free from surface vessels, and advanced 4 mm on the Prior manipulator, which carried them deep into the cerebellum, providing a spectacular display of unitary discharge. The track was then waxed in, and further advance made with the microdrive.

Signals were led from a silver wire dipping into the pipette via a cathodally screened lead to one grid of a double cathode follower valve. Recordings were made between this and an indifferent silver electrode connected to the other grid. A Tektronix 122 preamplifier and 502 oscilloscope were used. Responses were displayed upon the upper beam, the lower beam showing time-markers and stimulus-markers. Time-markers were obtained from Dekatron counters counting down the output of a crystal oscillator. Control of sweep repetition rate was also obtained from the counter, which provided a pulse to trigger the oscilloscope timebase.

Electrical stimuli were provided by two channels of a transistorized constant current stimulator (Bannister & Kay, 1965). This provided rectangular pulses, isolated from earth, of controlled duration and amplitude. Single pulses, or pairs of pulses with independent delay, or trains of pulses could be used. The stimulating arrangements enabled independent stimulation of the optic nerve-head and of the lateral tectum. The effects of stimulating the lateral tectum upon the isthmo-optic nucleus are described in a following paper (Holden, 1968 *c*).

Extensive films were taken at the time of the experiment with a Shackman camera.

Histological verification. All recordings were made with micropipette electrodes, and pilot experiments showed that it was not possible to make satisfactory DC lesions from their tips. It was then realized that verification would have to be of electrode tracks (cf. Powell & Mountcastle, 1959). Brains were fixed in formalin without perfusion, and prepared for embedding in LVN (low viscosity nitrocellulose). They were sectioned at 50 μ in a sagittal plane, and stained with cresyl violet. Electrode tracks were difficult to see near the tips, and were only clearly visible when they had become infiltrated with blood. In later experiments tracks were driven deep to the nucleus, and left *in situ* in the living bird for 2–3 hr. This produced enough local damage for successful histological verification.

RESULTS

Types of unitary response recorded on medial tracks

Recordings were made of 324 unitary responses which were fired by electrical stimulation of the optic nerve-head. They fell into three categories.

Axonal spikes. One hundred and twenty-seven of the unitary responses were judged to be fibre spikes by their fixed latency, short recovery time to paired stimuli, and by their spike conformations. Many of these spikes preceded a tectal N-wave. It was occasionally possible to make observa-

tions upon a near minimal N-wave, where, in response to paired twice-threshold stimuli both the axonal spike and the N-wave disappeared in a quantal step at the same stimulus separation.

The fibre spikes showed two typical conformations both seen previously in lateral tectal recording. The first was a triphasic spike, with a prominent second negative-going phase, and a duration of 0·5 msec. Spike amplitudes ranged from 100 to 500 μV. Multispike records were obtained more often on medial tracks than on lateral tracks, possibly because in the medial tectum there was a tendency for tracks to pass tangentially through tectal laminae. These axonal spikes never showed injury firing following electrode movement.

The second type of axonal spike was monophasic positive in conformation, with a linear rising and falling phase, and a duration of 0·5–1·0 msec. Spike amplitudes were up to 10 mV. These spikes were highly unstable, and could usually only be held for seconds. They could be distinguished from somatic records by their conformation and short duration, by their lack of A–B blocking to paired stimuli, and by their location where a tectal N-wave could be recorded. Notched positive spikes like those recorded at the lateral tectum were not observed in medial tracks.

Fifty-two of the axonal spikes were tested with paired twice-threshold stimuli. The second spike failed abruptly at a critical stimulus separation, without latency fluctuation. The distribution of recovery times is shown in Fig. 1. This distribution is substantially similar to recovery times of retinotectal axons recorded at the lateral tectum.

Interpretation. These responses are due to the direct activation of axons running between the retina and the medial tectum, as judged from their fixed latency and brief recovery times. They could therefore be recorded from retinotectal axons *or* from isthmo-optic axons. Many records preceded a tectal N-wave, which would be expected for retinotectal axons. However, it was not possible to distinguish between the two classes of axonal spike using the properties listed above. Nevertheless, they could be distinguished clearly from two classes of *somatic* spike described below. It is probable that most of the fibre spikes were from retinotectal axons, because there is a higher probability of tracks penetrating tectal laminae than the small isthmo-optic tract. The timing of axonal discharge (Fig. 2) also supports this conclusion. For both at the lateral tectum and at the medial tectum the fibre spikes show similar latency distributions, and precede trans-synaptic cell firing and the tectal N-wave.

Trans-synaptic cell firing. Ninety-nine cells were fired trans-synaptically but not antidromically in response to electrical stimulation of the optic nerve-head. Records were judged to be from cells because of their diphasic positive-negative conformation, large amplitude (from 0·5 to 2·5 mV) and

duration (from 1 to 2 msec), standing in contrast to axonal spikes on each count. They were judged to be trans-synaptically fired by their variable latency, long recovery time to paired stimuli, and for stably activated cells by the evidence of non-collision. None of these cells showed A–B inflexion to paired stimuli. Most of these cells were located in tectal laminae, as judged from the field potential profile or from subsequent histology.

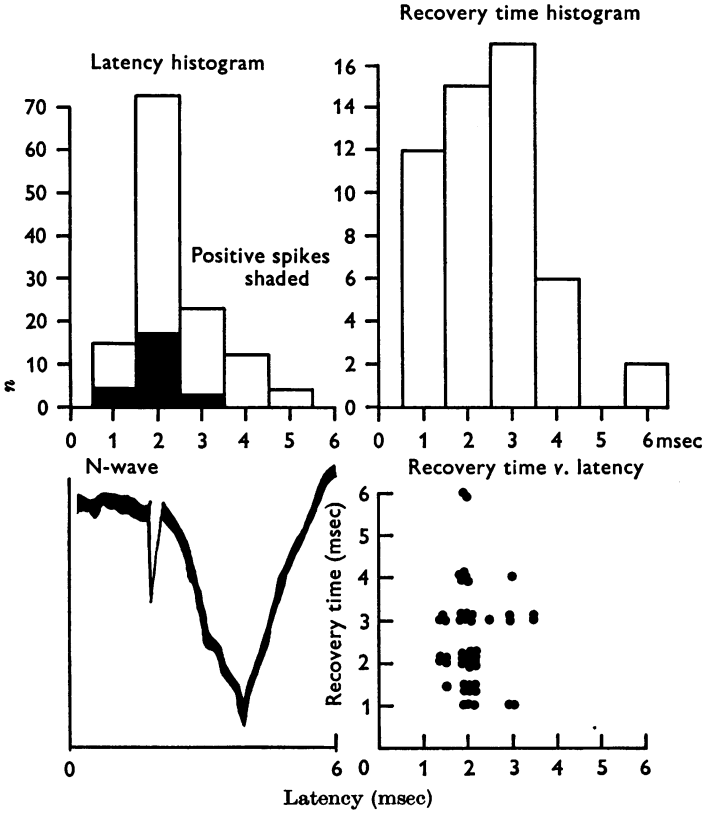

Fig. 1. Data on axonal spikes recorded on medial tracks. The upper two histograms show the distribution of latencies and of recovery times to twice-threshold stimuli. In both histograms ordinate = no. of units, abscissa = msec. The lower right-hand figure shows recovery time (ordinate) versus latency (abscissa). There is no systematic relationship between the two. The inset tracing shows a typical N-wave preceded by an axonal spike, projected to the same time-scale as the latency histogram above it.

Types of response. The commonest type of response was for the cell to fire one spike for each stimulus to the optic nerve-head, at a stable though not fixed latency. Even the most stably activated cells could be proved to be trans-synaptically activated by the evidence of non-collision

(see Holden, 1968*b*). Many showed a transient reduction in background firing following each response to retinal stimulation, suggesting the operation of a constraint to repetitive firing such as a membrane after-hyperpolarization, or the action of afferent inhibition.

Timing of trans-synaptic cell discharge. The latency histogram of cell firing is shown in Fig. 2, which allows comparison of the timing of cell

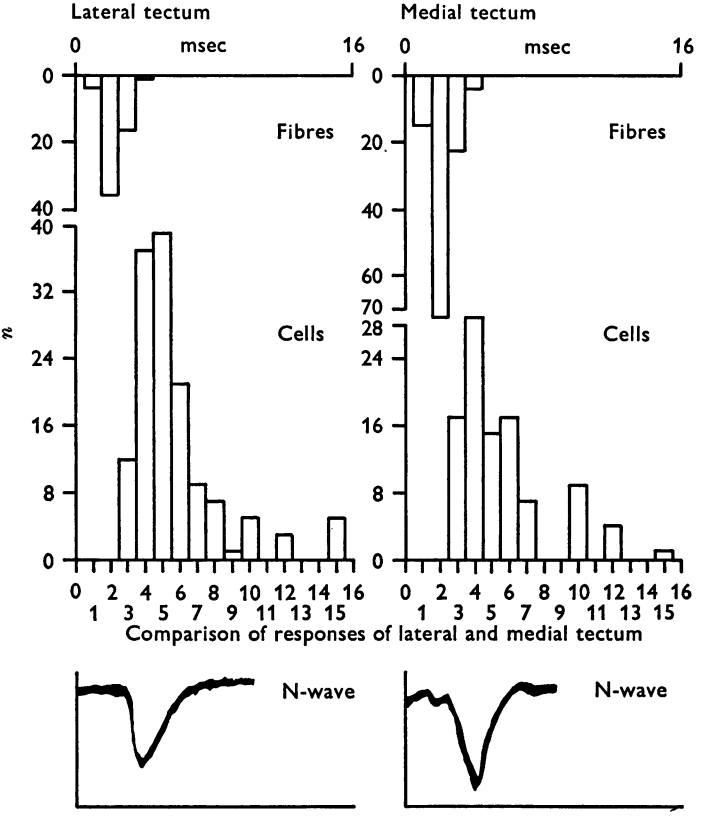

Fig. 2. Summary of timing of unitary firing of axons and of trans-synaptically fired cells at the lateral and medial tectum. In both situations the axonal spikes occupy the same intervals, and the most heavily sampled interval, at 2 msec, precedes the earliest cell firing. Below each histogram is a tracing of the N-wave, drawn with the same time-scale as the rest of the figure. Ordinates = no. of units. Abscissa = time in msec.

discharge at the lateral tectum. The earliest cell discharges in both groups fall in the 3 msec interval, and are probably monosynaptically activated. This suggests that the fastest conduction velocities of axons running from the optic nerve-head to the medial tectum (serving the lower retina) are comparable to those of axons running from optic nerve-head to the lateral

tectum (serving the central retina). The distributions are similar in skew and range.

Antidromically activated cells. Records were taken from ninety-four antidromically activated cells. Their properties are dealt with in detail below, so are only summarized here. Like the axonal spikes they were fired at short fixed latency, and showed short recovery times. But unlike the fibre spikes they showed a typically *somatic* diphasic positive–negative conformation, a larger amplitude and a longer duration. Unlike both fibre spikes and tectal cells, they showed A–B inflexion to paired stimuli. Unlike the positive fibre spikes they could be held stably for long periods, and showed injury firing to electrode movement. Many were fired at short latency trans-synaptically from the lateral tectum, while one-fifth were fired both antidromically and trans-synaptically from the optic nerve-head (differing in both respects from fibre spikes and from tectal cells). Antidromic cell responses were larger and stabler than tectal cell responses, which would be expected since cells in the isthmo-optic nucleus are larger than most tectal cells. Finally, it proved possible to interact their antidromic discharge with trans-synaptic firing to provide collision evidence.

Tectal tracks

Just as at the lateral tectum, penetration of tectal laminae showed a regular sequence of field potential profiles in response to optic nerve-head stimulation. The first event, in superficial laminae, was a graded negative field potential, the N-wave. Further descent encountered a diphasic positive–negative reversal of the wave-form, which converted into a positive-going P-wave.

Fixed latency fibre spikes were found in the N-zone and R-zone, but not in the P-zone. Trans-synaptically fired cells were found in all three zones. The timing and recovery times of cell discharge showed a differentiation between the N-zone and P-zone. The earliest cell discharge, in the 3 msec interval, was confined to the N-zone, while recovery times in the P-zone were longer than in the N-zone. Thus cell responses in the medial tectum show the same kind of radial organization as at the lateral tectum: superficial laminae are activated earlier and recover sooner than deep laminae. It should be emphasized that antidromically fired cells were never observed in an N-wave or R-wave. This would be expected, since the isthmo-optic nucleus is not located *in tectal* laminae, while these two wave forms are localized to tectal laminae.

Histological verification of tectal tracks. Tectal response profiles were recorded on ninety-seven of 165 tracks made in this experimental series. In several experiments in which only tectal profiles were recorded the brain was dissected post mortem to verify that the pipettes were placed in

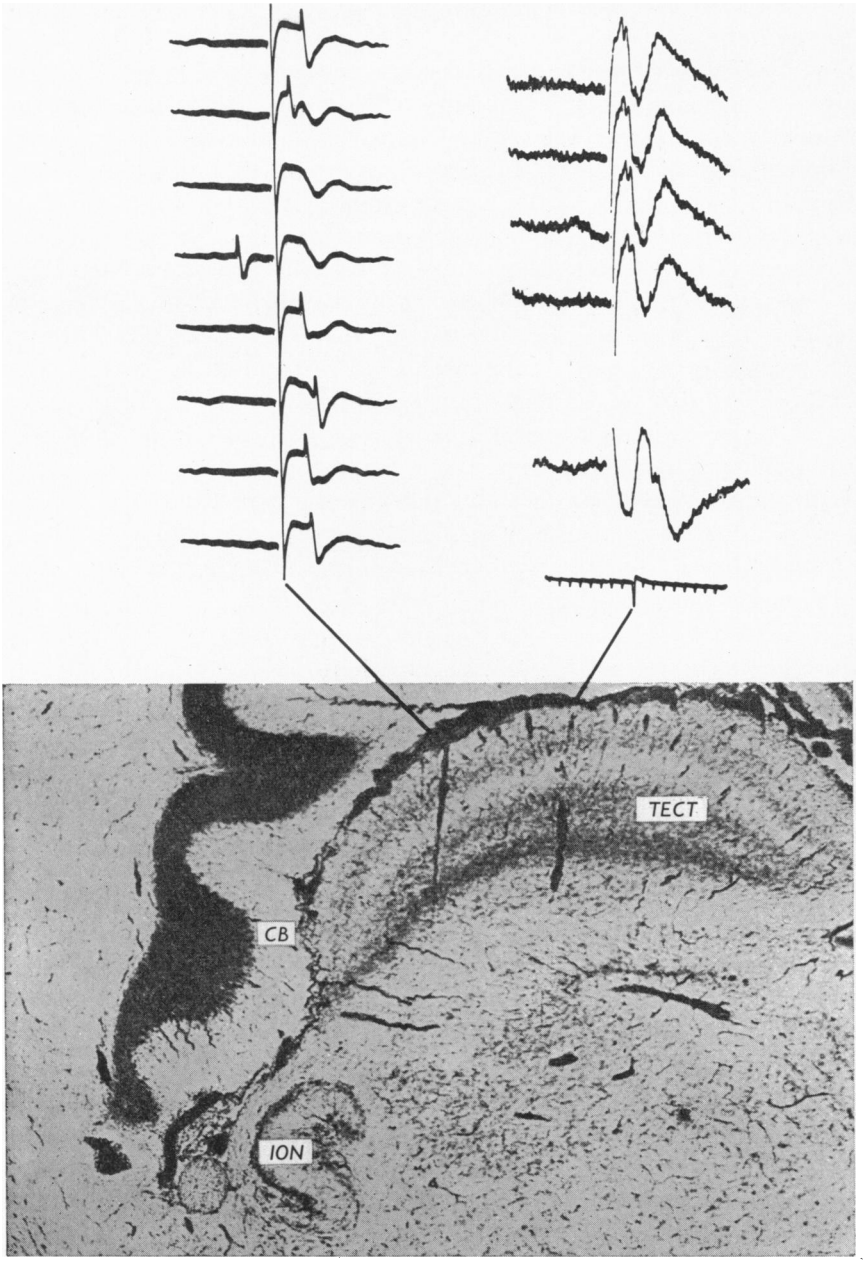

Fig. 3. Histological verification of two tectal tracks. The photomicrograph shows a sagittal section of the brain, with the tectal laminae as the crescent-like sweep occupying the upper third of the area photographed, rostral to the cerebellum. The insets show a typical N-wave recorded on the rostral track, which reverses polarity to yield a P-wave deeper in the track. For the caudal track the inset shows a tectal cell fired at short latency in the N-zone. Abbreviations: *TECT* = tectum; *CB* = cerebellum; *ION* = isthmo-optic nucleus. Time-marker shows msec.

the tectum. Histological controls were also made, in which the pipettes were pushed deep to the recording site to produce prominent tracks. Figure 3 shows a sagittal section from a brain in which two successive tracks encountered tectal profiles. Responses recorded on each track are included in the figure. The tectum lies immediately anterior to the cerebellum. The anterior track shows a typical N-wave, with reversal to the P-wave deeper in the track. The posterior track shows the same sequence, and the inset shows a trans-synaptically fired cell recorded in the N-zone.

Histological verification of tracks yielding antidromically activated cells

Ninety-four antidromically activated cells were recorded, on twenty-five of the 165 tracks aimed at the isthmo-optic nucleus. Histological verification was attempted for seventeen of these tracks. Pipettes were pushed deep and left *in situ*. This provided tracks that could be traced and photographed relatively easily. Figure 4 illustrates typical results produced in this way. It shows a track entering the dorsal pole of the isthmo-optic nucleus, and passing through both cell layers. This track recorded six antidromically activated cells, and the antidromic field potential which characterizes the nucleus (see below). In total, thirteen of the seventeen tracks could be traced to the isthmo-optic nucleus. These tracks yielded sixty-eight of the ninety-four antidromically activated cells. Thus this series can provide no evidence for centrifugal cells located in the *tectum*. All of the antidromically activated cells that could be verified were located in the isthmo-optic nucleus.

Tracks entering the isthmo-optic nucleus

A characteristic event enabling recognition that entry of the isthmo-optic nucleus had taken place was a fixed latency field occurring in response to stimulation of the optic nerve-head. This showed a prominent positive-going phase at a latency of 2–2·5 msec, rising to a peak at 3·0 msec, with a duration in the order of 3 msec. It was a graded response, and could follow repetition frequencies up to 500/sec. It was strictly localized in depth to positions where antidromically activated somata were encountered, and was due to the antidromic activation of the isthmo-optic nucleus. For this reason it was called the isthmo-optic A-wave. The A-wave could be distinguished from a tectal P-wave by several criteria. It showed a more spike-like conformation, and a briefer duration. It was usually encountered abruptly after traverse through the cerebellum, and never emerged after descent through a tectal N-zone and R-zone. It was strictly localized in depth, whereas the tectal P-wave was not. A tracing of the A-wave is included in Fig. 5.

Figure 5 shows the latency histogram for antidromically activated units.

An estimated conduction distance of 1·6 cm gives most of the sample conduction velocities ranging from 16 to 3·3 m/sec. The conversion factor of 3·2 m/sec/μ that applies to the monkey optic nerve (Ogden & Miller, 1966) gives the isthmo-optic tract a range of axonal diameters of 5–1 μ.

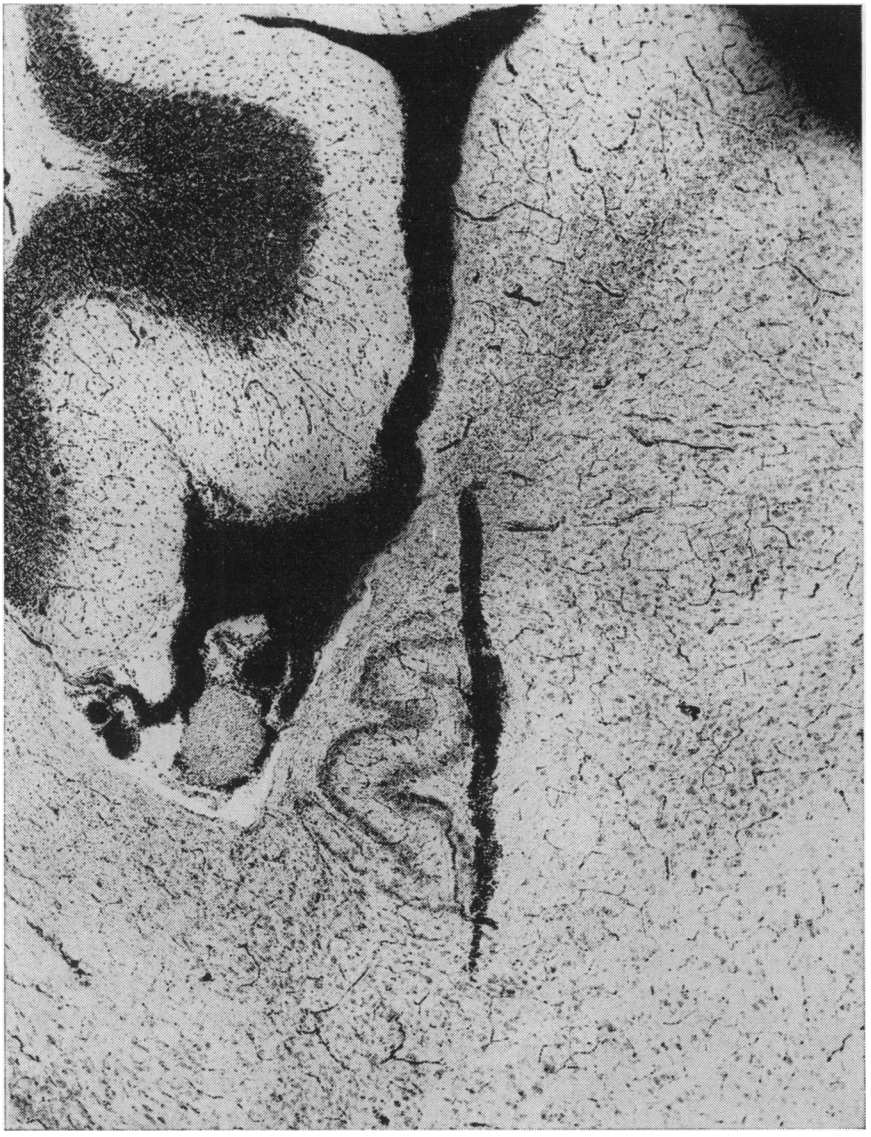

Fig. 4. Histology of a track entering the isthmo-optic nucleus. To produce this track the pipette was pushed deep and left *in situ* for several hours. Six antidromically activated cells and the A-wave (see text) were recorded upon this track.

A comparison with retinotectal axons (see Fig. 2) suggests that there may be a greater temporal dispersion in the isthmo-optic tract than in the retinotectal pathway. There is good agreement between the peak of the latency histogram and the peak of the A-wave, which also suggests that the A-wave is simply the field potential resulting from invasion of somata in the nucleus.

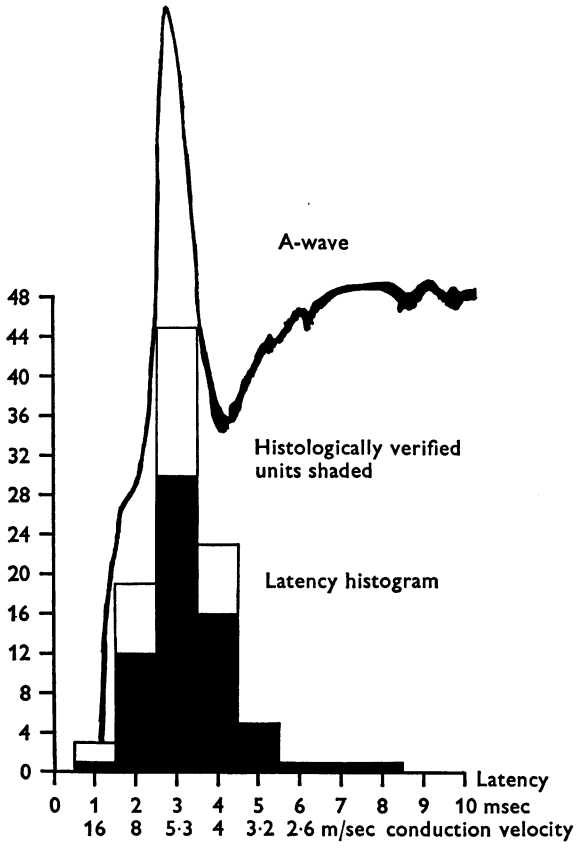

Fig. 5. Latency histogram of the antidromic activation of the isthmo-optic nucleus. Ordinate = no. of units, abscissa = latency in msec. Estimated conduction velocities corresponding to the first six response latencies are included under the histogram. An isthmo-optic A-wave has been projected upon the figure; its peak corresponds to the most heavily sampled response interval.

Somatic records. Particular care was taken to characterize the responses of somata in the nucleus, in order to distinguish between these units and axonal spikes. Figure 6 illustrates a unit in the isthmo-optic nucleus. The spike has a diphasic positive–negative conformation, with an inflexion on the rising phase. To closely paired stimuli the inflexion becomes more

prominent, with frank division into A and B components, and occasional refractoriness of the B component leaving the A component in isolation. This behaviour is typical of somatic records in a variety of C.N.S. sites (see Coombs, Curtis & Eccles, 1957; Phillips, Powell & Shepherd, 1963), and permits a confident identification of the unit as a somatic record. Ninety-four of the antidromically activated units were judged to be somatic in

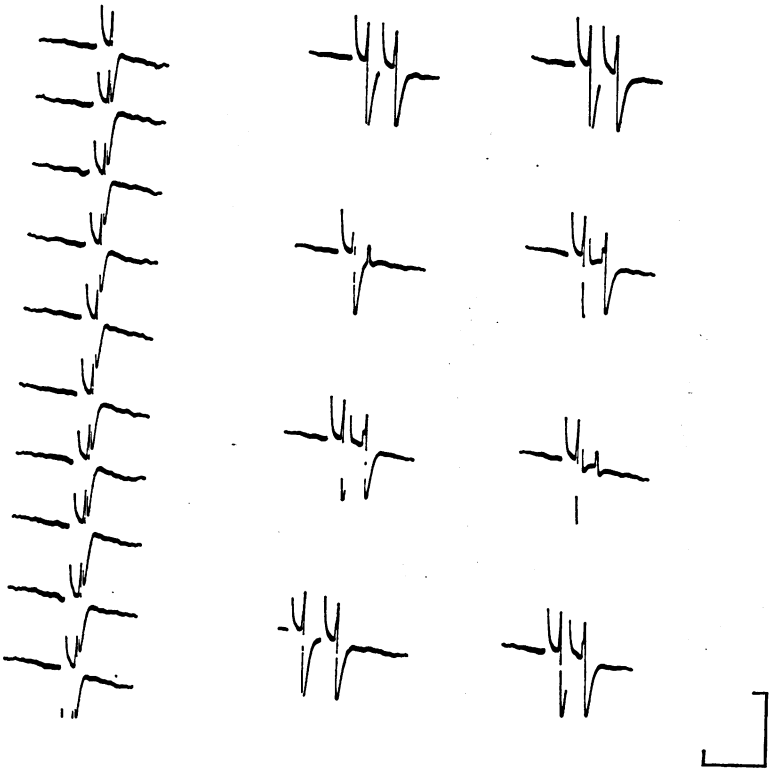

Fig. 6. Responses of an antidromically activated somatic spike recorded in the isthmo-optic nucleus. Calibration shows 5 msec and 2 mV. The spike has a fixed latency and a diphasic positive-negative conformation. To closely paired stimuli there is hesitation of the rising phase, with complete failure of the B component in two of the cases illustrated.

nature. Four were negative-going spikes of small amplitude. These were recorded at a location where the A-wave and where somatic spikes could be recorded, and probably represent axons running within the isthmo-optic nucleus.

Recovery times. The recovery times to paired twice-threshold stimuli were measured for forty-seven antidromically activated cells. Forty of the recovery times fell between 1 and 4 msec, a range similar to that of the

directly activated axons at the lateral tectum. None of the cells showed a brief recovery time coupled with a period of blocking of antidromic invasion at a longer inter-stimulus interval. Seven recovery-times exceeded 8 msec. These could be due to cell damage, or to after-hyperpolarization or trans-synaptic inhibition.

Collision evidence. Collision evidence is obtained by interacting an anti-

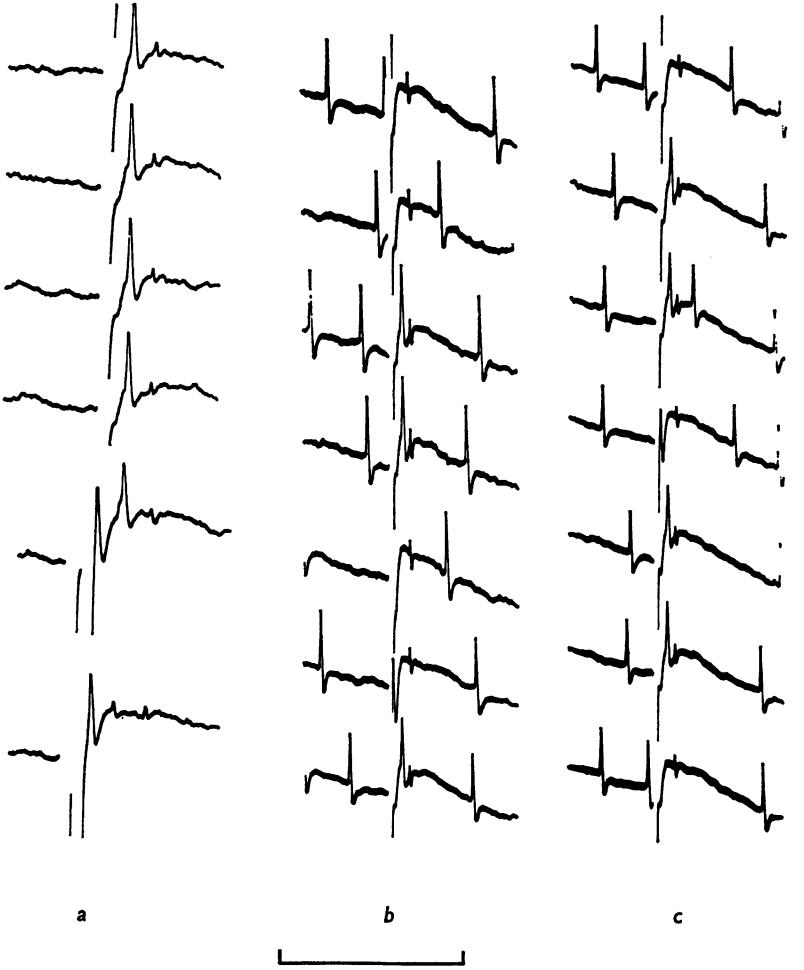

Fig. 7. Collision evidence for a cell in the isthmo-optic nucleus, obtained by interacting the antidromic response with background firing. Calibration shows 10 msec for column (*a*) and 25 msec for (*b*) and (*c*). Collision occurs in sweeps (*b*) 1, 2, 4 and 6 and in (*c*) 1, 4 and 7, whenever an impulse due to background firing precedes the antidromic impulse by an interval of 5·6 msec or less. See text for details. This establishes that the impulses due to background firing travel centrifugally to the optic nerve-head.

dromic response with orthodromic firing. In the isthmo-optic nucleus it was obtained in two ways, by interacting an antidromic spike with background firing of the unit concerned, or by interacting trans-synaptic firing produced in response to electrical stimulation of the lateral tectum with the antidromic response. Collision obtained in the first way is illustrated in

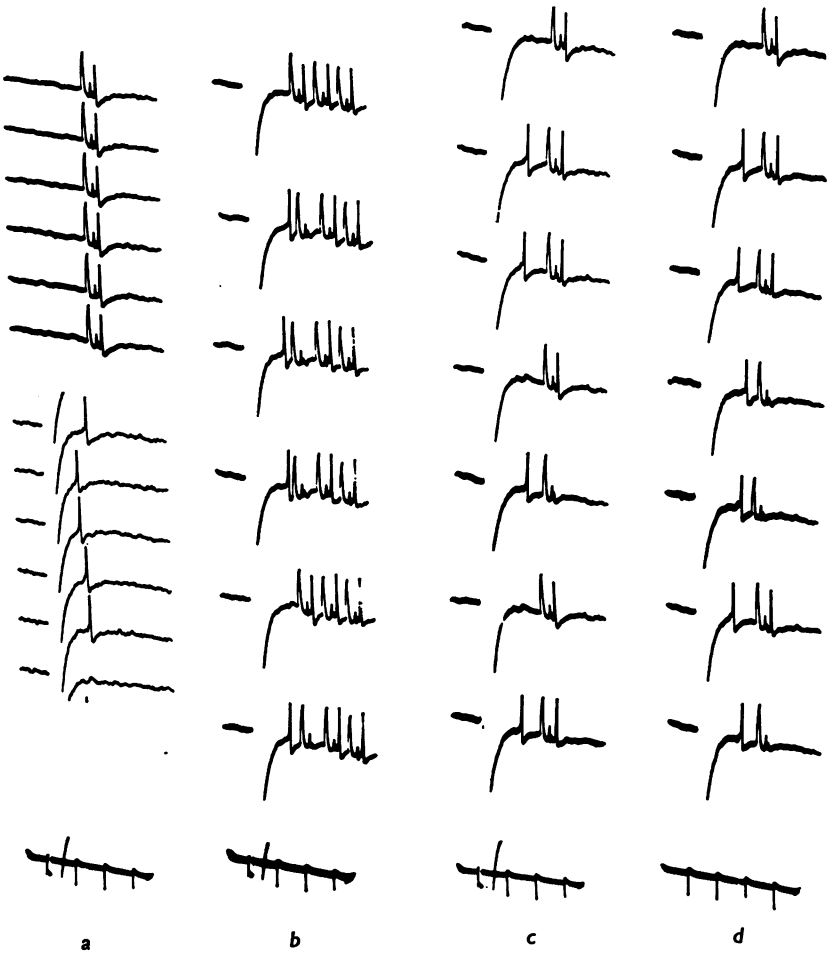

Fig. 8. Collision evidence produced by interacting an antidromic response (shown in the upper part of column *a*) with a trans-synaptic response to electrical stimulation of the lateral tectum (shown in the lower part of column *a*). Column *b* shows a train of three antidromic responses. In sweeps 2, 3, 4 and 6 the first of these is removed by a preceding trans-synaptic response. Columns *c* and *d* show a single antidromic response interacting with the trans-synaptic response. Collision occurs in sweeps *c* 5 and *d* 4, 5 and 7. The collision interval is 11 msec. See text for details. Time-markers show 10 msec. This establishes that the trans-synaptic response is conducted centrifugally to the optic nerve-head.

Fig. 7. Column (*a*) shows a diphasic cell response fired antidromically at a fixed latency of 2·2 msec from the optic nerve-head. The recovery time to paired twice-threshold stimuli was 1·2 msec, and is illustrated in the last two sweeps. Columns (*b*) and (*c*) show the inaction of this antidromic response with background firing. It can be seen that the antidromic response is absent whenever preceded by an orthodromic spike in an interval shorter than 5·6 msec. When produced in this way the blocking of antidromic firing cannot be due to refractoriness, for the recovery time to paired stimuli is only 1·2 msec. This type of collision establishes that the orthodromic impulse travels from the recording site in the isthmo-optic nucleus to the stimulation site at the optic nerve-head. The duration of the block is occupied by conduction away from the soma, axonal refractoriness, and conduction back to the soma in the antidromic direction.

Collision obtained by interacting an antidromic response with the response to electrical stimulation of the lateral tectum, thereby exciting the cell trans-synaptically, is shown in Fig. 8. The antidromic response of a large diphasic cell spike is shown in the upper part of column *a*. It occurs following the smaller response of another unit. The same cell is fired trans-synaptically from the lateral tectum, at a variable latency of 12–15 msec. In column *b* the tectal stimulus precedes a train of three stimuli to the optic nerve-head, each of which can evoke an antidromic response. Yet when the trans-synaptic discharge occurs and falls into an interval of 11 msec preceding the first antidromic response, that response is blocked. In this case the antidromic latency was 4 msec, and the recovery time to paired stimuli was 3 msec, giving a calculated collision interval of 11 msec. Column *c* shows trans-synaptic firing preceding a single antidromic discharge. Collision occurs whenever the inter-response interval is shorter than 11 msec. Here again this evidence establishes that the trans-synaptically evoked discharge travels centrifugally from the isthmo-optic nucleus towards the optic nerve-head.

DISCUSSION

This paper has shown that penetrations of the isthmo-optic nucleus can be recognized by the distinctive A-wave in response to retinal stimulation, and by the isolation of antidromically activated output cells. Penetrations of tectal laminae close to the nucleus can also be identified, and resemble tectal response profiles at the lateral tectum (Holden, 1968*a*, *b*). Properties of output cells enable them to be distinguished both from directly activated axonal spikes and from trans-synaptically activated tectal cells.

Having identified output cells of the isthmo-optic nucleus by antidromic activation, it is possible to use the collision technique to demonstrate conclusively one feature of their response. When they generate

action potentials, conduction takes place centrifugally from the cell soma towards its terminals in the retina. Thus a physiological proof that the isthmo-optic pathway is centrifugal can be added to its full anatomical description (Cowan *et al.* 1961; McGill, Powell & Cowan, 1966*a*, *b*).

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