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SYNCHRONIZED REACTIONS IN THE OPTIC GANGLION OF *DYTISCUS*

By E. D. ADRIAN

From the Physiological Laboratory, Cambridge

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THE electrical activity of the optic ganglion of the Water Beetle (*Dytiscus marginalis*) was studied some years ago and the main results were summarized in a note [Adrian, 1932]. One feature of particular interest was the occurrence of rhythmic potential oscillations in the ganglion and of rhythmic bursts of impulses in the nerve, due apparently to synchronous activity in the nerve cells. The tendency to synchronization is now recognized to play a considerable part in the reactions of the cerebral cortex, and it has become important to know more about the conditions which promote it. The optic ganglion of *Dytiscus* is in some ways an ideal preparation for a study of this kind. It has a relatively complex structure—indeed according to Zawarzin [1914] it is equivalent to the retina and optic lobes of a vertebrate—but its electrical reactions are simple and there is the great advantage that its activity can be readily controlled. It has therefore been re-examined in the hope that the results would show what might be expected of nerve cells in general. Although they illustrate a principle which has been invoked before, many of the results can be simply explained and they suggest an explanation for some of the reactions of the cerebral cortex.

STRUCTURE OF THE GANGLION

The ganglion is a small conical mass with its base applied to the compound eye and its apex forming the optic nerve which runs immediately into the supra-œsophageal ganglion on that side (Fig. 1). A section through the median plane of the ganglion shows the nerve cells forming an outer layer surrounding a central spherical mass of dendrites.

A similar mass forms a curved plate near the base of the cone. The optic nerve fibres come from the central mass, and the whole arrangement of cells, dendrites and axons probably resembles that of the optic ganglion of the Dragon fly (*Aeschna*) which has been described by Zawarzin [1914].

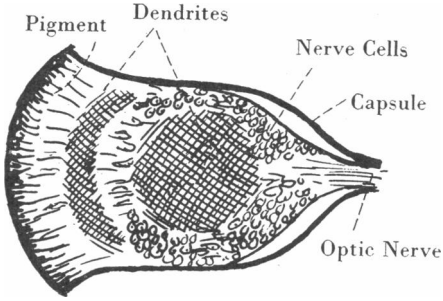

Fig. 1. Diagram to show arrangement of nerve cells and dendrite layers in the optic ganglion of *Dytiscus*.

Method of preparation

The preparation is made by decapitating the beetle, removing the chitin which forms the roof of the head, and clearing away the muscles, tracheal tubes, etc., to expose the paired supra-œsophageal and lateral optic ganglion. The supra-œsophageal ganglion on one side is separated from its fellow and from all other structures but the optic nerve. It is then used as a handle for the optic nerve, and is lifted on to a small ring of silver or platinum wire which forms one of the recording electrodes. The wire is carried in a glass tube fixed in a block of plasticene so as to allow its position to be adjusted. The other electrode is a similar glass handled wire touching the optic ganglion or the tissues near it in the front of the head. Three or four electrodes of the same kind are sometimes used for making simultaneous records from different parts of the ganglion. Non-polarizable electrodes with the metal parts shielded from the light have been used occasionally to make sure that the results have not been due to photoelectric currents from the electrodes. The preparation is set up in a metal box lined with wet cloth, and is irrigated occasionally with Ringer's fluid. As a rule the optic nerve fibres do not survive as well as the ganglion, since they are more liable to damage from drying, but optic nerve impulses can usually be recorded for 3 or 4 hours, and a ganglion has shown typical nervous activity as long as 36 hours after it was set up.

Recording system

The potential changes were amplified with a direct-coupled or condenser-coupled amplifier leading to a Matthews' oscillograph. When the nerve impulses were to be recorded, small coupling condensers were often used to reduce the size of the slower potential changes due to the ganglion, for when the potential rhythms are well developed the ganglion may produce a sinusoidal oscillation of half a millivolt or more. Simultaneous records were sometimes made with two or three oscillographs, used with balanced input amplifiers.

Illumination

Between observations the eye was kept in complete darkness in the preparation box. As the room was darkened for the oscillographic recording the eye could be exposed to dim light by merely raising the velvet curtain covering the front of the box. For very bright light a small 18 watt electric lamp with a metal reflector was set at distances ranging from 24 to 2 in. Intermediate degrees of illumination were given by an opal screen lit by a lamp with a variable resistance to control it, or by altering the general lighting of the room. This was usually so dark that raising the curtain admitted scarcely enough light to make the eye visible to an observer looking into the box, but the illumination as given by a photo-electric cell was generally about $\frac{1}{20}$ m. candle or more. The illumination given by the lamp 2 in. from the eye was about 50,000 times as great. No attempt was made to estimate the exact light intensity or to mark the exact moment of illumination. In the records a hand-operated signal indicates the periods of light and darkness, but it is not intended to give a measure of the latency of the response.

Results

In the fresh uninjured preparation there is no sign of a co-ordinated rhythm except under very bright light. Within 3 or 4 hours a slow potential oscillation appears when the eye is in complete darkness and disappears whenever it is illuminated. The abolition of this "dark rhythm" by light is one of the phenomena with which we shall be concerned, but it must be recognized that the rhythm is to some extent an abnormal event. Its appearance is probably connected with the death of some of the nerve elements from drying, for it can be produced in the fresh preparation by injuring the ganglion. Before the dark rhythm is considered, therefore, the response of the intact ganglion and nerve will be described.

Reactions of the fresh preparation

When the fresh preparation is in complete darkness, the record usually shows occasional spikes indicating an irregular discharge of small impulses at a low frequency in the optic nerve and an irregular oscillation, indicating some electrical activity in the ganglion. The supra-œsophageal ganglion, if attached to the optic nerve, does not contribute to this activity, for it may be crushed or removed without affecting the record appreciably.

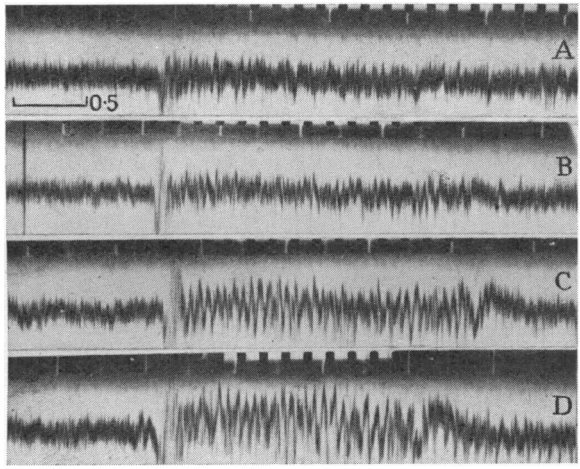

Fig. 2. Discharge of impulses in the optic nerve in response to different illuminations. Fresh preparation. Condenser-coupled amplifier with short time constant. A, very feeble light, raising curtain of box, room dark. B, feeble light, raising curtain, room lighter. C, lamp at 18 in. D, lamp at 3 in. Discharge at 20 per sec. and rhythmic after-discharge at 14 sec. Time: 0·5 sec. marked on record.

On exposure to the feeblest light (by raising the curtain a little way) there is an immediate increase in the impulse discharge. The records in Fig. 2 show the effect of different intensities of light: in all of them the frequency is greatest at the moment of illumination, but there is evidently a progressive increase in the discharge with increasing light. Unfortunately it has not been possible to record from single nerve fibres; with very bright light however (D), there is a regular series at 20 per sec. after the initial peak is over. With moderate illumination (B) the discharge shows no regularity, and is what might be expected from any collection of receptors acting independently of one another. The impulses in each fibre probably form a regular series, though the total response is irregular.

In a few preparations after exposure to medium illumination there has been a slight and momentary increase in the discharge when darkness is restored. More often there is no "off" discharge; the irregular discharge of small impulses continues in the dark but does not vary in frequency.

The first sign of a generalized rhythm appears when the eye is very brightly lit, e.g. by an 18 watt lamp a few inches away. The discharge

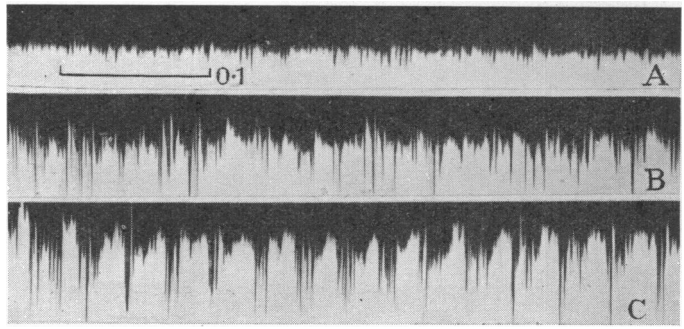

Fig. 3. Grouping of impulses on exposure to very bright light. A, darkness. B, lamp at 18 in., irregular response. C, lamp 2 in. from eye. Initial frequency of groups 36 per sec. Amplifier with short time constant. A downward movement of the base line indicates negativity of the ganglion relative to the nerve. Time: 0·1 sec. marked on record.

Fig. 4. Development of bright rhythm on exposure to very bright light. Waves at 25 per sec. falling to 17 after 1 sec. exposure. A further sudden slowing of the rhythm can be seen when the light is cut off. Time: 0·1 sec. marked on record.

then begins as usual with a crowded irregular succession of impulses, but within a second or less the impulse spikes tend to fall into definite groups recurring with a frequency of between 20 and 40 per sec. as in Figs. 3 and 4—and at the same time a corresponding potential oscillation develops in the ganglion. This effect will be referred to in future as the "bright rhythm". If the lighting is continued the frequency declines slowly, reaching a value of 15–25 per sec. within 5 sec.; if the illumination is reduced the rhythm occasionally continues at a slightly lower frequency, but usually the regular potential waves and impulse groups disappear leaving an irregular discharge.

In the fresh preparation, therefore, a regular response appears only with intense excitation, and ceases when the excitation is diminished. There is no generalized rhythm with illuminations which would be normal for the beetle under water, and none when the eye is in darkness. This frequency of the bright rhythm is not invariable, since it declines with time, but as a rule it cannot be varied by altering the illumination.

Later reactions

If the preparation has been 2–3 hours in the moist chamber the bright rhythm often shows more flexibility. There is still no rhythm in the dark or with moderate light, but the frequency of the rhythm with bright light

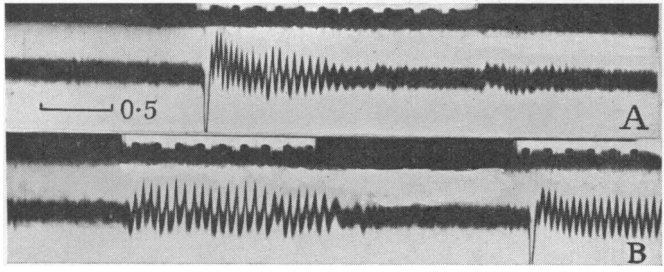

Fig. 5. An example of a bright rhythm showing some variation with the intensity of the light. A, lamp at 2 in. Initial frequency 28 per sec. Rapid fall to 20 followed by disappearance of waves. B, first exposure, to lamp at 18 in. Frequency 20 per sec. Second exposure, to lamp at 6 in. Initial frequency 24 per sec. Time: 0·5 sec. marked on record.

can often be varied over a small range by a change of illumination. Thus, in one experiment when the bright rhythm had fallen to 18 per sec., it could be reduced to 16 by moving the light farther away and restored to 18 by bringing it back. In another, illustrated in Fig. 5, a very bright light gives a rhythm of 28 per sec. with a rapid failure, and a less bright light gave a longer discharge with a rhythm of only 20 per sec. Besides the greater variability in the bright rhythm, there is often a tendency for the waves to persist for a short time after the light is cut off. During these rhythmic after-discharges the frequency falls rapidly, sometimes to values as low as 8–10 per sec.

The dark rhythm

Sooner or later (sooner if the preparation is not kept moist) a rhythmic potential change appears when the eye is in complete darkness, disappearing whenever it is feebly lit. The dark rhythm usually starts as a sequel to the bright rhythm, the frequency falling to 7–9 per sec. when

the light is turned off. In fact in its earlier stages the dark rhythm might be considered an after-discharge which tends to perpetuate itself. To produce it the eye must be adequately stimulated. Thus, in Fig. 6, a brief exposure to bright light gives a short after-discharge at a frequency of about 14 per sec. A longer exposure gives an after-discharge with larger waves and a slower decline, and with a still longer exposure the waves continue at a steady rhythm of 12 per sec. as though the level of excitation could now maintain itself at a fixed value.

In the next stage the slow dark rhythm, once started, will persist, but once abolished will not reappear except as a sequel to the bright rhythm.

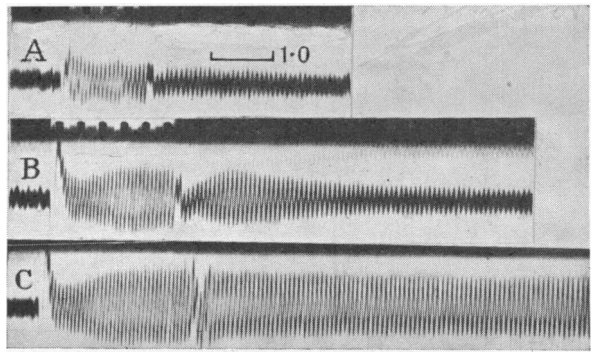

Fig. 6. Development of the dark rhythm as a persistent after-discharge. A, short illumination, short after-discharge of small waves. B, longer illumination (same intensity), after-discharge lasting 5 sec. C, still longer illumination. After-discharge continues indefinitely. The frequency has fallen to 12 per sec. at the end of the record. Time: 1·0 sec. marked on record.

Eventually, however, the waves return whenever the eye is restored to complete darkness, and may persist for several hours with little change in frequency and an amplitude as large as 1 mV. The rhythm sometimes disappears if the eye is kept continuously in the dark for a long period, but brief illumination followed by darkening will restore it, usually to a frequency which is relatively high and declines slowly to the steady value. Thus occasional periods of illumination seem to be necessary both to start the rhythm and to maintain it, as though without them the activity tends to fall below the level required for a regular discharge.

Although the preparation may remain active for many hours after the dark rhythm has appeared, there can be little doubt that it is a sign of some pathological change. If there has been any damage in the dissection the rhythm may appear at once, and it can be produced at any time by

cutting into the optic ganglion at the point where the nerve leaves it. Cutting the nerve itself seems to have less effect than damage to the ganglion, though the damage need not be extensive and may be confined to the apical part. In some preparations puncturing the capsule has been enough to start the dark rhythm. That injury should favour synchronization is not surprising, for it is known to do so in peripheral nerves. In this case, however, it is difficult to say how it operates, as we do not know what particular structures have to be damaged.

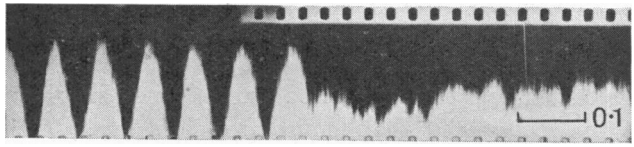

Fig. 7. The dark rhythm. Potential waves at 12·5 per sec. abolished by exposure to faint light. In this record the amplitude of the waves is about 80μV.: they are often as large as 500 μV. Time: 0·1 sec. marked on record.

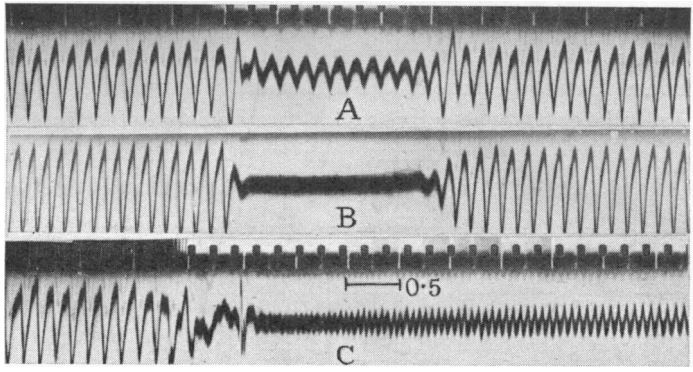

Fig. 8. The dark rhythm is reduced or abolished by light. If the light is intense the bright rhythm appears. A, dark rhythm, 7 per sec. Waves diminished by very faint light (raising curtain slightly). B, waves abolished by faint light (raising curtain fully, room dark). C, appearance of bright rhythm. Lamp at 4 in. abolishes the dark rhythm and produces a bright rhythm of initial frequency 20–18 per sec., falling to 14 per sec. Time: 0·5 sec. marked on record.

Absence of rhythm in dim light

When the dark rhythm has appeared there is a regular potential oscillation at 7–10 per sec. in complete darkness as well as that at 20–30 per sec. in bright light. But exposure to dim or medium light always abolishes the large potential waves of the dark rhythm and restores a steady base line. Figs. 7 and 8 show the partial and complete abolition

of the dark rhythm by dim light, as well as the development of the bright rhythm by intense light. There is sometimes a slight fall in the frequency of the waves when they are reduced, but the main effect is the change in size (Fig. 16). The amount of light needed is very small—raising the curtain of the preparation box is enough—and, except with the very feeblest light, there is no return of the dark rhythm until the curtain is lowered again.

Thus the ganglion will now give the two rhythms, in complete darkness and in very bright light; but in dim or medium light there is no regular response. Both in the bright and the dark rhythm the frequency may show a progressive decline from its initial value, but the rhythms are fixed in the sense that they cannot be altered (except over a small range for the bright rhythm) by altering the illumination of the eye.

Analysis of results

The nature of the two response rhythms

It is evident that the rhythmic potential changes must be due to synchronized action in a large number of units. It does not follow that all the neurones tend to become active and inactive simultaneously; in fact it will appear later that the active region may be conducted from one part of the ganglion to another during each wave. None the less, for such regular oscillations there must be a very close co-ordination between the different neurones. Thus a failure of the waves might result from a failure of co-ordination as well as from a failure of activity. The question which then arises is why the ganglion tends to give only the two fixed rhythms which have been labelled "bright" and "dark". Why does feeble illumination suppress the waves of the dark rhythm and give none of its own in return? The problem is important, because in other groups of neurones we find the same tendency to give definite potential rhythms which appear and disappear but do not change appreciably in frequency. Berger's α rhythm in the human cortex is a good example; indeed the abolition of the α waves in man has already been compared with that of the dark rhythm in the water beetle [Adrian & Matthews, 1934 *b*]. An analysis of the optic ganglion response may help to decide whether these fixed rhythms are merely those at which synchronization can occur, or whether they are due to certain neurones which can only respond at a fixed rate. The latter possibility is unlikely, for in sensory endings and motor neurones the response frequency can vary over a wide range, but there are some kinds of active cell in which it varies very little.

The fixed rhythms in the optic ganglion can be simply explained on the first supposition, but not on the second. It is reasonable to assume that the neurones will be unable to develop a synchronous beat unless they are all excited to much the same degree, and so are all tending to beat at much the same frequency. With a very bright light they would be excited to their maximum rate, and it will need very little adjustment to make the periods of discharge coincide in all of them. Thus the bright rhythm can be obtained even in the fresh preparation. If it can be assumed that both the bright and the dark rhythm are due to the same neurones the latter might occur because in complete darkness there will be no external stimulation to determine the frequency in each neurone: some will give a spontaneous "resting" discharge at a low rate, probably more will do so if the preparation is injured and they will be more able to influence their neighbours. Thus in the dark there will be nothing to prevent the neurones beating together at a slow rate corresponding to the resting discharge. In feeble or moderate light, however, the neurones will be excited to very different degrees, since the thresholds for the different receptors in the eye will vary and so will the amount of light falling on them. Thus the frequencies will differ too widely to allow synchronization.

This effect of feeble illumination is illustrated in Fig. 9 which gives three imaginary intensity-frequency curves of the usual exponential form. The curves are all alike, but the first is supposed to apply to a receptor which needs one light unit for threshold excitation, the second needs two units and the third three. It is assumed that a tenfold increase of light intensity covers the whole frequency range, whereas in reality we are dealing with intensities varying from 1 to 10,000 or more, but the form of the curve is all that matters and this agrees with Hartline & Graham's [1932] measurements on the eye of *Limulus* and with the curves from other receptors. To show the effect of dim and of bright light two vertical lines have been drawn corresponding to illumination of 4 and 16 units. With the dim light the frequencies of response in the three receptors are 10, 14 and 24 per sec. With the bright light they are 26·5, 29 and 30. Thus with the dim light the frequency of two of the receptors would have to be changed by about 40 p.c. to produce a synchronous beat. With the bright light a change of less than 10 p.c. would bring all three to the same value.

Evidently with very bright light a synchronous beat could develop even though the neighbouring units could only exert a slight effect on one another. A much greater degree of interaction would be needed to synchronize the responses to dim or medium light; and if it existed the

eye would be valueless for pattern vision since it would no longer be possible for differences in intensity to be signalled by different frequencies of discharge in the various nerve fibres. In darkness the different units, if isolated from one another, might give resting discharges at widely different rates, but a slight degree of interaction could bring them into line, since there is no external stimulus to set the pace.

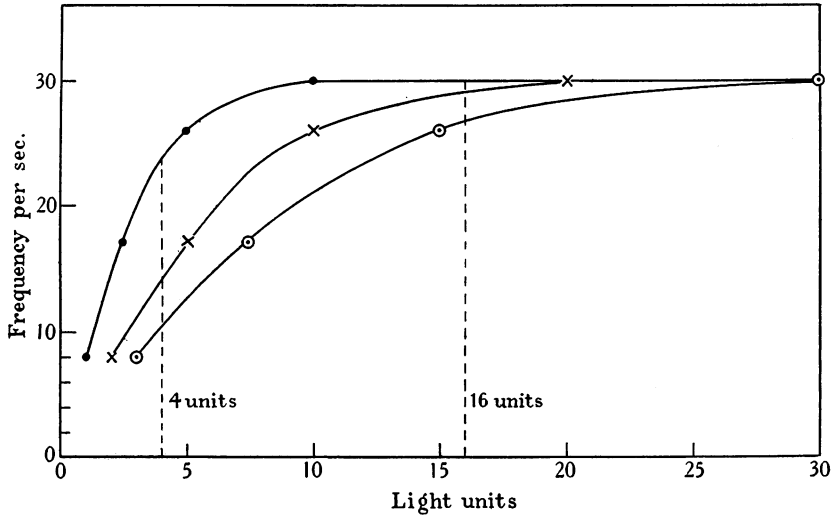

Fig. 9. Imaginary curves relating stimulus (light units) and frequency of discharge for three receptors of different threshold. In faint light (4 units) the frequencies differ much more than in bright light (16 units).

If this explanation of the two rhythms is to be accepted, we must be satisfied that the same neurones produce both slow and rapid rhythms and can respond at intermediate frequencies as well. This would at once rule out the alternative hypothesis of neurones with a fixed rate of response. It will be shown that intermediate frequencies can certainly occur in conditions which would favour synchronization. It is possible that the persistent dark rhythm has a different explanation, but this does not affect the general argument.

Intermediate frequencies

It has already been stated that if bright light is followed by complete darkness the bright rhythm may slow down gradually until the frequency is that of the dark rhythm (cf. Fig. 10). A gradual slowing does not happen in every preparation but it has happened often enough to make it improbable that the two rhythms are due to two completely independent

sets of neurones. Moreover, when there is an abrupt fall from the high rate to the low there is no definite interruption in the sequence of waves. This is illustrated in Fig. 4.

With a preparation which gives the gradual slowing when the light is turned off, an interesting variation is obtained when bright light is

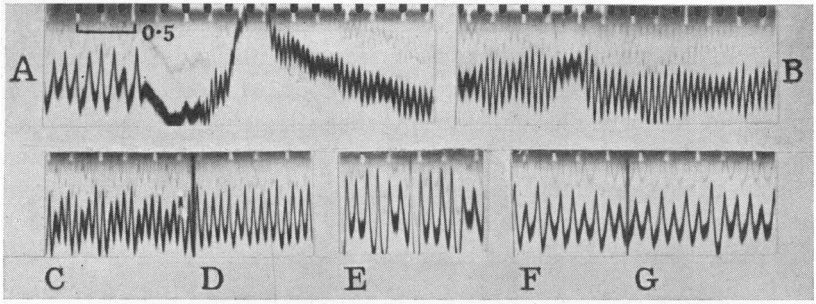

Fig. 10. Gradual slowing of the rhythm when bright light is followed by darkness. A, dark rhythm, 10 per sec. Light on. Bright rhythm, 24 per sec. B, light on 5 sec. Bright rhythm, 18 per sec. Light off. Complete darkness. C, 7 sec. after B. Rhythm 16 per sec. D, 12 sec. after. 14 per sec. E, 19 sec. after. 11 per sec. F, 26 sec. after. 10 per sec. G, 35 sec. after. 9 per sec. Time: 0·5 sec. marked on record.

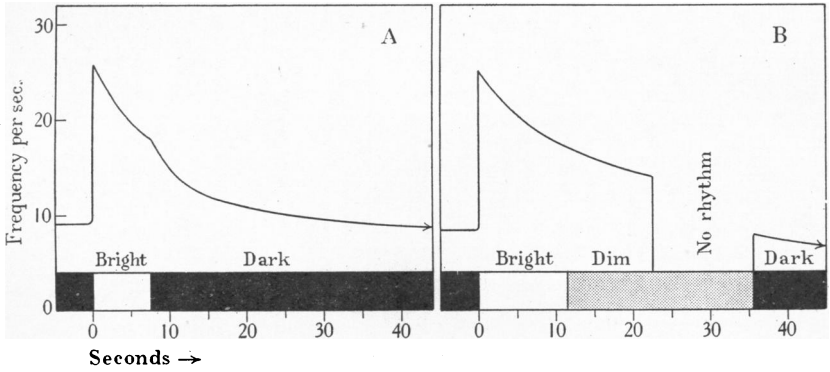

Fig. 11. Abolition of the middle frequencies of an after-discharge by dim light. A, frequency of potential waves when bright light is followed by complete darkness. B, frequency when bright light is followed by dim light and then by complete darkness.

followed by dim light. The rhythm declines until it reaches a value well above that of the dark rhythm, and the waves then cease abruptly. The dimmer the light the lower is the frequency to which the rhythm will fall before the waves disappear. Complete darkness restores them, usually at a lower frequency, and the decline then continues until the normal dark value is reached. The result is shown in the curves in Fig. 11.

On the hypothesis outlined above it can be explained without difficulty as follows. In the preparations in which there is a gradually declining after-discharge, the change from bright light to complete darkness evidently produces a slow instead of an abrupt fall in the level of excitation. The synchronous beat can continue as the excitation declines because, in the darkness, there is no external stimulation to impose different rates on the different neurones. But if bright light is followed by dim light, the waves can only persist as long as the uniform excitation left by the bright light exceeds the non-uniform excitation produced by the dim light. As soon as the frequency has fallen below the average value imposed by the dim light, the inequalities of excitation will assert themselves and will abolish the rhythm; and if the light is moderately bright the rhythm will not decline much from its maximum value before it is broken up.

Thus if there is no chance of uneven excitation, we can often obtain synchronized rhythms over the whole frequency range from 6 to 40 per sec. But with dim or medium light the excitation is bound to be uneven: consequently, the middle range of frequencies can only be obtained as an after-effect of the bright rhythm. It is clear, however, that the neurones can respond over a wide range and are not restricted to one or two fixed rhythms.

The dark rhythm and its abolition by light

In the hypothesis put forward above, it was assumed that the neurones which give rise to the potential waves are all of the same kind, and that the dark rhythm represents their spontaneous discharge when they are not stimulated. The abolition of the dark rhythm by faint light would then be due to their increased but non-uniform activity, for this would make synchronization impossible. If this view is correct, we ought to find that the abolition of the waves by light, however feeble, is always associated with an increased impulse discharge in the optic nerve. This has been found in every preparation in which there were surviving nerve fibres. The records in Figs. 12 and 13 illustrate the increased discharge associated with the failure of the rhythm—they were made with small coupling condensers to show the impulses more clearly. It can be seen also that when either the dark or the bright rhythm is present the impulses are discharged in groups more or less in phase with the potential waves, whereas in dim light the grouping is lost and the waves abolished.

There is, however, another possibility which cannot be completely ruled out, namely, that the persistent dark rhythm is due to a distinct group of neurones which are only active when the eye is in darkness. Hartline [1937] has found that the eye of *Pecten* contains receptors

which are stimulated by the change from light to darkness, and may continue in action for long periods in the dark. The vertebrate eye also shows an increased discharge in the optic nerve when the light is cut off [Adrian & R. Matthews, 1928], and Hartline finds that the nerve fibres in which the "off" discharge takes place are not the same as those which are active when the eye is illuminated [Hartline, 1935]. In some *Dytiscus* preparations a small "off" discharge has appeared when the

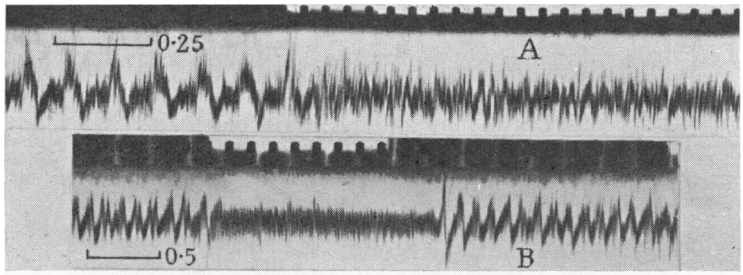

Fig. 12. Increased discharge of impulses when the dark rhythm is abolished by dim light. Amplifier with short time constant to reduce the size of the slow waves. A, dark rhythm at 9 per sec. Faint light (raising curtain) gives an irregular discharge of impulses. B, another preparation. Dark rhythm at 10 per sec. with impulse groups at the same frequency. Faint light gives a rapid discharge. Time: 0·25 and 0·5 sec. marked on records.

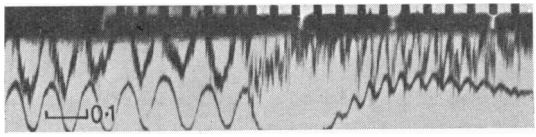

Fig. 13. Exposure to bright light abolishes the dark rhythm, giving an impulse discharge which becomes grouped as the bright rhythm developes (at 24 per sec.). Upper tracing made with amplifier with short time constant to show impulses, lower with amplifier with long time constant to show waves. Time: 0·1 sec. marked on record.

previous exposure to light was carefully adjusted: in most there is no increase in activity when the light is cut off, but there is always a persistent irregular discharge of small impulses whilst the eye is in darkness (cf. Fig. 3). Presumably the dark rhythm is obtained when the neurones which give these small impulses become synchronized; and as the impulses produced by illumination are on the whole larger, it is evident that there are some neurones which contribute to the bright rhythm but not to the dark. But the continuous decline in frequency which may occur on darkening shows that the neurones responsible for the

bright and dark rhythms cannot be completely independent. In records of the impulses accompanying a rhythmic after-discharge it is found that the groups which appear with each wave are made up of spikes of all sizes: as the frequency of the waves declines the number of large spikes in each group becomes less, and as the final value of the dark rhythm is approached the large spikes are found only occasionally. It is possible that the large spikes are due, not to larger nerve fibres, but to several acting in unison; but in any case it is only at the lowest frequencies that they are completely absent. The arguments as to synchronization can therefore be applied to all but the lowest rhythms, and may be applicable to these also. If the persistent dark rhythm is due to special receptors and needs stimulation by darkness to maintain it, the abolition

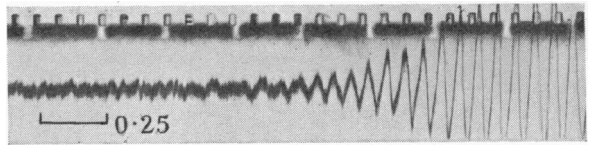

Fig. 14. Sudden development of a bright rhythm at 16 per sec., 4 sec. after the beginning of illumination. Time: 0·25 sec. marked on record.

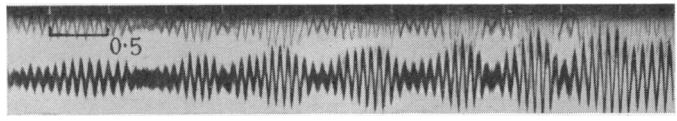

Fig. 15. Progressive return of the dark rhythm (12 per sec.) after its abolition by light. Time: 0·5 sec. marked on record.

of the waves by light must be attributed merely to the withdrawal of the stimulus. But where a rapid rhythm has been present during illumination, has declined gradually when the light is cut off and is then abolished by light of medium intensity, it is more likely that the waves fail owing to desynchronization than owing to the absence of a dark stimulus.

There are other lines of evidence to support the view that the conditions in which the rhythms appear are merely those in which synchronization is possible. One point is that the waves often appear and increase rapidly in size some time after the appropriate conditions have been established. In Fig. 14, for instance, the waves of the bright rhythm develop in this way without any change in frequency. Fig. 15 shows the progressive return of a dark rhythm after its abolition by light, and both figures recall the rapid, progressive development of large potential waves

in injured nerve trunks and other collections of excitable cells which can respond synchronously [cf. Adrian, 1930; Adrian & Gelfan, 1933; Hoagland, 1933].

During the abolition of the dark rhythm by dim light there is sometimes a slight but distinct fall in frequency (Fig. 16). This certainly

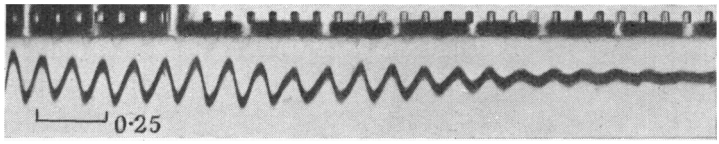

Fig. 16. Dark rhythm reduced in amplitude by gradually increasing light. The frequency falls from 10 per sec. to 9. Time: 0·25 sec. marked on record.

favours the view that the rhythm is abolished because the dark stimulus has failed; on the other hand it might result from the elimination of the more excitable neurones which would set the pace of the resting discharge, but would cease to control it if their frequency was increased by the light.

As we might expect, when the waves of the dark rhythm are reduced in size by light, although there may be little change in frequency there is

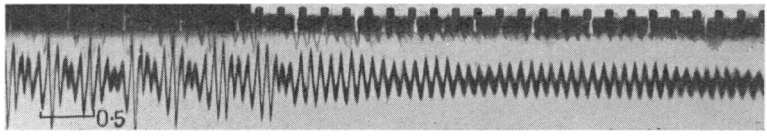

Fig. 17. Complex dark rhythm. Exposure to faint light eliminates one component. Time: 0·5 sec. marked on record.

evidently a reduction in the number of neurones which contribute to the synchronized waves. This can often be seen when the waves are complex, for a very feeble light may then simplify them by eliminating one of the components (Fig. 17).

Summation of potentials in the ganglion

The increased discharge of impulses in the nerve shows that on the whole the activity of the ganglion is increased by feeble light, although the large waves are abolished. Further evidence of the increased activity comes from records of the steady potentials which are developed on exposure to light. When these are made (with a direct-coupled instead of a condenser-coupled amplifier) they show a sustained negativity of the ganglion,

increasing with the light; there is no sign of any reversal of the effect associated with the failure of the dark rhythm.

A typical record is given in Fig. 18 A. The dark rhythm is present at the beginning. When the eye is brightly illuminated the ganglion develops a negative potential with respect to the nerve, and it is on this new base line that the waves of the bright rhythm appear a third of a second later. With continued illumination the potential difference declines gradually, and so does the rate of oscillation. When the light is turned off the base line returns to its original position, the dark rhythm appearing later (not

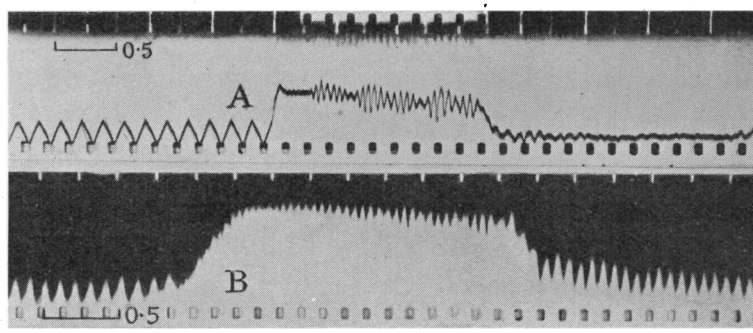

Fig. 18. Records with battery-coupled amplifier showing the persistent negativity of the ganglion on exposure to light. With this amplifier the base line moves upwards when the ganglion becomes negative to the nerve. A, dark rhythm, 6 per sec. Exposure to lamp at 6 in. makes the ganglion 0·7 mV. negative to the nerve and induces a bright rhythm at 22 per sec. B, another preparation. Dark rhythm, 12 per sec. Exposure to bright light makes the ganglion 0·8 mV. negative and induces a bright rhythm at 22 per sec. After-discharge at 14 per sec. with the ganglion remaining 0·15 mV. negative. Time: 0·5 sec. marked on record.

shown in the figure), but in Fig. 18 B there is both a remainder of the potential shift and a higher frequency than at the beginning. These figures bear a close resemblance to Fröhlich's string-galvanometer records from the cephalopod eye, published 25 years ago [Fröhlich, 1913].

The sustained negativity of the ganglion is found in every preparation. Its magnitude seems to depend on the degree of excitation, for it varies with the nerve discharge when different illuminations are used, tending to a maximum with illuminations strong enough to give the bright rhythm. Also it outlasts the light when there is an after-discharge, but declines abruptly when there is none. That there is a close relation between the potential level and the frequency of the waves can be seen in

Fig. 18, and more clearly in Fig. 19. This was made by moving an electric torch up to and away from the eye. As it is moved nearer the negative potential of the ganglion increases: when the excitation reaches its maximum the neurones begin to work in unison and the waves of the bright rhythm appear, and as the light is moved away the potential declines and the frequency falls from 22 to 15 per sec. before the waves are broken up.

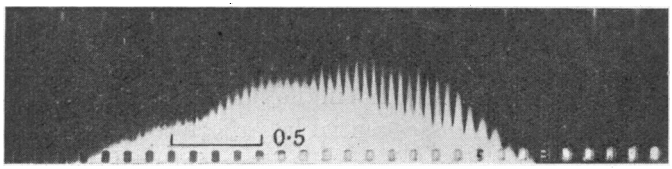

Fig. 19. Battery-coupled amplifier. A bright light is moved towards the eye and then away from it. The ganglion becomes increasingly negative and the bright rhythm appears at 22 per sec. As the light is moved away the potential declines and the rhythm falls to 14 per sec. before the waves fail. Time: 0·5 sec. marked on record.

The close relation between the waves and the steady potential suggests that both may arise in the same neurones. As far as can be discovered by varying the position of the electrodes, they certainly come from the same part of the ganglion. Both the waves and the steady deflexion are greatest when the middle and apical regions are included between the leads, and no arrangement has been found to give greater prominence to one effect rather than the other. But the ganglion is so small that a difference in focus might well have been overlooked.

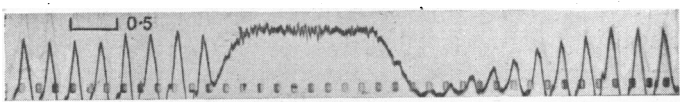

Fig. 20. Battery-coupled amplifier. Dark rhythm (4 per sec.) abolished by faint light. A small negative potential (0·08 mV.) develops in the ganglion. Time: 0·5 sec. marked on record.

Fig. 20 shows that a dim light gives a potential difference in the same sense as a bright light. The illumination may be only just enough to stop the dark rhythm, but invariably the ganglion becomes negative to the distal part of the nerve. The waves in this case were small (less than $\frac{1}{10}$ mV.) for the dark rhythm had only just appeared: later they increased so much that, in comparison with them, the shift in the base line produced by dim light would have been inappreciable, for the potential shift for a given light remains fairly constant whether the dark rhythm has appeared or

not and whatever the size of its waves. The constancy of the steady potential and the variation in the waves fits in very well with the hypotheses that the former is a measure of the total activity and the latter of the degree of synchronization.

Nature of ganglion potentials

As regards the nature of these potential changes, the present observations do not add much to the account given some years ago [Adrian, 1931]. This dealt with the spontaneous activity of the ventral ganglia of *Dytiscus*, where a discharge of impulses along the motor nerve is always associated with a negative potential in the ganglion (relative to the nerve). It was found that the fall of potential began before the nerve discharge and persisted until it was over: from this it was inferred that the activity of the cells and dendrites must involve a relatively slow depolarization, as a result of which one or more impulses are discharged along each axon. The same explanation was suggested for the potential waves in the optic ganglion [Adrian, 1932].

Later work on the potentials in the cerebral cortex [Adrian & Matthews, 1934 *a*] threw some doubt on the possibility of a sustained negative potential in the individual cells, and emphasized the composite nature of many of the slower changes. In the present experiments we are certainly dealing with composite effects: for instance, the persistent negativity when the eye is brightly lit must be due in part to a summation of potential waves in neurones beating out of phase—for the waves are obvious when the neurones come into phase. But however large and regular the waves of the bright rhythm may be, there is always a persistent negativity of the ganglion in addition, and this suggests some persistent depolarization in the individual neurones. Such an analysis would agree with (and was suggested by) Barron & Matthews's [1936] recent findings in connexion with spinal cord potentials: their results are so much more definite, however, that a discussion of probabilities from the *Dytiscus* records would be a waste of time.

Whatever the steady potential may imply, it is clear that the waves are an index of the rhythmic activity of the nerve cells and dendrites. The records in Fig. 21 show the relation between waves and nerve impulses in two records made at a higher speed than usual. The eyes are brightly illuminated, but the bright rhythm has developed to different degrees of regularity. When it is poor (Fig. 21 A) the groups of impulses, though occurring with nearly the same frequency as the waves, are sometimes on the crest and sometimes in the trough, as though they came

from a group of neurones slightly out of phase with the majority. But when the waves are quite regular (Fig. 21 B) the impulses always appear in a fixed relation to them, when the negativity of the ganglion is nearing its maximum.

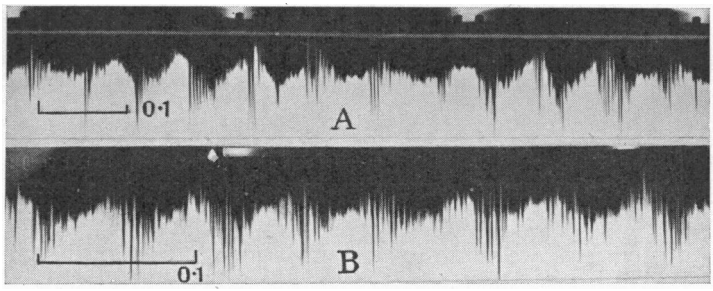

Fig. 21. Relation between waves and nerve impulses during the bright rhythm. Condenser-coupled amplifier with short time constant. The base line moves down when the ganglion becomes negative to the nerve. A, the waves vary in size and the impulses have no constant relation to them. B, another preparation. Regular waves with impulse groups always appearing as the ganglion becomes negative.

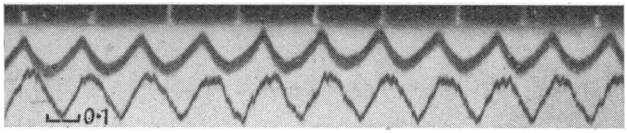

Fig. 22. Simultaneous records of potential change at two pairs of electrodes. Upper tracing, electrodes on nerve and near apex of ganglion. Lower tracing, electrodes near apex and near base. Dark rhythm, 5 per sec. Phase difference between waves in upper and lower tracings, showing movement of active region during each wave. Time: 0·1 sec. marked on record.

The form of the potential waves varies with their frequency, and there is often a near approach to a sinusoidal oscillation both with the dark and the bright rhythm. But the form is governed to some extent by movements of the active region in the course of each wave. The existence of such movements can be detected by recording simultaneously from two pairs of electrodes, one on the base and middle of the ganglion and the other on the middle and apex. The maximum potential change seems to arise from the middle and upper third, but there is often a phase difference between the waves in the two records (Fig. 22), indicating a shifting region of activity. Owing to the small size of the ganglion, no attempt has been made to work out an accurate map of the potential distribution from moment to moment; it can only be said that the apparent wave

form does not necessarily show the rate of rise and fall of activity at a given point any more than the electro-cardiogram shows what is happening at a given point in the heart.

Discussion

This account has been mainly concerned with two abnormal phenomena, the potential oscillations produced by a very bright light and those occurring in the dark when the ganglion has suffered injury. In these conditions the neurones of the optic ganglion react as a mass giving a single rhythm: in conditions nearer the normal for the living animal they react independently, giving a total response in which no rhythm can be seen. Since the eye must be useless for pattern vision when the neurones are working as a single unit, it is scarcely surprising to find that they are only synchronized at the extreme ends of the visual scale—in complete darkness and intense light. But can such abnormal effects tell us anything about the general reactions of nerve cells?

In one respect they confirm what was already fairly clear, namely, that neurones cannot be expected to work synchronously unless the degree of excitation is the same for all of them, so that all tend to beat with the same frequency. This has been pointed out in explanation of the definite 50–80 per sec. rhythm in an intense motor discharge, for the motor neurones are then excited to the maximal degree [Adrian & Bronk, 1929]. It has also been used to explain the 10 per sec. potential waves in the cerebral cortex (Berger's α rhythm), the assumption being that the neurones are unstimulated and are beating at the minimal rate [Adrian & Matthews, 1934*b*]. In the *Dytiscus* ganglion, however, we have both a rapid and a slow rhythm and the results show that these are quite compatible with a response over the entire frequency range in the individual units. This is the main outcome, for in face of it there is no need to suppose that the fixed potential rhythms of the cerebral cortex must imply any inflexible response in the cortical neurones.

When we come to the factors which prohibit intermediate rhythms, it may be more risky to generalize from the *Dytiscus* results. In a discussion of the rhythmic potentials from the vertebrate retina [Adrian & R. Matthews, 1928], a uniform illumination of the visual field was considered necessary for synchronization: the present results emphasize the fact that what is essential is uniform excitation of the neurones rather than a uniform stimulus. Uniform excitation is not likely to be achieved by anything but a very bright light or no light at all, for it is unlikely that all the receptors will have the same threshold and so will give the same

frequency when they are equally lit. The light used by Adrian & R. Matthews to produce a rhythm in the vertebrate retina would certainly rank as bright, and in view of the present results it is probable that a uniform field, if dimly lit, would not give a synchronized response. The distinction between uniform stimulation and uniform excitation is worth drawing, for it may apply to other structures than sense organs. For instance, uniform stimulation of a cortical area might break up a threshold rhythm by exciting the neurones to different levels.

The dark rhythm in the *Dytiscus* ganglion needs abnormal conditions to develop at all and breaks down as soon as any light falls on the eye. If the failure is due to increased but asynchronous activity, it implies a very loose linkage between the different neurones, one which allows them to act quite independently when they are stimulated. This kind of association would be secured if the active period in one neurone were able to exert a slight stimulating effect on its neighbours, for this would ultimately bring all the neurones into line if there were nothing else to affect their responses. It is interesting to find that the association is favoured by injury, for there are several examples of synchronized discharges in nerve trunks which have been injured (e.g. the phrenic [Adrian, 1930]; the lateral line nerve [Hoagland, 1933]). In these there is some reason to believe that the interaction is due to electrical stimulation of one fibre by another, the injury having caused a breakdown of the normal insulation. There are many parts of the vertebrate central nervous system where injury favours interaction as it does in the *Dytiscus* ganglion, but to account for the synchronization at high frequencies we must suppose that in both some degree of interaction must be perfectly normal. In abnormal conditions (e.g. convulsant drugs, asphyxia, repeated stimulation) the degree of interaction in the vertebrate central nervous system may become so much exaggerated that the neurones can no longer work independently. In complex systems, however, the factors which promote a synchronized response may differ to some extent from those considered here; for instance, a single pace-maker in some part of the brain stem might impose its rhythm on a distant group of neurones, which would then react in unison as they would in response to rhythmic stimulation. Where this occurs there should be no restriction to particular frequencies: indeed we may conclude that the restriction of potential waves to a fixed frequency is evidence that the neurones are not very closely linked and are capable of acting independently at different frequencies.

Conclusions

The electric responses of the optic ganglion and nerve of the Water Beetle (*Dytiscus marginalis*) show that the neurones are often working synchronously, giving rhythmic potential waves and a grouped impulse discharge. The factors which promote or hinder the synchronous response have been investigated in the hope that the results might apply to other collections of nerve cells. The results are as follows:

1. In a fresh preparation a generalized rhythm is only obtained when the eye is exposed to very bright light. The bright rhythm lies between 20 and 40 per sec., declining in frequency with time. If the light is reduced the potential waves disappear.

2. Some hours after the preparation has been made, a potential rhythm at 7–10 per sec. appears when the eye is in complete darkness. The dark rhythm is an abnormal reaction, and can be brought on by injuring the ganglion. If the eye is exposed to the faintest light the waves disappear.

3. When the dark rhythm has developed, the ganglion shows the two fixed potential rhythms corresponding to bright light and no light. With dim or medium light there is no generalized rhythm, but there is the usual irregular discharge of impulses in the optic nerve.

4. Since bright light may cause a rhythmic after-discharge of declining frequency, it is clear that the two rhythms are not due to two completely independent groups of neurones with fixed rates of response. The neurones can respond over a wide frequency range and the fixed potential rhythms are those at which synchronization can occur.

5. It is shown that in a collection of receptors of differing threshold the frequencies of response will tend to differ widely when the stimulus is of small or medium intensity. Synchronization will then be impossible. When the stimulus is strong the frequency will be maximal in all and synchronization can occur. When there is no stimulus synchronization might occur at a low rate corresponding to the "resting" discharge.

6. The persistent dark rhythm may possibly depend on the stimulation of certain neurones by darkness: its abolition by light would then be due merely to the withdrawal of the stimulus. It is more likely that the rhythm represents a spontaneous discharge in neurones which respond at a higher rate on illumination. The failure of the waves on illumination would then be due to the neurones being stimulated to respond at different frequencies. In darkness a few neurones might give rise to the rhythm, for there would be nothing to prevent the others coming into line with them provided that some interaction can occur.

7. The potential changes in the ganglion resemble those in other groups of nerve cells. The waves coincide with the discharge of impulses in the nerve.

8. From the reactions of the *Dytiscus* ganglion it is clear that fixed potential rhythms (e.g. in the cerebral cortex) need not imply a fixed frequency of response in the neurones which contribute to the waves. Synchronized waves will be most likely to occur when the frequencies are near the maximal or the minimal values for these neurones.

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