J. Physiol. (1963), **168**, *pp.* 205–218
With 7 text-figures
Printed in Great Britain

OSCILLATORY POTENTIALS IN THE VISUAL SYSTEM OF CATS AND MONKEYS

BY R. W. DOTY AND D. S. KIMURA*

From the Center for Brain Research, University of Rochester, Rochester, New York, U.S.A.

(*Received* 21 *November* 1962)

Intense illumination of the eye often evokes from it a dramatic series of electrical oscillations. Such rhythms are seen in both vertebrate and invertebrate eyes, but there is no certainty that their origin is the same in each case. Comprehensive reviews of work on rhythmic activity in relation to the electroretinogram (e.r.g.) are available (Kohlrausch, 1931; Jolly, 1936; Granit, 1947).

These oscillatory potentials were discovered by Fröhlich (1914) using the octopus *Eledone moschata*. Eyes from *Eledone*, and sometimes from *Octopus vulgaris*, while in good condition and at temperatures below 15° C, yield sinusoidal oscillations from about 20 to 90/sec upon illumination, and from 20 to 40/sec for up to 6 min after cessation of illumination. Amplitude and frequency increase with increasing light intensity. Some of the essential features of Fröhlich's observations have been confirmed with micro-electrodes in the optic nerve of the squid (MacNichol & Love, 1961).

In the optic nerve of the eel Adrian & Matthews (1928) found oscillations apparently similar to those seen by Fröhlich. As in *Eledone*, the frequency increases with the intensity of the illumination, but changes greatly during the course of a 'response'. Upon onset or cessation of illumination oscillations beginning at 25/sec gradually fall to 6/sec after 1–2 min. Strychnine applied to the eye induces 2–4/sec oscillations in the absence of light. Since unit activity could be seen in phase with the oscillations, the latter were attributed to synchronized bursts of action in optic nerve fibres (Adrian & Matthews, 1928). Chaffee & Sutcliffe (1930) supported this conclusion since they could record the oscillations but not the e.r.g. from the optic disk of the frog. Volkmer (1956/57) found for the frog that the frequency remains constant despite great changes in light intensity and that the oscillations can be detected with intensities too low to elicit the usual e.r.g. Oscillations at about 25/sec are seen in the e.r.g. of the

* Present address: Biosciences Dept. Aracon Laboratories, Concord, Mass., U.S.A.

turtle (Noell, 1951; Armington, 1954). Their frequency is independent of intensity and colour, but their amplitude is greater with stimulation at the red end of the spectrum, suggesting that they arise from a photopic system (Armington, 1954). With intraretinal recording in the bullfrog by Tomita & Funaishi (1952) and in the frog by Brindley (1956), the oscillations were largest in the region of the bipolar cells.

Granit (1933) was the first to observe such oscillations in mammals. Recording from the optic nerve of decerebrate cats he found the usual frequency range to be 100–150/sec for 'on' and 'off' responses. They are quickly abolished by ether anaesthesia or carotid occlusion. As in the frog, these oscillations change in amplitude but not in frequency with changes in light intensity. Noell (1951, 1953) confirmed these observations and extended them to the monkey, rabbit and pigeon, Dodt & Wirth (1953) have also observed oscillations at 100/sec in the e.r.g. of pigeons, and they occur at 140–160/sec in the e.r.g. of man (Cobb & Morton, 1954; Bornschein & Goodman, 1957).

Oscillatory potentials are prominent in the optic ganglia of insects (Adrian, 1937; Crescitelli & Jahn, 1939; Roeder, 1939; Bernhard, 1942; Burkhardt, 1954) but they cannot be recorded from the isolated eye (Bernhard, 1942). The rhythms may originate and persist in the dark, and in some cases are favoured by damage.

The experiments reported here continue the exploration of the characteristics and origin of these oscillations in the optic system of mammals.

METHODS

The experiments were performed on 22 cats; 12 under Nembutal (sodium pentobarbitone; Abbott Laboratories) anaesthesia, 10 unanaesthetized after mid-pontine pre-trigeminal transection (Batini, Moruzzi, Palestini, Rossi & Zanchetti, 1959). Pertinent observations have also been made on many other cats, squirrel monkeys (*Saimiri sciureus*) and pigtailed or cynomolgus macaques (*Macacus nemestrina* and *Macacus irus*) used in other experiments. The mid-pontine pre-trigeminal transections (Batini *et al.* 1959) were made within 10 min after intravenous administration of a short-acting barbiturate anaesthetic thiamytal (Surital; Parke Davis). The floor of the fourth ventricle was exposed by retracting or removing the ventral posterior portion of the cerebellum to reveal the lingula. Complete transection was made with an angled, blunt blade at or anterior to the posterior border of the lingula, passing fully through the entrance zone of the trigeminal nerve and into the middle or anterior pons. Such a transection totally isolates the forebrain from painful stimuli. Cutting the trigeminal zone was always accompanied by vigorous contraction of the temporal muscle, and this served as an additional indication of the correctness of the level of transection. This was also true for 3 cats in which the transection was made electrolytically. Artificial respiration was utilized for a few hours after the cut was made.

Subcortical recording was accomplished with stereotaxically placed bipolar electrodes made from 0·3 mm diameter platinum-plated nichrome wires with uninsulated tip separation of about 1 mm. Placements were histologically confirmed, and were usually made so that only one electrode of the pair entered the structure being studied, e.g. optic tract.

Recordings were sometimes obtained from the ventral surface of the chiasma, optic nerve and tract after unhinging the lower jaw and drilling through the presphenoid bone. In such cases the cisterna magna was opened to curtail accumulation of cerebro-spinal fluid in the chiasmal opening. The pupils were dilated with 2 drops of a 0·1 % solution of hyoscine. One eye was stimulated at a time, the other being covered with an opaque patch. The animals were stimulated while in the dark but no effort was made to maintain dark-adaptation. The procedure for opening the eye for electrical stimulation, recording and drug application has been previously described (e.g. Doty & Grimm, 1962).

Light flashes of 10–500 msec duration were normally given at a rate of 0·3/sec with a miniature tungsten filament lamp (General Electric Co. No. 327; Doty, 1958), 4 mm in diameter, 1 cm or less from the eye. The flashes generated when a 60 V pulse is applied to this 28 V lamp have a luminance of about 12700 cd/m^2, beginning in up to 5 msec after the onset of the voltage pulse and reaching its peak within about 20 msec. A Grass photic stimulator, delivering 10 μsec flashes from a xenon gas strobotron, was also used in some experiments. Measurements indicate the peak retinal illuminance of the latter source to be 10^4–10^5 times that of the tungsten source under the conditions in which they were used. Most of the difference in the latency of responses to these two types of flash must be ascribed, however, to the slow onset of incandescence with the tungsten source.

Four different pre-amplifier, multichannel CRO set-ups have been used, including Grass and Tektronix equipment, with frequency response flat from 0·8 to 10,000/sec.

RESULTS

Responses to illumination

General characteristics. Provided that the animal is in good condition and not too deeply anaesthetized and that the recording electrode has not produced much damage, the optic tract responses shown in Figs. 1–6 can always be recorded following a brilliant flash. The initial deflexion is invariably positive and ranges from 100 to over 1000 μV, depending upon intensity and abruptness of the flash. In most cats this initial deflexion is composed of two closely approximated waves (Figs. 1 and 4) or can be resolved into such a doublet by various procedures. The subsequent oscillations range in frequency from about 50/sec in animals under deep barbiturate anaesthesia to about 160/sec in unanaesthetized animals. The frequency is often unchanged throughout an experiment of many hours duration or even following moderate doses of barbiturate anaesthetics (Fig. 6*G*), and is definitely not a function of intensity (Fig. 1).

It is remarkable that this complex series of potentials is at any given moment exactly reproducible. Note, for instance, the diminished eighth wave in Fig. 1*B* (counting the first as a pair; also distinguishable on original records of Fig. 1*A*), or the consistent differences in the first few waves for flashes to right versus left eyes in Fig. 2. Responses in the two optic tracts are very similar for stimulation of one eye. As a flash is continued beyond some 50 msec the oscillatory response to it falls off in amplitude (Fig. 3*B*, *C*). Usually by 500 msec duration the rhythm is lost. Longer flash durations have not been studied adequately. With 500 msec

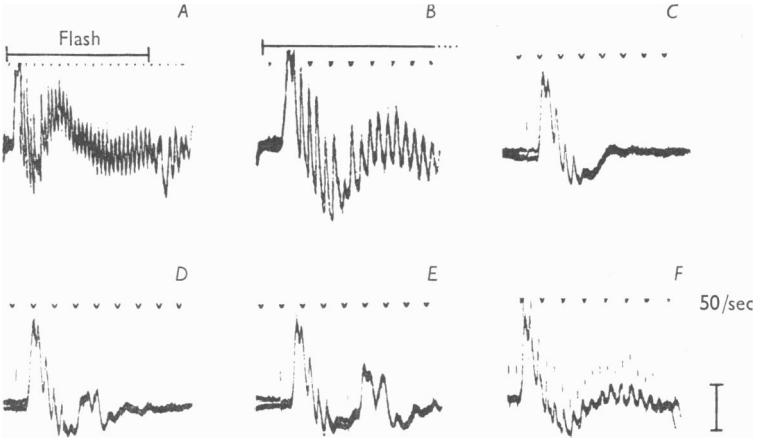

Fig. 1. Oscillations in optic tract. Cat 632. Nembutal, chiasma exposed ventrally, recorded shortly after penetration of tract with needle electrode contralateral to stimulated eye. Sweeps superimposed in each record. *A*, *B*, 350 msec flash of tungsten lamp, oscillations at 100/sec while light is on, latency 22 msec. *C–F*, 10 μsec strobotron flash, latency 10 msec, 5 cycles of oscillation identical in frequency with those in *A* and *B*. *C*, single flash; *D*, second flash after 45 msec recovery. *E*, 65 msec recovery. *F*, burst of flashes at 140/sec does not greatly disturb rhythm to first flash. Time marker, 50/sec. Amplitude calibration 250 μV for *A* and *B*, 500 μV for *C–F*. Positivity of active electrode represented by upward deflexion in all figures.

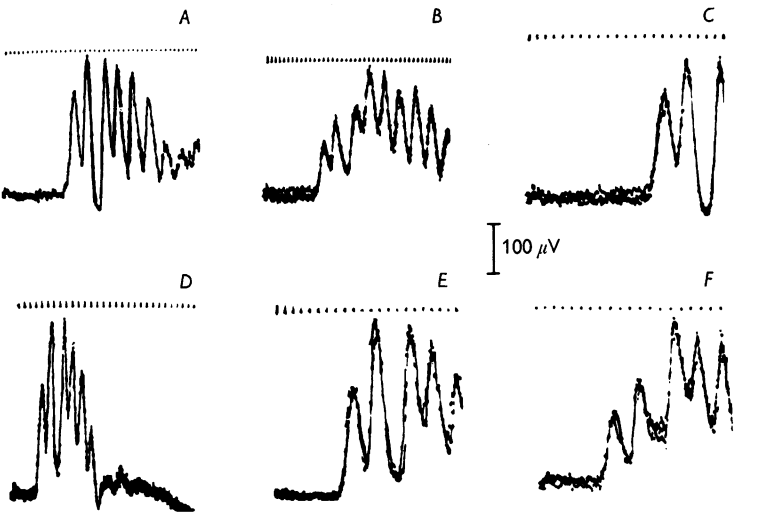

Fig. 2. Oscillations in optic tract. Squirrel monkey 73, light Nembutal anaesthesia, left tract at A 6·5, L 7, −1. *A–C*, 200 msec flash of tungsten lamp, 24–27 msec latency; *D–F*, 10 μsec strobotron flash, 13 msec latency. *B* and *F* to right eye, others to left eye. Time marker, 500/sec, except 250/sec in *D*. A maximum of 9 oscillations was obtained with 200 msec flash at this level of anaesthesia. Pattern of oscillations differs reliably for the two eyes in ½ hr period of these records and the initial components are nearly identical for each eye for the two different types of flash. Frequency of oscillations in latter portion of *B* is about 140/sec.

flashes in unanaesthetized preparations the rhythm is sometimes broken after the first 4–8 oscillations and then returns after some 50–100 msec. With increasing Nembutal anaesthesia it is the later oscillations which are lost first (Fig. 6) and with moment-to-moment reproducibility the later pattern may become one of alternating large and small waves or waves of irregular amplitude. In the deepest levels of anaesthesia nothing but a single or double wave is seen as the 'on' response.

Under all conditions there is a distinct tendency for the amplitude and regularity of even the first few waves to be augmented by repeated flashing (0·3/sec), so that the responses to the first 1–3 flashes are often different from subsequent ones. With cats under Nembutal anaesthesia

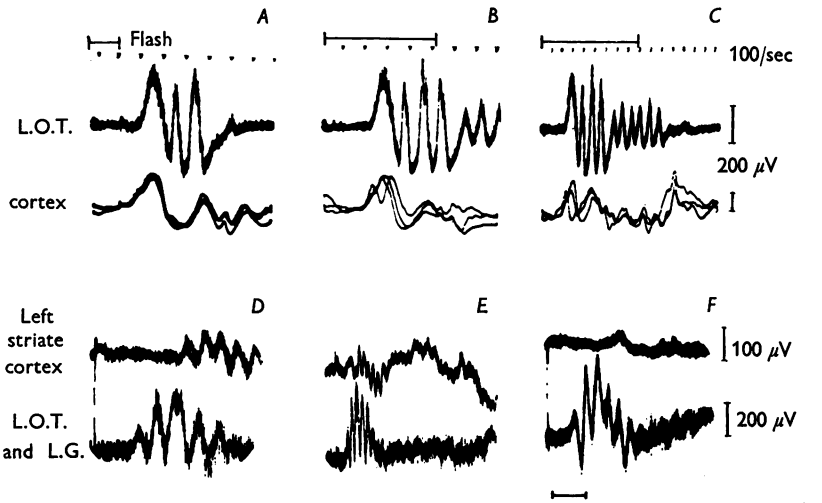

Fig. 3. Relation of oscillations to cortical evoked potentials. Records *A–C*: Cat C-89, mid-pontine pretrigeminal preparation, tungsten lamp, pre-amplifier filters set to eliminate frequencies below 80/sec for left optic tract (L.O.T.). Flashes to right eye: *A*, 12 msec; *B*, 50 msec; *C*, 90 msec. Latency 22 msec for 'on', same for cessation of oscillations at 'off'; oscillations 125/sec. Complex cortical response shows very limited relation to tract oscillations although it is obviously multiple. Records *D–F*, Squirrel monkey 8, 18 days after implantation of electrodes. The pair for recording the upper trace was situated with one electrode at the edge of striate area 8 mm from the mid line and the other 3 mm anterior to it. Lower trace from electrodes at lateral edge of anterior pole of left lateral geniculate nucleus. Strobotron flash, pupils not dilated. Trace superimpositions *D* and *F*. For *D* and *E*, animal alert and free to close eyes or change direction of gaze; *F* at a surgical level of Nembutal anaesthesia. In *D* and *E* cortical oscillations at 200/sec are faster than the same group at about 160/sec in tract. With anaesthesia (*F*) the oscillatory frequency has fallen to 140/sec and the latency for tract response increased from about 11 msec (*D*) to 13 msec (*F*). Note lag of 15 msec between tract and cortical response in *D*, about 20 msec in *F* with loss of cortical oscillations in the anaesthetized state. Time marker: *A–C* 100/sec; *D*, 10 msec; *E*, 40 msec; *F*, 20 msec.

the first 3–5 waves can follow frequencies of strobotron flashing up to about 5/sec and they may be enhanced at frequencies of 3/sec. A strobotron flash given during the course of oscillations to a continuing tungsten flash has almost no effect. In the unanaesthetized monkey provided with permanently implanted electrodes the oscillations appear after each flash even at 20 flashes/sec.

In many instances the only 'off' response of note in the tract is the cessation of the oscillations (Fig. 3*C*). When a distinct 'off' response is present, it is always smaller than the 'on'. Not uncommonly the initial 'off' elevation will be followed by a few waves at about half the frequency of the 'on' oscillations (Fig. 1*A*).

Control procedures. The oscillations seen following a 10 μsec flash discount the possibility that fluctuations in the output of the tungsten filament lamp normally used might be responsible for the rhythms observed. Such explanation is further eliminated by the absence of oscillations in photocell records of the tungsten lamp's output and by production of the usual oscillations in the optic tract with flashes from a battery-operated lamp.

An electrode resting lightly upon the ventral surface of the uninjured optic nerve, chiasma or tract records the same wave forms and the same polarity of response as an electrode thrust into their substance. The surface recordings are, understandably, less than half the amplitude of intra-tract recordings, because of the high-resistance connective-tissue sheath investing the optic paths and some inevitable shunting by cerebrospinal fluid. For perhaps less than 1 min after inserting an electrode into the exposed tract the amplitude is as much as double that which it subsequently becomes, suggesting that even slight injury diminishes the response. An electrode in the brain 1 mm directly above the tract records almost nothing of the photic response.

Components of the e.r.g. do not seem to be recorded from the optic tract with the electrode arrangement employed. Slower waves, such as those of Fig. 1, have no temporal correlation with the e.r.g. and do not attenuate with distance from the eye. Central connexions make no significant contribution to the oscillatory potentials, since the latter are unchanged in the tract or nerve after removal of the forebrain.

The oscillations can be recorded without dilating the pupil with hyoscine, though of course they increase in amplitude upon increase of effective retinal illumination. Intravenous adrenaline 0·05 mg/kg has no effect on the photic responses, nor do varying degrees of over-ventilation in the artificially respirated cat, up to 50 ml./stroke at 40/min for $3\frac{1}{2}$ min. Asphyxia produced by increased intraocular pressure (Fig. 4), tracheal occlusion or under-ventilation does not augment the oscillations. Bathing the exposed chiasmal region in an isotonic solution of sodium citrate has

no great effect. Strychnine, 0·3 mg/kg intravenously or a 0·01 % solution sprayed upon the exposed retina, augments the random background activity recorded from the tract and eliminates all rhythmical components of the photic response.

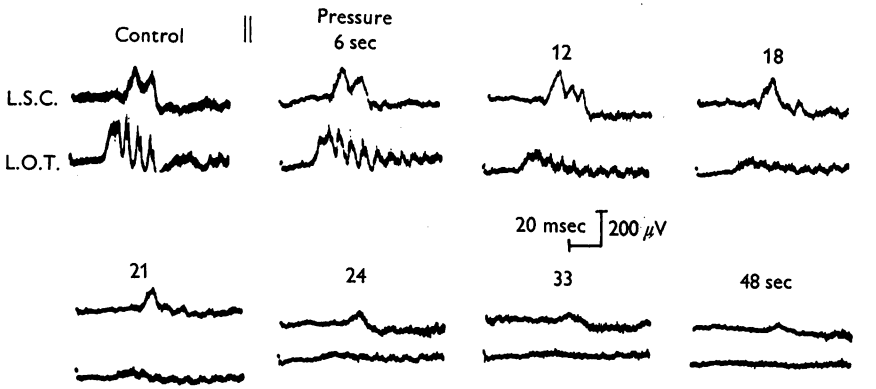

Fig. 4. Superior colliculus response and effect of increased intraocular pressure. Cat C-45, 7 hr after mid-pontine transection. Colliculus surgically exposed. Contralateral eye stimulated with 100 msec flash. Control latencies of 20 msec for tract (L.O.T.), more than 30 msec for colliculus (L.S.C.), unchanged until 18 sec after raising intraocular pressure to 200 mm Hg. Last collicular response seen at 48 sec, 24 sec after tract response appears to be absent. Tract oscillations at 130/sec diminish greatly but remain nearly unchanged in frequency.

Origin and nature of the oscillations. Electrodes at different points several millimetres apart along the tract usually record identical patterns of oscillation. Since there is thus no attenuation with distance from the retina, the oscillations must be propagated. Phase differences of the order of 1 msec in the expected direction can be seen occasionally, but the slow rise time of the potentials makes such measurement unreliable. Significant differences in pattern are sometimes seen between two tract positions. It seems likely that such differences may be attributed to the possibility of localized recording among the fascicles of fibres in the tract.

Recordings from the dorsal portion of the lateral geniculate body naturally are contaminated with a contribution from optic-tract terminals and fibres of passage. Nevertheless, there is a great difference from optic-tract responses. The initial deflexion is negative, particularly in the anterior half in the cat, and unit activity is more distinguishable than in the tract. Although oscillation seen in the tract is not faithfully followed, there is sufficient correspondence between geniculate and tract oscillations to suggest transmission from the tract.

In cats not more than three waves can be seen in the brachium of the superior colliculus. Their period is similar to the initial waves in the tract,

but their latency is about 10 msec longer. The double wave seen in Fig. 4 is more common and is characteristic of the colliculus proper. It is difficult to decide what relation the waves in the colliculus bear to those in the tract. Figure 4 indicates that at least some portion of the collicular response is carried by fibres with a negligible contribution to tract potentials.

Oscillatory potentials in the area striata are commonly seen in unanaesthetized monkeys, especially when chronically implanted electrodes are used (Fig. 3). The relation of oscillations at the cortex to those in the tract is puzzling because the frequencies are not always identical (Fig. 3). However, since the number of oscillations (when they can be detected at the cortex in these chronic preparations) is the same as in the tract, it seems likely that the cortical oscillations truly reflect action propagated in the tract. In unanaesthetized monkeys with mid-pontine pre-trigeminal transection the oscillations in the area striata may begin at greater than 200/sec and fall during a 500 msec flash to 50–100/sec, yielding an intricate yet reproducible pattern. However, the relation of these cortical oscillations to those in the tract is so inconstant in experiments so far that no conclusion is warranted.

The situation is similar in unanaesthetized cats with pre-trigeminal mid-pontine transection. Oscillations to flashes are not common in the cortical visual areas of this preparation. The response patterns are complex (Fig. 3) and not readily referrable to patterns in the tract. The second cortical wave seen in Fig. 3*A* is characteristic of the response to short flashes (20 msec or less) and often an enhanced wave in the tract can be directly correlated with it. It seems that only in this circumstance can the multiple responses of the cat's visual cortex be related with assurance to the continuing rhythms in the tract.

Direct evidence that the oscillations of nerve and tract arise as grouped discharges of retinal ganglion cells is offered in Fig. 5. With the relatively gross electrode employed it cannot be determined whether the same units discharge repeatedly at 'preferred' times, but bursts of discharge are punctuated unmistakably by periods of inhibition. Somewhat surprisingly the same rhythmic sequence of 'silence' and discharge as seen to a flash can be provoked by intracranial electrical stimulation of the optic nerve (Fig. 5*C*). This requires high-intensity stimulation of the optic nerve or tract, and the repetitive response (Fig. 5*C*) cannot be obtained if there are any signs of retinal deterioration, if the earliest response (Fig. 5*G–K*) is less than 1–2 mV, or if the stimulating electrodes are raised 1–2 mm above the optic nerve. This retinal 'after-discharge' sometimes follows a single 1 msec 15 V pulse after a latency of about 15 msec. It is overwhelmed by the response to a flash (Fig. 5*E, F*). Yet a single pulse or a burst of electrical stimuli, timed to occur about 5 msec or less before a wave in the tract, can

eliminate this wave (Fig. 6). This does not always occur, however (Fig. 5*D*, *F*). In general the rhythmic mechanism is remarkably resistant to interference by interposed optic nerve stimuli, as shown in Figs. 5 and 6.

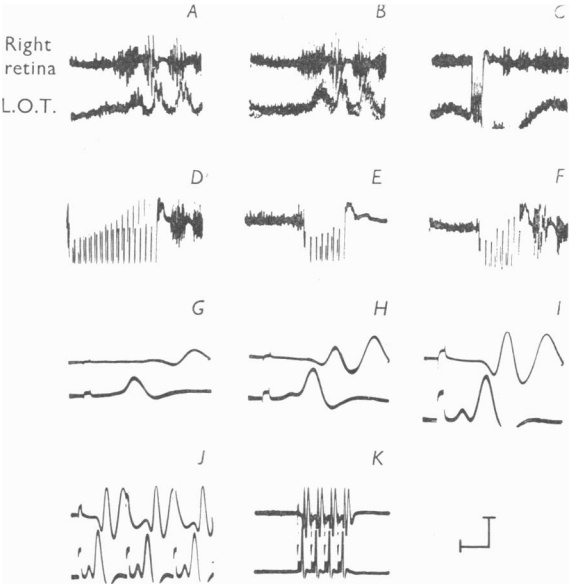

Fig. 5. Comparison of retinal and tract responses. Cat C-48, 6½ hr after mid-pontine transection, 2½ hr after opening right eye, temperature 35° C, oscillations 130/sec. Stimulation of right optic nerve with 0·1 msec pulses; 100 msec flash. Retinal recording electrodes 1·5 mm from optic disk at '10 o'clock', frequencies filtered below 80/sec and 'bipolar' recording eliminate e.r.g. *A*, *B*, controls, single and multiple sweeps respectively; note retinal action some 4 msec before that in tract. *C*, slower sweep; 4 pulses at 15 V, without flash and with precaution taken that light from CRO sweep cannot reach eye, produces rhythmic bursts of retinal action. *D*, burst at 500/sec, 15 V during latent period, eliminating most of first two retinal bursts, has no effect on subsequent action to flash. *E*, electrical pulses alone are followed by silent period, but with flash (*F*) the normal course of action breaks through this silent period. *G*, *H*, *I*, fast sweep, low gain, 3, 5 and 15 V respectively. *J*, *K*, responses follow perfectly in 500/sec burst. Amplitude calibration: *A*–*F*, 200 μV; *G*–*K*, 2 mV. Time marker, *A*, *B*, *D*–*F*, 10 msec; *C*, 20 msec; *G*–*I*, 0·4 msec; *J*, 1 msec; *K*, 4 msec.

Oscillations in the dark

Under light levels of Nembutal anaesthesia oscillations from 3 to 30/sec can be recorded from the optic tract while the animal is in the dark (Fig. 7). After a minute in the light it takes about 10 sec or more for these oscillations to develop, and they do so gradually. They are interrupted by a flash, though if the flash is brief and dim the dark rhythm is resumed within a second or so (Fig. 7*D*).

A localized retinal stimulus from a point of light does not affect the oscillations. At times it seemed as though electrical stimulation of the optic nerve might re-set the rhythm, but such reaction, if real, is minimal.

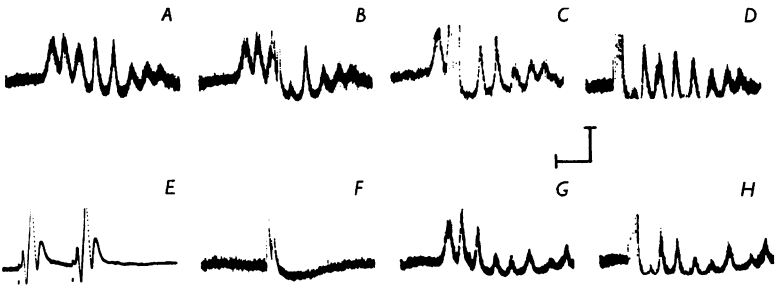

Fig. 6. Deletion of optic-tract waves without influence on rhythm. Cat C-44, 4 hr after mid-pontine transection, temperature 35° C. Electrical pulses, 15 V, 0·05 msec, to right optic nerve; 100 msec flash to right eye. Records from left optic tract with slight contamination from lateral geniculate nucleus. (Similar 'deletions' occurred, however, in right optic tract, well out of range of lateral geniculate recording.) *A*, control. *B*, 2 pulses at 300/sec nearly delete fourth wave without affecting subsequent waves. *C* and *D*, 1000/sec burst deletes third and first wave, respectively. *E*, fast sweep, low gain, two pulses at 300/sec. *F*, same, at usual gain and sweep speed. *G*, flash alone following intravenous administration of half the anaesthetic dose of Surital. *H*, same, with deletion of first wave by 4 pulses at 1000/sec. Calibration: *E*, 1 mV and 2 msec; others 200 μV and 20 msec.

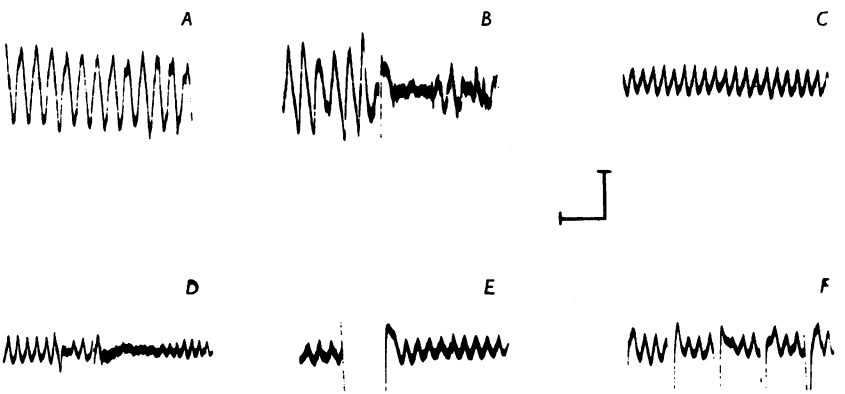

Fig. 7. Dark oscillations. *A*, *B*, Cat 632, Nembutal, presphenoidal exposure of tract. *A*, steady oscillations at about 3/sec after some 10 sec in the dark; *B*, overhead lights switched on and off momentarily ('off' indicated by large artifact), disrupting rhythm. *C–F*, Cat 987, Nembutal, also presphenoidal approach. Oscillations appearing at about 4/sec when the eye is in the dark are interrupted for some time by a 10 msec flash (*D*), but not by electrical stimulation of the optic nerve in bursts, at 500/sec, of 15 V, 0·1 msec pulses (*E*, *F*). Amplitude calibration, 200 μV: time, 1 sec.

The oscillations often wax and wane in amplitude in unpredictable fashion, and no method has been found for inducing them at will. Strychnine and asphyxia abolish them. The conclusion that they are related to barbiturate anaesthesia is indicated by two facts: (1) The oscillations in the dark are never seen in unanaesthetized mid-pontine pre-trigeminal preparations; (2) they occurred for a few minutes in a squirrel monkey with chronically implanted electrodes as the animal passed through the initial stages of anaesthesia following intraperitoneal injection of Nembutal.

It might be thought unlikely that the extremely slow, 400 μV rhythms seen in Fig. 7*A* could be produced by synchronized fibre discharges. This appears to be the case, however. If on one CRO channel the slow components are filtered out and amplifier gain increased, unit activity in the tract can be seen to increase abruptly during one phase of each oscillation concurrently recorded on another channel. The oscillations have no obvious influence on the background activity of the visual cortex.

DISCUSSION

For the normal visual system of cats and monkeys it is clear that the oscillations evoked in optic nerve or tract by intense flashes are a consistent phenomenon and arise from synchronized bursts of activity in optic fibres. The factors responsible for the rhythmic nature and the synchrony of this activity are unknown. The precision of the synchronization is difficult to judge, because differences of several milliseconds can be expected in arrival of impulses at the optic disk from various portions of the retina (Dodt, 1956). If a flash were to excite the ganglion cells nearly simultaneously and produce a continuing excitatory drive, rhythmic synchronized discharge might result simply from identity in recovery characteristics of the responding elements. Were this the case, it must also be true that most spontaneously active ganglion cells respond to the flash, since it is often found that the interburst pause is complete (e.g. Fig. 5).

Such rhythmic action has not been reported in various recent studies of single units in the visual system, but there are many obvious reasons why such a fact might not be noted. Such studies, however, could determine whether the rhythm is generated mainly by sequential action of different units or by repetitive action of a particular group. The latter seems the more likely.

Since intense antidromic stimulation does not re-set the rhythm elicited by flashes, it must be inferred either that antidromic impulses do not invade the ganglion cell bodies or that the rhythm is not set by the ganglion cells. The electrical stimulation of the nerve which does not re-set the rhythm is nevertheless able to elicit it (Fig. 5). It has also been shown

(Doty & Grimm, 1962) that electrical stimulation of the retina can elicit rhythmic action in the tract similar to that evoked by light. It is unlikely that stimulation of the optic nerve could elicit rhythmic activity solely by antidromic invasion of ganglion cells; and retinal nerve fibres lack collaterals (Cajal, 1955) to effect more complex connexions. The centrifugal fibres, which seem to end mostly about the bipolar cells (Cajal, 1955; Polyak, 1957), are thus inferred to have access to the rhythm-generating mechanism. Ogden & Brown (1962) find optic-nerve stimulation does not elicit potentials in the fovea of monkeys; hence neural reaction upon the photoreceptors can probably be excluded from consideration.

As already mentioned, flashes induce oscillatory potentials in the bipolar cell layers of the frog retina (Tomita & Funaishi, 1952; Brindley, 1956). Svaetichin (1961) using isolated retinas has extended this observation to the dog and monkey. Most of the available data thus suggest that the bipolar cells originate this 'on' rhythm. A still more peripheral origin, in the photoreceptors, may be present in the octopus. Fröhlich (1914) could obtain oscillations (although poor) from isolated retinas of *Eledone*, and Young (1962) in an admirable study now confirms earlier indications that photoreceptors and supporting elements would be the only structures operative under Fröhlich's conditions. Perhaps the supporting cells should not be overlooked, since Laufer, Svaetichin, Mitarai, Fatehchand, Vallecalle & Villegas (1961) find in the fish retina that cells which are apparently glial are capable of sustained sinusoidal oscillations in potential.

The brilliant flashes employed in these experiments produce dramatic after-images. It thus might be possible, under carefully controlled conditions, to relate the oscillatory pattern in the optic tract and cortex of primates to certain aspects of our own subjective experience. Oscillations probably identical to those studied herein have been recorded in the human e.r.g. (Cobb & Morton, 1954; Bornschein & Goodman, 1957) and from the human occipital lobe (Cobb & Dawson, 1960; Walter, 1961). Hughes & Mazurowski (1962) have also noted the oscillatory potentials recorded from the visual cortex of the unanaesthetized monkey in response to photic stimuli.

It would be of interest to understand the peculiarities of the recording situation, probably related to the impedance of glial or connective-tissue barriers, which yield such a conspicuous sum of the individual impulses in the optic nerve or tract. We cannot readily explain why the potentials observed are almost entirely positive. The 'killed-end effect' might explain the positivity in most recordings, but the positivity prevails even in records taken from the patently uninjured surface of the chiasma.

SUMMARY

1. If trauma is minimal, a bright flash evokes in the optic nerve and tract a series of rhythmic waves ranging in frequency from 50/sec with deep barbiturate anaesthesia to 160/sec in the chronically prepared, unanaesthetized monkey. In many instances the oscillations persist throughout a 500 msec flash. The frequency is independent of intensity.

2. These waves are produced by grouped discharge of retinal ganglion cells. The rhythmic discharge is apparently propagated to the striate cortex in unanaesthetized monkeys, where oscillations above 200/sec are often found, but propagation to cortex and superior colliculus in the cat is poor or absent.

3. In the cat electrical stimulation of the optic nerve can block any wave in the series without re-setting the rhythm. It can also elicit rhythmic discharge in the retina similar to that seen with flashes. From these facts it can be inferred that the rhythm does not originate in the ganglion cells.

4. Under light barbiturate anaesthesia continuous oscillations at 3–30/sec may develop after several seconds of darkness. They too represent grouped discharge in optic nerve fibres. Photic but not electrical stimulation abolishes this rhythm.

This work was begun in the Department of Physiology, University of Michigan and supported by grants B–1068 and 2B–5252 from the National Institute of Neurological Diseases and Blindness, National Institutes of Health. It has been continued at the University of Rochester under grant B–3606 from the same agency.

REFERENCES

ADRIAN, E. D. (1937). Synchronized reactions in the optic ganglion of *Dytiscus*. *J. Physiol.* **91**, 66–89.

ADRIAN, E. D. & MATTHEWS, R. (1928). The action of light on the eye. Part III. The interaction of retinal neurones. *J. Physiol.* **65**, 273–298.

ARMINGTON, J. C. (1954). Spectral sensitivity of the turtle, *Pseudemys*. *J. comp. physiol. Psychol.* **47**, 1–6.

BATINI, C., MORUZZI, G., PALESTINI, M., ROSSI, G. F. & ZANCHETTI, A. (1959). Effects of complete pontine transections on the sleep-wakefulness rhythm: the midpontine pretrigeminal preparation. *Arch. Ital. Biol.* **97**, 1–12.

BERNHARD, C. G. (1942). Isolation of retinal and optic ganglion response in the eye of *Dytiscus*. *J. Neurophysiol.* **5**, 32–48.

BORNSCHEIN, H. & GOODMAN, G. (1957). Studies of the A-wave in the human electoretinogram. *A.M.A. Arch. Ophthal.* **58**, 431–437.

BRINDLEY, G. S. (1956). Responses to illumination recorded by micro-electrodes from the frog's retina. *J. Physiol.* **134**, 360–384.

BURKHARDT, D. (1954). Rhythmische Erregungen in den optischen Zentren von *Calliphora erythrocephala*. *Z. vergleich. Physiol.* **36**, 595–630.

CAJAL, S. R. (1955). *Histologie de l' Système Nerveux de l'Homme et des Vertébrés*, vol. II, pp. 323–325. Madrid: Consejo Superior de Investigaciones Científicas, Inst. Ramón y Cajal.

CHAFFEE, E. L. & SUTCLIFFE, E. (1930). The differences in electrical response of the retina of the frog and horned toad according to the position of the electrodes. *Amer. J. Physiol.* **95**, 250–261.

COBB, W. A. & DAWSON, G. D. (1960). The latency and form in man of the occipital potentials evoked by bright flashes. *J. Physiol.* **152**, 108–121.

COBB, W. & MORTON, H. B. (1954). A new component of the human electroretinogram. *J. Physiol.* **123**, 36*P*.

CRESCITELLI, F. & JAHN, T. L. (1939). The effect of temperature on the electrical response of the grasshopper eye. *J. cell. comp. Physiol.* **14**, 13–27.

DODT, E. (1956). Geschwindigkeit der Nervenleitung innerhalb der Netzhaut. *Experientia*, **12**, 34–35.

DODT, E. & WIRTH, A. (1953). Differentiation between rods and cones by flicker electroretinography in pigeon and guinea pig. *Acta physiol. scand.* **30**, 80–89.

DOTY, R. W. (1958). Potentials evoked in cat cerebral cortex by diffuse and by punctiform photic stimuli. *J. Neurophysiol.* **21**, 437–464.

DOTY, R. W. & GRIMM, F. R. (1962). Cortical responses to local electrical stimulation of retina. *Exp. Neurol.* **5**, 319–334.

FRÖHLICH, F. W. (1914). Beiträge zur allgemeinen Physiologie der Sinnesorgane. *Z. Psychol. Physiol. Sinnesorg. II. Abt. Sinnesphysiol.* **48**, 28–164.

GRANIT, R. (1933). The components of the retinal action potential in mammals and their relation to the discharge in the optic nerve. *J. Physiol.* **77**, 207–239.

GRANIT, R. (1947). *Sensory Mechanisms of the Retina*, pp. 26–68. London: Oxford University Press.

HUGHES, J. R. & MAZUROWSKI, J. A. (1962). Rhythmical oscillatory activity in visual responses from the unanaesthetized monkey. *Physiologist*, **5**, 158.

JOLLY, W. A. (1936). On amphibian retinal currents. *Quart. J. Exp. Physiol.* **26**, 97–107.

KOHLRAUSCH, A. (1931). Elektrische Erscheinungen am Auge. *Handbuch der Normalen und pathologischen Physiologie*, ed. BETHE, A., VON BERGMANN, G., EMBDEN, G. & ELLINGER, A. Band 12, Hälfte 2, Receptionsorgane II, pp. 1394–1496. Berlin: Springer Verlag.

LAUFER, M., SVAETICHIN, G., MITARAI, G., FATEHCHAND, R., VALLECALLE, E. & VILLEGAS, J. (1961). The effect of temperature, carbon dioxide, and ammonia on the neuron-glia unit. In *Neurophysiologie und Psychophysik des visuellen Systems*, ed. JUNG, R. & KORNHUBER, H. pp. 457–463. Berlin: Springer Verlag.

MACNICHOL, E. F. Jr. & LOVE, W. E. (1961). Impulse discharges from the retinal nerve and optic ganglion of the squid. In *Neurophysiologie und Psychophysik des visuellen Systems*, ed. JUNG, R. & KORNHUBER, H. pp. 97–103. Berlin: Springer Verlag.

NOELL, W. K. (1951). The effect of iodoacetate on the vertebrate retina. *J. cell. comp. Physiol.* **37**, 283–307.

NOELL, W. K. (1953). Studies on the electrophysiology and metabolism of the retina. *USAF School of Aviation Medicine, Project no.* 21–1201–004. Randolph Field: Texas.

OGDEN, T. E. & BROWN, K. T. (1962). Intraretinal responses of the monkey retina to electrical stimulation. *Physiologist*, **5**, 190.

POLYAK, S. (1957). *The Vertebrate Visual System*, pp. 250–287. University of Chicago Press.

ROEDER, K. D. (1939). Synchronized activity in the optic and protocerebral ganglion of the grasshopper *Melanoplus femurrubrum*. *J. cell. comp. Physiol.* **14**, 299–307.

SVAETICHIN, G. (1961). Origin of the R-potential in the mammalian retina. In *Neurophysiologie und Psychophysik des visuellen Systems*, ed. JUNG, R. & KORNHUBER, H. pp. 61–64. Berlin: Springer Verlag.

TOMITA, T. & FUNAISHI, A. (1952). Studies on intraretinal action potential with low-resistance microelectrode. *J. Neurophysiol.* **15**, 75–84.

VOLKMER, E. (1956–57). Über Oszillationen im Elektroretinogramm des Frosches. *Z. Biol.* **109**, 203–209.

WALTER, W. G. (1961). In: Computer Techniques and EEG Analysis. Suppl. 20. *Electroenceph. clin. Neurophysiol.* 81–87.

YOUNG, J. Z. (1962). The retina of cephalopods and its degeneration after optic nerve section. *Phil. Trans.* B, **245**, 1–18.