J. Physiol. (1960), **154**, 514–546
With 10 *text-figures*
Printed in Great Britain

SYNAPTIC POTENTIALS, AFTER-POTENTIALS AND SLOW RHYTHMS OF LATERAL GENICULATE NEURONES

By P. O. BISHOP and R. DAVIS
From The Brain Research Unit, Department of Physiology, University of Sydney, Australia

(*Received* 6 *July* 1960)

A detailed study has recently been made (Bishop & Evans, 1956; Bishop & Davis, 1960) of the recovery cycle of the sensory synapses in the lateral geniculate nucleus extending from absolute refractoriness till 5 sec after the discharge of the geniculate neurones. Following their discharge by an orthodromic volley, these neurones respond normally as soon as conduction becomes possible in the presynaptic fibres and, for a brief period while the presynaptic fibres are still recovering from refractoriness, they pass through a phase of relatively supernormal responsiveness. At shock intervals between about 3 and 6·7 msec the geniculate neurones pass through a phase of true supernormality, to be followed in turn by a deep and prolonged subnormality from which recovery is not complete until 2 sec have elapsed. In view of the well-known relationship which exists between the recovery cycle of peripheral axons and their after-potentials (Erlanger & Gasser, 1937) it was natural that we should attempt to record the after-potentials of the geniculate neurones and to examine the extent to which the recovery cycle of the geniculate synapses can be correlated with them. The morphological arrangement of the dendrites, cell bodies and axons of the principal cells in the lateral geniculate nucleus provides a favourable situation both for recording these after-potentials and for assigning electrical activity to histological structure. Vastola's earlier investigations (1957, 1959) of the after-potentials of geniculate neurones were largely concerned with antidromic activation. The present report is principally concerned with the after-potentials generated by orthodromic volleys, although antidromic activation has also been employed.

The ability to block transmission at junctional regions has for long been a powerful technique for studying synaptic mechanisms. In relation to the synapses in the lateral geniculate nucleus two new blocking techniques have recently become available, namely the use of the drug D-lysergic acid diethylamide (LSD) (Evarts, Landau, Freygang & Marshall, 1955; Bishop, Field, Hennessy & Smith, 1958; Bishop, Burke & Hayhow, 1959*b*)

and the phenomenon of post-tetanic delayed depression (Hughes, Evarts & Marshall, 1956; Evarts & Hughes, 1957*a*, *b*; Bishop, Burke & Hayhow, 1959*a*). When the synapses are blocked by these means the post-synaptic potentials produced by orthodromic volleys are strikingly similar to the after-potentials generated by the same neurones when normal synaptic transmission occurs and post-synaptic spikes are discharged. A detailed study was therefore made of the geniculate synaptic potentials produced under conditions of synaptic block, and this is now reported. The relatively small changes that take place in the synaptic potentials when normal synaptic transmission again becomes possible leads to the idea that the after-potentials as recorded have in them a very important component which is derived from the continued presence of the synaptic depolarization on parts of the neurone (i.e. dendrites) either subjected to prolonged action by the transmitter substance or possibly not normally invaded by the spike process. The available data relating to the synaptic potentials and after-potentials of spinal motoneurones will be reviewed from this point of view.

In this paper we also examined the relationship between the synaptic potentials and after-potentials of geniculate neurones and the spontaneous rhythmic potentials that may be recorded in the nucleus. Our results are interpreted as indicating that the spontaneous waves represent rhythmic fluctuations or the membrane potential of the dendrites and possibly the cell bodies of geniculate neurones. These apparently arise as a result of mechanisms intrinsic to the dendrites themselves although they can also be triggered off or modified both by orthodromic and antidromic volleys.

METHODS

Adult cats were anaesthetized with intraperitoneal allobarbitone solution (100 mg/ml., 0·5 ml./kg; Dial, Ciba) supplemented if necessary (usually at the onset) by the intravenous administration of small amounts (to a total of 0·3 ml., unless otherwise stated) of sodium pentobarbitone solution (64 mg/ml.; Sagatal, May and Baker). In most of the experiments the animals were also paralysed with gallamine triethiodide (Flaxedil, May and Baker, 8 mg intravenously at half-hourly intervals), in order to eliminate potentials of muscular origin, which can seriously distort the slower geniculate wave forms. Respiration was maintained by means of a pump. The body temperature was controlled with the aid of an electric heating blanket. The general methods used were the same as those described in previous papers from this laboratory (cf. particularly Bishop, Jeremy & Lance, 1953). After resection of the eyeball the optic nerve was suspended clear of orbital tissue. The optic nerve was stimulated electrically and the responses in and about the opposite lateral geniculate body were recorded by means of a stereotaxically directed steel micro-electrode introduced down through the intact cerebral cortex. All the potentials were recorded extracellularly between the micro-electrode and an 'indifferent' electrode inserted into the occipital muscles.

Steel micro-electrodes are subject to slow drifts in potential due to polarization effects, though these are minimized by the high-impedance input stage of our amplifiers. A control experiment, however, with the glass micro-pipette type of electrode, showed that the form

and time course of the slow geniculate wave forms were the same with either method. In the earlier experiments the uninsulated tips of the steel micro-electrodes were coated with platinum black, but this was discontinued when it was shown that the wave forms were not changed by its use. The deposition of platinum black is, however, a useful method for determining the extent of the uninsulated tip of the micro-electrode. The steel micro-electrodes were preferred because they are more convenient for stereotaxic use, they have a low resistance and the recording site can be easily labelled for subsequent histological identification by the electrolytic deposition of iron. Glass micropipette electrodes offered no advantage, because the geniculate responses were always recorded against a background of biologically generated 'spontaneous' slow waves of potential, which completely mask any polarization effects. These base-line fluctuations were offset by recording and measuring large numbers of traces. One stage of condenser-coupling was used in the amplifiers, but the time constant chosen was always shown to be adequate by recording the responses with a still longer time constant.

The geniculate neurones were activated antidromically by embedding a bank of 12 steel micro-electrodes in the gyrus lateralis of the cerebral cortex. These stimulating electrodes, similar to those used for recording, were arranged in two parallel rows of six each, with the rows 2 mm apart and the individual electrodes in a row also 2 mm apart. The rows were positioned along the gyrus with the centre of the bank at approximate Horsley–Clarke co-ordinates of 3·5 mm lateral and 7·5 mm anterior and the tips of the electrodes 3–4 mm below the surface of the cortex among the terminating optic radiation axons. The recording electrode was usually located in the anterior half of the lateral geniculate body and histological studies in this laboratory (W. R. Hayow & C. Webb, unpublished) have shown that the stimulating electrodes were placed in a part of the cortex to which this portion of the geniculate projects (cf. Doty, 1958). Any combination of six pairs of stimulating electrodes could be chosen by means of six switches and, by trial and error, the combination was found which gave a large response with the least distortion due to stimulus escape. The stimulus artifact was further reduced by means of a balancing circuit.

In the experiments with D-lysergic acid diethylamide (LSD-25, Sandoz) the drug was introduced in the carotid stream by retrograde injection into the lingual artery. For details of the method see Bishop, Field, Hennessy & Smith (1958).

RESULTS

The details of the notation used to describe the various geniculate potentials are given in earlier publications from this laboratory (cf. Bishop & Evans, 1956). The two optic tract spikes and their corresponding post-synaptic spikes are designated t_1 and t_2 and r_1 and r_2, respectively. The corresponding synaptic potentials are s_1 and s_2 respectively and further subscripts of r (r_3, r_4, etc.) are used to refer to the repetitive waves (Fig. 1, *a*) that frequently follow the r_1 spike (Bishop, Jeremy & McLeod, 1953). The recording sites were usually situated in the anterior portion of the lateral geniculate body and the crossed optic pathway was nearly always used.

In determining the duration of the various slow post-synaptic potentials it has already been pointed out (see Methods) that the major source of error is due to interference from the background of 'spontaneous' slow waves of electrical activity. This may be largely offset by measuring a sufficiently large number of records. There are, however, other difficulties,

both theoretical and practical. When the afferent volley is restricted to t_1 fibres, the commencement of post-synaptic activity is clearly defined and there is therefore no difficulty with respect to the time of onset of potentials which are subliminal for a propagated response (synaptic potential (s_1), Fig. 5, *a*). The time of onset of the after-potentials which follow the propagated spike is however rather indefinite. Furthermore, both the synaptic

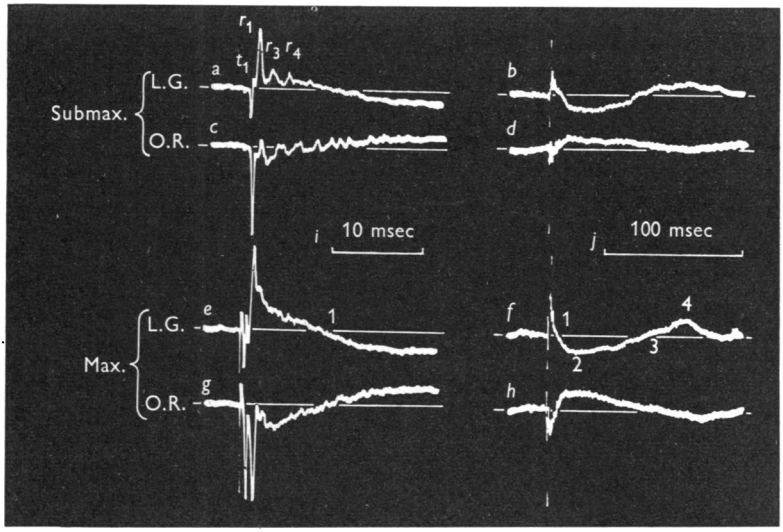


Fig. 1. Responses recorded in the lateral geniculate nucleus (L.G.) and in the optic radiation (O.R.) following single shocks applied to the contralateral optic nerve. t_1, presynaptic spike; r_1, r_3, r_4, post-synaptic spikes. Submaximal shock is 65% maximal for t_1. Maximal shock is maximal for whole optic nerve (t_1 and t_2). Numerals associated with traces *e* and *f* indicate how the time course of the after-potentials was measured. Time scale *i* refers to traces *a*, *c*, *e* and *g*, and time scale *j*, to traces *b*, *d*, *f* and *h*. Upward deflexions in this and subsequent records indicate negativity of micro-electrode.

potentials and the after-potentials associated with t_1 volleys are of such a low amplitude that their subsequent time course is also rather uncertain. Increasing the stimulus strength to include t_2 fibres increases the amplitude of the slow post-synaptic potentials, making them much easier to measure, but at the same time distorting their time course because of the later onset of the post-synaptic activity associated with the more slowly conducting group of afferent fibres. The times of onset of these later-arising potentials are also much more uncertain, not only because they are superimposed on potentials generated by the t_1 volley but also because the wave forms themselves may be difficult to interpret. This is particularly the case when the recording site is near the anterior pole of the lateral geniculate nucleus (Fig. 1, *e*). Although not many t_2 fibres project to the anterior

portion, those that do are among the fastest of the t_2 group, so that t_2 and r_1 spikes may be practically synchronous.

Because of these uncertainties measurements were usually made both with submaximal (t_1 only) and maximal optic nerve shocks. For practical purposes it was decided to make all measurements with respect to the stimulus artifact and subsequently to subtract the appropriate latency to arrive at the correct time course. It was decided also to regard the after-potentials as commencing at the same time as the synaptic potentials, i.e. before the spike potential in each case. With a t_1 volley the mean latency of the s_1 potential was 1·14 msec, but when the shock strength became maximal this latency shortened to 1·05 msec. With increasing shock strength the stimulating current spreads more proximally, so that the latency of the t_1 volley decreases from about 0·85 to 0·75 msec. The mean latency of the t_2 post-synaptic activity was 2·22 msec, corresponding to a t_2 volley latency of about 1·7 msec. In order to arrive at the correct time course for the synaptic potentials and after-potentials associated with a maximal optic nerve volley it was decided to subtract 1·6 msec from the measured values in each case, this being the mean of the latencies of the t_1 and t_2 post-synaptic potentials (i.e. 1·14 and 2·22 msec respectively).

In summary, therefore, all measurements were made with respect to the stimulus artifact and either 1·1 or 1·6 msec was subtracted, depending upon whether the stimulus was submaximal or maximal. This correction is, of course, only of significance in respect to the first 10–20 msec of post-synaptic activity. No systematic attempt was made to measure the amplitudes of the potentials.

After-potentials in lateral geniculate body

Systematic recordings were made in the nucleus at thirteen sites in eleven preparations. Responses to maximal optic nerve shocks were obtained in every case and, at seven of the recording sites, submaximal shocks were also used. Typical wave forms (labelled L.G.) are shown in Fig. 1; *a* and *b* are due to a submaximal shock and *e* and *f* to a maximal shock. The submaximal shock was, in this instance, about 65 % maximal for t_1. The after-potentials are seen to consist of a sequence of oscillations of potential, initially negative. These oscillations may continue for half a second or more (Fig. 8) until they gradually become indistinguishable from the slow waves of the background activity. The times of the significant features in the course of the after-potentials (and also the synaptic potentials, see below) have been measured at the times indicated by the numerals in Fig. 1, namely: (1), at the end of the first negative potential; (2), at the maximum amplitude of the first positive wave; (3), at the end of the first positive wave and (4), at the peak of the late negative wave (see

Tables 1–3). It was usual to record the wave forms at the two time-base durations of approximately 30 and 170 msec respectively so that the first and later portions of the response could be measured accurately. Each response was recorded between 10 and 50 times, the stimuli being applied at 5 sec intervals.

The small repetitive waves (r_3, r_4, Fig. 1, *a*) superimposed upon the first negative after-potential are due to the approximately synchronous repetitive firing of a considerable number of geniculate neurones (Bishop *et al.* 1953). While repetitive firing is a constant feature of the geniculate response, particularly in relation to the large fibres at low levels of stimulation, it is not usually so prominent as that shown in Fig. 1. These repetitive waves are nearly always sharply limited to the duration of the first negative after-potential, and it is obviously difficult to decide how much of the latter is true after-potential process and how much of it is due to summed asynchronous repetitive firing. The repetitive firing may be obscured or possibly eliminated when large stimulating shocks are used (Fig. 1, *e*). The relationship of the repetitive firing to the negative after-potential is shown more clearly in Fig. 2. Trace *a*, which is due to a shock submaximal for t_1, has been obtained on one preparation, and *b* and *c* from another preparation. In the latter instance maximal stimulation was used, the traces being recorded successively at the same site at different time-base sweep speeds. The repetitive firing stops sbruptly in every case as the trace goes positive. The relationship of repetitive firing to the slow negative potentials that may be recorded from the region of cell bodies has been observed by several investigators (e.g. Baumgarten & Jung, 1952; Tasaki, Polley & Orrego, 1954; Li, Cullen & Jasper, 1956). Strong support for the view that the slow negative potentials in the lateral geniculate nucleus act as a generator potential for the repetitive firing has been provided by single-unit studies in this laboratory (P. O. Bishop, W. Burke & R. Davis, unpublished).

The negative after-potential associated with a t_1 volley is usually not very prominent and may be obscured at its onset by a transient reversal of current flow that follows the discharge of the r_1 spike as the dendritic-cell body membrane becomes a source of current for the spike in the axon. By the time that the negative after-potential can again be recorded it may then be of very low amplitude. It is to be expected that increasing the shock strength would increase the amplitude of the after-potentials without very significantly altering their time course. Inspection of Fig. 1 and Table 1 shows that this is the case. The first negative after-potential has a duration slightly over 7 msec. The subsequent positive after-potential has a sharp decline to a maximum positivity at about 22 msec, thereafter returning rather more slowly to the base line at about 80 msec. The positive

after-potential is devoid of any evidence of repetitive firing. The late negative wave is usually more symmetrical in form, having a peak soon after about 100 msec (see below). Associated with this peak there is frequently a repetitive discharge of geniculate neurones, seen as unit spikes in Fig. 8, *b*, *c*. While the late negative wave was present in every experiment it was not always prominent. The subsequent oscillations of potential are difficult to distinguish from background activity. It should be stressed that occasionally the positive after-potential failed to regain the base line and occasionally also the tracing, although it crossed the base line, had not achieved a late peak of negativity by the time it reached

TABLE 1. Duration of after-potentials (msec)

(*a*) Recording in lateral geniculate nucleus

Afferent volley	No. of recording sites	1*	2	3	4
t_1	7	7·0 (4·1–12·3)	25 (21–37)	70† (51–96)	95† (89–119)
t_1 and t_2	13	7·3 (4·2–9·8)	22 (10–30)	80† (49–116)	100† (75–160)

(*b*) Recording in optic radiation

Afferent volley	No. of recording sites	1*	2	3	4
t_1	2	7·1	24 (17–31)	80† (64–91)	95† (84–102)
t_1 and t_2	6	8·6 (7·1–10·9)	33 (18–41)	90† (67–114)	100† (88–116)

The values given are mean values, the range of variation being given in parentheses.

* These numerals refer to the labelled wave forms in Fig. 1. † These figures are underestimates to the extent that certain records have been neglected (for explanation see text).

the edge of the cathode-ray tube screen. In arriving at the mean figures given above and in the preparation of the various tables these records were neglected. For this reason certain values in the tables have been labelled as approximate. The peak of the late negative wave therefore occurs somewhat later than the value of 100 msec given above.

There is probably no subliminal fringe associated with a maximal optic nerve volley, and the subliminal fringe associated with a submaximal t_1 volley is quite small (Bishop & Davis, 1960). The potentials described above can, therefore, be regarded as being generated in neurones all of which have been brought to discharge by the afferent volley.

After-potentials recorded in optic radiation

The cells in the lateral geniculate nucleus are arranged in sharply defined and fairly closely packed layers, and the axons issue from the dorsal surface, proceeding upwards to the cerebral cortex. At the upper surface of the nucleus, therefore, the axons above are fairly sharply

separated from the cellular elements of the nucleus below. The potential fields in the nucleus will be determined very largely by currents flowing into or out of dendrites and cell bodies, though the fields will be modified by the presence of the initial portions of the optic radiation axons among their cells of origin. Above the nucleus, on the other hand, the flows of current will be associated only with axons. A study of the distribution of the flows of current associated with such a favourable histological arrangement provides valuable clues to the nature of the after-potentials and the structures responsible for their production. Bishop (1953) and Bishop & McLeod (1954) have already made a study of the distribution of synaptic currents, particularly in relation to blocked geniculate synapses, during the first 5–10 msec after the arrival of the afferent volley. Vastola (1957, 1959) has also carried out similar investigations in relation to the slow after-potentials following both orthodromic and antidromic activation. An earlier report (Bishop, Burke, Davis & Hayhow, 1958) and the results reported here amplify and extend these investigations.

After-potentials were systematically recorded at six sites in five preparations about 1 mm above the dorsal surface of the nucleus, and with both submaximal and maximal optic nerve shocks. The records (labelled O.R.) shown at *c*, *d*, *g* and *h* in Fig. 1 are fairly typical, the submaximal shock being again 65% maximal for t_1. These records were actually obtained about 4 hr after traces *a*, *b*, *e* and *f* in Fig. 5, when the synapses had recovered from block caused by the drug LSD (for details see below). The presynaptic volley is now recorded as an entirely positive spike and the post-synaptic spike is here a low-amplitude initially-positive triphasic response. Except for the reversal in polarity, the after-potentials are very similar in form and time course to those recorded in the nucleus. They are, however, of lower amplitude and it was usually only possible to make satisfactory measurements when maximal optic nerve shocks were used. The times of occurrence of significant features of the wave forms, measured in the same way as in the case of the intra-nuclear responses, are given in Table 1 (*b*). The initial slow positive wave has a mean duration of 8·6 msec. The subsequent slow negative wave reaches a peak at about 33 msec and declines to the base line again at about 90 msec. The latter time and the late positive wave that followed were very variable and difficult to measure. Not infrequently the negative wave failed to regain the base line and the late positive wave was in fact only positive-going and not actually positive.

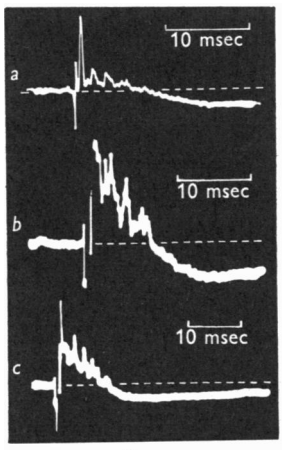
It has already been noted that, in the nucleus, repetitive spikes were superimposed on the initial negative after-potential and that unit spikes were also frequently recorded on the late negative wave. In the optic radiation these are again recorded but this time superimposed upon and

generally confined to the corresponding positive waves. The initial positive wave in response (*c*) of Fig. 1 is largely obscured by a train of repetitive spikes which is no longer evident when the optic nerve shock becomes maximal (Fig. 1, *g*). There can be little doubt that the repetitive waves in Fig. 1, *c* represent the nearly synchronous repetitive firing of single optic radiation axons, because we have recorded repetitive firing of this kind from single radiation axons with micropipette electrodes having tip diameters of 0·5 μ or less. The repetitive firing described above is very similar to the recurrent efferent discharge that may occur in a few motor axons following antidromic impulses in the same axons (Renshaw, 1941). This discharge is again limited to the slow negative phase of the antidromic potentials (Brooks, Downman & Eccles, 1950*a*).

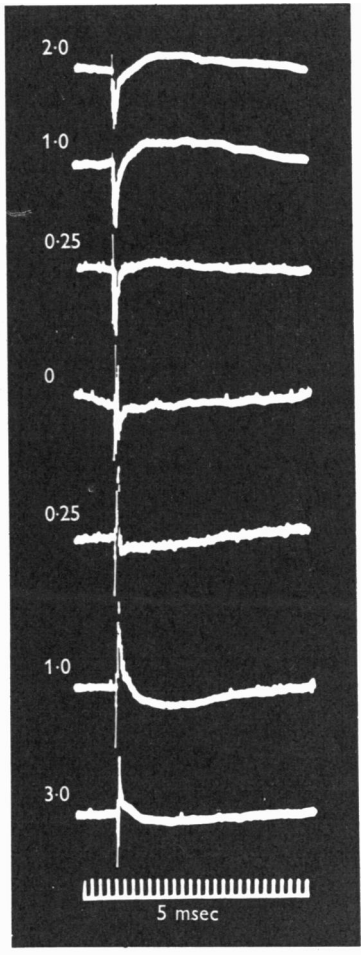
As the micro-electrode is inserted down through the optic radiation into the lateral geniculate nucleus the after-potentials must at some stage reverse their polarity. A detailed study of this change in polarity was made in a number of preparations by recording the after-potentials at successive small intervals, both as the micro-electrode was inserted downwards into the nucleus and also on its withdrawal. The records shown in Fig. 3*A* were selected from such a series. At level (0) the trace is virtually isopotential throughout the time when after-potentials are recorded at sites above and below this level. In five recording series in four preparations this reversal level occurred at a mean depth of 12·0 mm from the surface of the cerebral cortex, but for descriptive purposes the reversal level will be regarded as zero. Thus the numerals in Fig. 3*A* indicate recording sites in millimetres above and below this level. The reversal point was necessarily somewhat arbitrary, since it was taken to be that level at which the oscillograph trace was most nearly isopotential taking into account interference from background activity. Approaching from above, the after-potentials appear between 2 and 3 mm above zero and increase in voltage until the slow negative wave reaches a maximum amplitude at a mean level of 0·9 mm above. Thereafter the potentials decline until zero level is reached. Just below this a low-amplitude slow positive wave appears which has an abrupt onset immediately after the end of the spike potential. On penetrating more deeply a small negative-going hump appears on the positive wave 6–8 msec after the start of post-synaptic activity; the hump grows in amplitude, helping to fill out and make negative the earliest portion of the positive wave at depths between 0·2 and 0·55 mm below zero. The after-potentials, negative and positive, thereafter grow in amplitude so that the positive wave reaches a maximum amplitude at a mean level of 1·2 mm below. On going deeper the amplitudes gradually diminish, again without change in form.

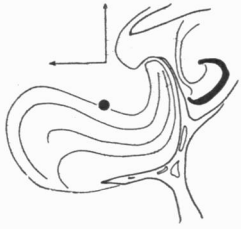
Although the pattern described above was usually observed, very

occasionally the small negative-going hump was observed at levels at and even above zero. In the centre trace of Fig. 4*A*, which was regarded as zero level, a very small negative hump can be observed immediately after



2





3*B* 3*A*

Fig. 2. Repetitive firing of geniculate neurones in relation to the negative after-potential. *a*, response following a single shock to the contralateral optic nerve which was submaximal for t_1; *b* and *c*, responses in another preparation following single shocks which were close to maximal for t_1 and t_2.

Fig. 3. *A*. Responses following single shocks applied to the contralateral optic nerve recorded, at distances indicated in mm, above and below the locus at which the trace labelled 0 was obtained. *B*. Outline drawing of a parasagittal section of the lateral geniculate nucleus showing recording site (●) used to obtain record labelled 0 in Fig. 3*A*; arrows indicate horizontal and vertical Horsley–Clarke planes.

the spike potentials. Rarely a similar but still negative-going hump has been recorded at levels above zero, where it appears on the initial positive wave. The significance of the negative-going hump and its relation to the negative after-potential is not clear. It is possibly due to a late post-synaptic propagated discharge from the lateral geniculate nucleus.

It was clearly important to determine the precise histological location of the tip of the micro-electrode at zero level and this was done by labelling the recording site with electrolytically deposited iron. Due allowance for slow-wave background activity is fairly readily made if the sequence of cathode-ray tube photographs is available for study. This was obviously not practicable and judgement concerning the location of the zero level had to be made by observing the cathode-ray tube screen at the time of the actual recording. Despite this uncertainty, the labelled sites were all very close to the upper surface of the lateral geniculate nucleus. The location of the zero level for the series in Fig. 3*A* is shown as a filled circle in the parasagittal outline drawing of the lateral geniculate body (Fig. 3*B*). Because of the larger amplitude of the after-potentials maximum stimulation was always used to locate the zero level. In one experiment a bank of three micro-electrodes was assembled with the shafts, 1 mm apart, in parallel array and the tips offset in depth, again 1 mm apart. The bank was gradually inserted vertically down through the cortex, with the plane of the electrodes parallel to the Horsley–Clarke anteroposterior plane, until the central electrode was judged to be at zero level. A series of recordings were then taken from the three electrodes (Fig. 4*A*) and then each site was labelled with iron. The labelled sites are shown in the micro-photograph (Fig. 4*B*) of a parasagittal section through the nucleus. The zero level is again at the upper surface of the cellular layers.

It is interesting to observe that at about the zero level the traces nearly always showed much greater evidence of unit activity and repetitive firing. Although the electrodes used were not suitable for unit recording, increased unit activity round the zero level is clearly evident in the traces in Fig. 3*A* and 4*A*, occurring along the whole length of the traces.

Synaptic potentials

Bishop & McLeod (1954) studied the synaptic potentials that may be recorded in and about the lateral geniculate body when synaptic transmission has been blocked. They showed that the synaptic potential produced by a t_1 volley is recorded as a negative wave in the nucleus, reversing to a positive wave when the recording site is in the optic radiation above the nucleus. Particular attention was given to the level of reversal of this potential which, in the case of a contralateral volley, was shown to be at, or very close to, the upper surface of the nucleus. At that time also it

was noted that the synaptic potential was followed by a prolonged after-positivity which was recorded as an after-negativity in the optic radiation. Further observations in this laboratory while studying the blocking action

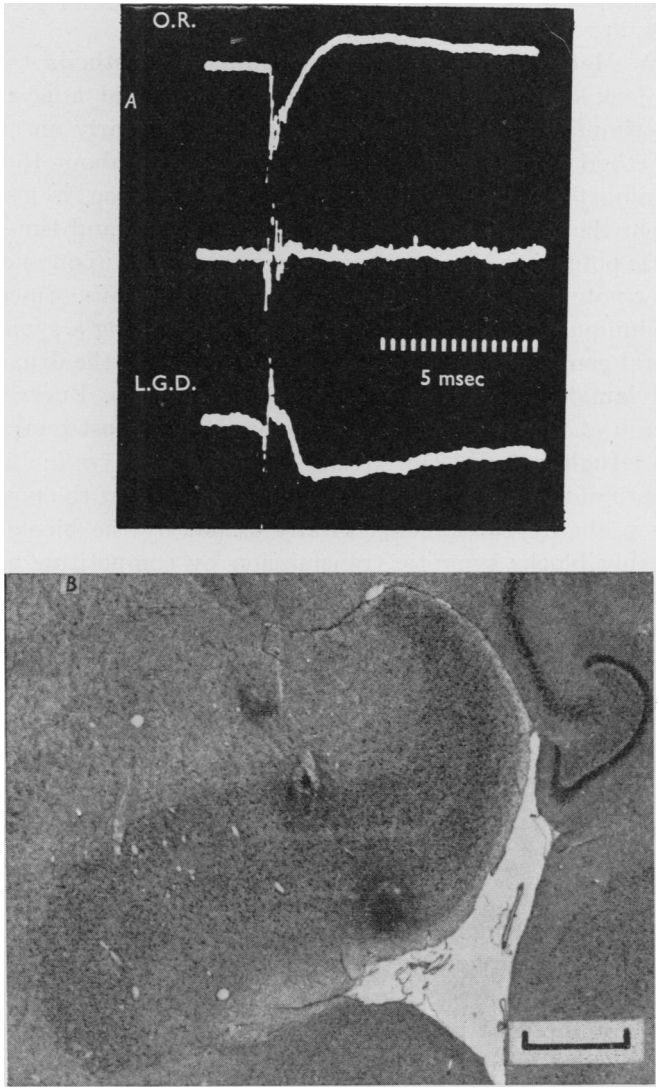


Fig. 4. *A*. Responses following single maximal shocks applied to the contralatera optic nerve and obtained by inserting three micro-electrodes simultaneously into the region of the lateral geniculate nucleus. O.R., optic radiation; L.G.D., lateral geniculate nucleus. *B*. Microphotograph of a parasagittal section through the lateral geniculate nucleus (Nissl stain). The recording sites used to obtain the records in Fig. 4*A* are indicated by the three 'blue' spots. Scale, 1 mm.

both of drugs and of prolonged repetitive stimulation confirmed these earlier findings, and the striking resemblance, both in respect to form and time course, between the synaptic potential and after-potentials as described above prompted a more detailed study of the synaptic potentials, particularly in respect to their later time course.

Bishop & McLeod (1954) used three different methods to obtain a synaptic block, namely asphyxia, deep pentobarbital anaesthesia and repetitive stimulation. The first two methods were clearly unsatisfactory. The last method, which entailed placing a test volley about 15 msec after a conditioning tetanus of 3–5 volleys at about 300/sec, is also unsatisfactory from the point of view of studying the form and time course of the synaptic potentials because they are superimposed upon and distorted by the after-potentials following the conditioning tetanus. Since then two further techniques have become available for producing a synaptic block in the lateral geniculate nucleus, namely the action of the drug D-lysergic acid diethylamide (LSD) (Evarts *et al.* 1955; Bishop, Burke & Davis, 1958; Bishop *et al.* 1959*b*) and the phenomenon of post-tetanic delayed depression (Hughes *et al.* 1956; Evarts & Hughes, 1957*a*, *b*; Bishop *et al.* 1959*a*). Both of these methods have the advantage that the post-synaptic neurone is probably not affected to any extent by the blocking action. LSD probably blocks synaptic transmission by competitive antagonism to the normal transmitter and the phenomenon of post-tetanic delayed depression is probably due to presynaptic changes. In studying the synaptic potentials produced by these means, two criteria were used to decide when synaptic transmission was blocked, namely, the absence of any spike-like discharges on the post-synaptic wave form and the absence of any cortical response. The cortical response was first sampled at a number of places and the site chosen for the monitor electrode was always the one which gave the maximal response. The cortical response was then used as a continuous monitor of the level of the geniculate block.

Although about 100 μg LSD injected into the carotid artery will normally block transmission through the lateral geniculate nucleus, recovery commences almost immediately. It was, in fact, difficult to maintain a complete block for the time necessary to take a sufficiently large number of records, and even with the much larger doses that were actually used (500 μg repeated at intervals of 10–20 min) the block was usually rather uneven. On the other hand, the phenomenon of post-tetanic delayed depression produces a very stable and prolonged block in transmission. This depression is produced by tetanic stimulation of the optic nerve for 15 sec using maximal stimuli at about 400/sec. Following the tetanus the geniculate response is depressed for a few seconds, may then be larger than

normal (1–2 min), and finally is markedly reduced, and a complete block may remain for a period lasting hours.

In a number of preparations the attempt was made to compare the after-potentials and the synaptic potentials by recording them both at the same locus, in the nucleus on the one hand and in the radiation on the other. As full recovery from a large dose of LSD may take several hours and the effects of tetanic stimulation are even more prolonged, it was not possible to take all the recordings at only two sites, the first in the nucleus and the second in the radiation. Figures 1 (*a–h*), 5 (*a–h*) and 6 (*g–j*), which were all taken from the one preparation, illustrate the procedure that was adopted. Records *a*, *b*, *e* and *f* in Fig. 1 were first obtained in the nucleus

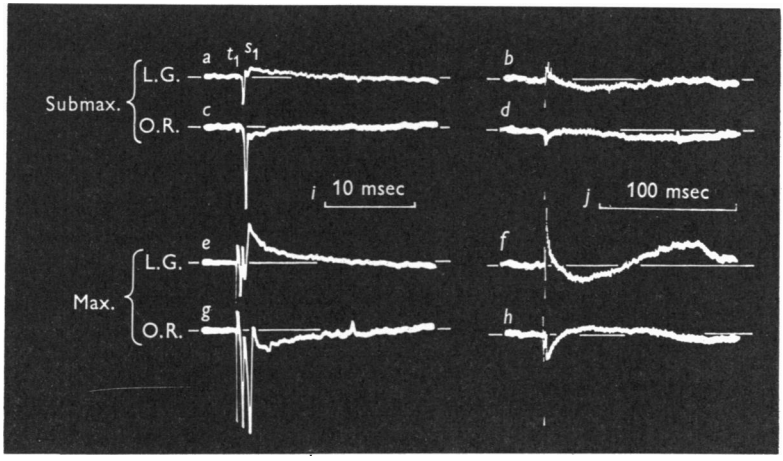


Fig. 5. Responses recorded in the lateral geniculate nucleus (L.G.) and in the optic radiation (O.R.) following single submaximal and maximal shocks applied to the contralateral optic nerve during LSD synaptic block. Same preparation and similar recording conditions as those used to obtain records in Fig. 1. t_1, presynaptic spike; s_1, synaptic potential. Records arranged as in Fig. 1. For details see text.

by using two strengths of stimulation which were thereafter kept constant throughout the experiment. The synapses were then completely blocked with repeated injections of LSD and the records *a*, *b*, *e* and *f* in Fig. 5 taken. About 4 hr later, when recovery from the drug was complete, the electrode was moved up into the optic radiation and records *c*, *d*, *g* and *h* in Fig. 1 were taken. Further amounts of LSD were then injected and records *c*, *d*, *g* and *h* in Fig. 5 were obtained during the height of synaptic block. Following partial recovery from the drug, a 15 sec tetanus was delivered to the optic nerve and, when complete synaptic block had been re-established, records were again taken at the radiation site (*i* and *j*, Fig. 6). The electrode was then returned to the nucleus as near as possible

to the site used to obtain the earlier geniculate responses and the records *g* and *h* of Fig. 6 were taken. Thus records *g* and *h* of Fig. 6 are not strictly comparable with the earlier geniculate records, though they are closely similar to them. It is interesting to note that after all these procedures in this experiment, a 10 min tetanus to the optic nerve, as is usual, restored the geniculate response to normal (cf. Evarts & Hughes, 1957*b*; Bishop *et al.* 1959*b*). The other traces in Fig. 6, taken from another preparation, also show the responses to maximal afferent volleys during post-tetanic delayed depression, in the nucleus (*a* and *b*) and in the radiation (*c* and *d*) respectively.

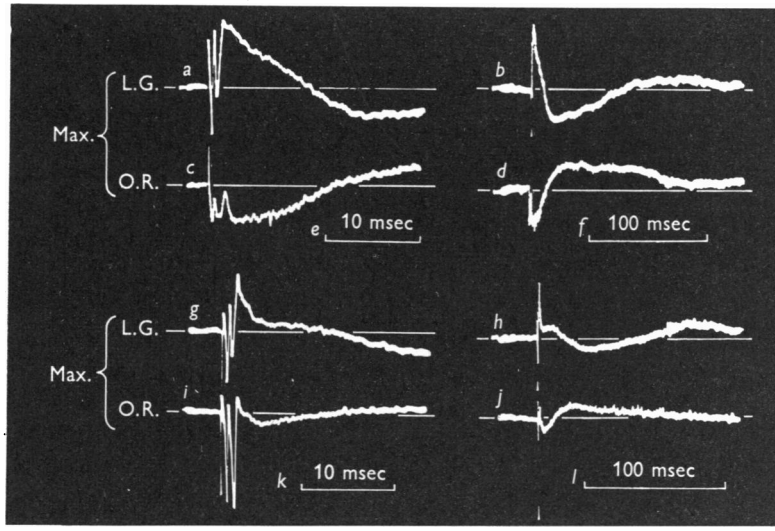


Fig. 6. Responses recorded in the lateral geniculate nucleus (L.G.) and in the optic radiation (O.R.) following single maximal shocks applied to the contralateral optic nerve during synaptic block obtained by post-tetanic delayed depression. Records *g*, *h*, *i* and *j* were obtained from the same preparation and under similar recording conditions as those of Fig. 1. Records *a*, *b*, *c* and *d* were obtained from another preparation. Each time scale refers to the two traces immediately above. For details see text.

The synaptic potentials found in both kinds of block were measured in the same way as the after-potentials and the results were pooled to produce Table 2. It can be seen that there is again relatively little difference between the responses due to volleys submaximal for t_1, and maximal nerve volleys. The initial negative wave has a duration of about 12 msec, the succeeding positivity reaches its maximum at about 25 msec, the base line is recrossed soon after 60 msec and the late negative wave has a peak at about 100 msec. Insufficient recordings have been made in the optic radiation for a satisfactory comparison between the time course of

the synaptic potentials in that site and in the nucleus. Except for the reversal in polarity the potentials are however very similar. The after-potentials and synaptic potentials recorded at the same locus are compared in Table 3. The initial negativity of the synaptic potential in both forms of block lasted consistently longer than the corresponding negativity of

TABLE 2. Duration of synaptic potentials (msec)

(*a*) Recording in lateral geniculate nucleus

Afferent volley	No. of recording sites	1*	2	3	4
t_1	5	11·8 (9·8–16·9)	33 (24–41)	70† (51–85)	100† (97–107)
t_1 and t_2	5	11·9 (8·9–16·1)	25 (18–33)	61 (40–92)	103 (96–111)

(*b*) Recording in optic radiation

Afferent volley	No. of recording sites	1*	2	3	4
t_1	2	8·6 (6·9–11·1)	37 (27–50)	68† (64–71)	95† (82–105)
t_1 and t_2	2	11·9 (11·0–12·9)	33 (27–40)	86	118 (112–128)

The values given are mean values, the range of variation being given in brackets.

* These numerals refer to the labelled wave forms in Fig. 1. † These figures are underestimates to the extent that certain records have been neglected (for explanation see text).

Note. At two sites in the nucleus and at one site in the radiation synaptic potentials were recorded both as a result of LSD and post-tetanic delayed depression.

TABLE 3. Duration of after-potentials and synaptic potentials (msec)

		1	2	3	4
A	After-potentials	7·7	22	68	101
	LSD synaptic potentials	13·1	23	47	98
B	After-potentials	8·5	23	74	103
	PTDD synaptic potentials	10·3	28	84	114

Mean values at times indicated by numerals (1–4) in Fig. 1.

the after-potential. There is, however, probably no significant difference between the responses as far as the time course of the remaining features of the waves is concerned. Earlier histological studies (Bishop & McLeod, 1954) established that the site of the reversal of polarity of the synaptic potentials was at the upper surface of the nucleus and this has been confirmed by further work in this laboratory using maximal optic nerve volleys (P. O. Bishop & R. Davis, unpublished observations).

Antidromically evoked after-potentials

The form of the antidromically evoked after-potentials is seen in Fig. 7. In our few experiments with antidromic stimulation measurements of the duration of the negative after-potential were uncertain because of distortion by stimulus escape and the difficulty in deciding when this escape

had been adequately controlled by the balancing circuit in the stimulus leads. In the case of the experiment illustrated in Fig. 7, with the stimulus artifact judged to be adequately controlled, the onset of the initial negative spike had a mean latency of 0·7 msec and the mean duration of the after-negativity was 4·9 msec (range 4·3–6·3) measured from the onset of the negative spike. Thereafter the trough of the after-positivity was reached at 16 msec, the base line was recrossed at 110 msec and the late negative wave had a peak at 150 msec. The after-positivity and later time course

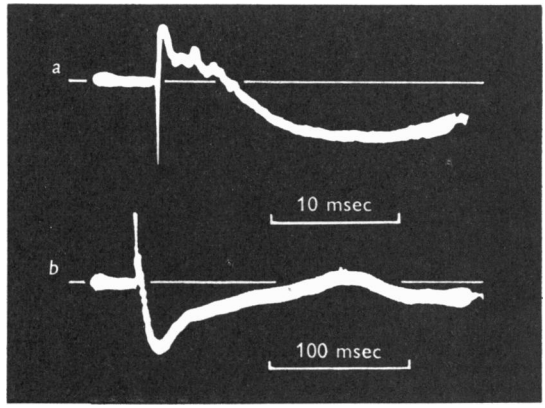


Fig. 7. Responses recorded in the lateral geniculate nucleus following antidromic volleys discharged down the optic radiation. The same response has been recorded successively with fast and slow time bases respectively.

of the orthodromic response were closely similar (Table 4*A*). The repetitive firing of geniculate neurones which occurs in response to antidromic volleys (Fig. 7, *a*) is again limited to the duration of the negative after-potential. This discharge almost certainly involves the repetitive firing of the same neurones, because the latter has actually been observed to occur when recording from single antidromically activated geniculate neurones (P. O. Bishop, W. Burke & R. Davis, unpublished). A similar antidromic repetitive discharge occurs in the ventral roots of the spinal cord (Renshaw, 1941; Brooks *et al.* 1950*a*; Granit, Pascoe & Steg, 1957). Our findings (Fig. 8) confirm Vastola's observation that the after-positivity in the antidromic responses corresponds to that seen in the orthodromic responses recorded at the same points. Records *a*–*g* of Fig. 8 were obtained from the same experiment as those of Fig. 7. Preliminary observations indicate that the antidromic after-potentials are unchanged by the drug LSD or during the period of post-tetanic delayed depression of the orthodromic response.

Slow geniculate rhythms

In seven preparations a study was made of the spontaneous slow oscillations of potential that may be recorded in the lateral geniculate nucleus. Except on one occasion both optic nerves were prepared for

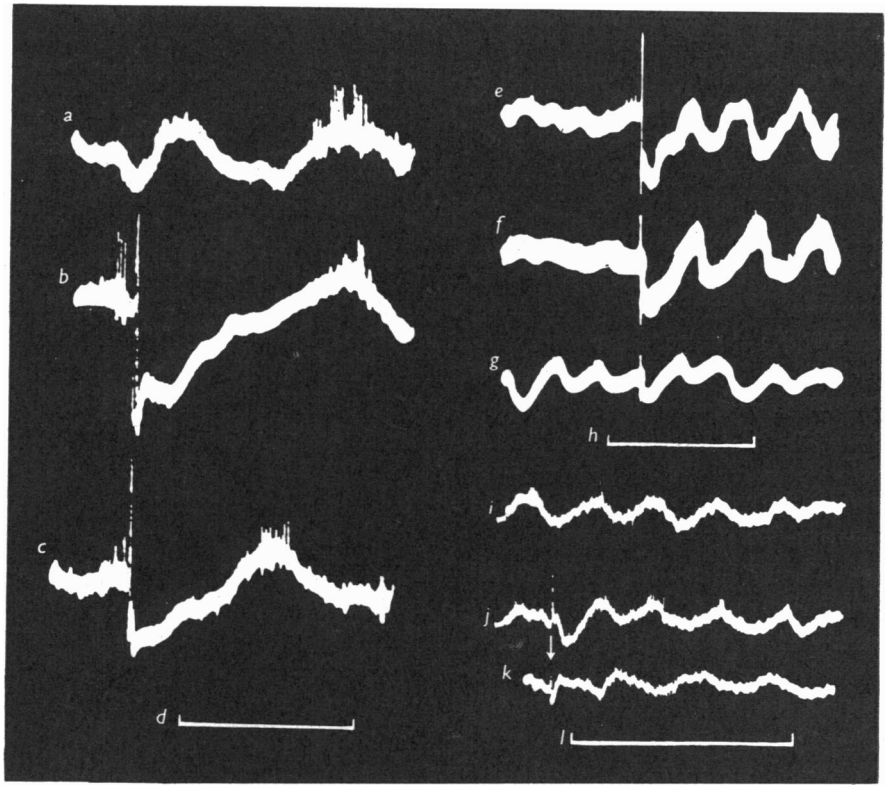


Fig. 8. Spontaneous and evoked slow rhythms. Traces *a–g* were obtained at the one site in the lateral geniculate nucleus. *a*, spontaneous rhythms; *b* and *e*, responses to maximal stimulation of the contralateral optic nerve; *c*, *f* and *g*, responses to antidromic volleys discharged down the optic radiation. *i*, *j* and *k* were obtained from another preparation during PTDD synaptic block, *i* and *j* being taken at a common site in the nucleus, and *k* just above the nucleus in the optic radiation. *i*, spontaneous rhythms; *j* and *k* responses to maximal stimulation of the contralateral optic nerve. Arrow indicates stimulus artifact in trace *k*. Each time scale refers to the three traces above it; *d*, 200 msec; *h*, 0·5 sec and *l*, 0·5 sec.

stimulation. Since this involves the removal of most of the retina and probably the inactivation of the small remnant, the spontaneous potentials do not depend upon retinal mechanisms. Recordings were taken about six or more hours after the induction of the anaesthesia (see Methods). The slow waves under these conditions are fairly irregular, being broken up

by fast activity, but there is a predominant rhythm between 6 and 15 c/s (mean 9 c/s; Fig. 8, *a*). Bursts of unit-spike activity also occur particularly in relation to the negative-going peaks of the waves. It is possible that the rhythmical bursts of repetitive firing that may follow single orthodromic volleys (Bishop, Burke, Davis & Hayhow, 1958) are also related to these spontaneous geniculate rhythms.

In four preparations the effect of a sudden increase in the depth of anaesthesia was studied. Within 1–2 min after the intravenous administration of 0·3–0·5 ml. sodium pentobarbitone solution the fast activity was markedly reduced and the predominant rhythm slowed to 3–9 c/s (mean 5 c/s). The waves generally increased in amplitude and the bursts of unit-spike activity were reduced (1 experiment) or eliminated (3 experiments).

The relationship of the spontaneous waves to those produced as a result of orthodromic and antidromic volleys has also been studied (Fig. 8). If either orthodromic or antidromic volleys are fired into the nucleus at a time when the spontaneous waves are minimal or absent, long-continuing oscillations of potential are produced which are indistinguishable from those arising spontaneously (Fig. 8, *e*, *f*). In these circumstances it is hard to avoid the conclusion that the mechanism responsible for the oscillations was activated by the arriving volleys. It is also obviously difficult to determine when triggered activity ceases and 'spontaneous' activity takes over. If we measure the first cycle of triggered activity from the stimulus artifact to the peak of the late negative wave (labelled 4 in Fig. 1) it has a mean duration of slightly over 100 msec (Table 1), corresponding to a frequency of about 9 c/s, the mean frequency of the spontaneous oscillations. When spontaneous oscillations of potential are occurring in the nucleus the arriving volley appears largely to halt this rhythm and to trigger it anew in phase with the stimulus (Fig. 8, *g*).

In one experiment a detailed comparison was made between the spontaneous rhythms and those produced by contralateral, ipsilateral and antidromic volleys, the latter three being again measured from stimulus artifact to the first late negative wave. The results (mean values) are shown in Table 4*A*. In the same experiment the later waves in the antidromic response were compared with the spontaneous rhythms recorded immediately beforehand, with an oscillograph time base permitting 3 or 4 later waves to be measured. The antidromically evoked waves had a mean frequency of 6·1 c/s, while that of the spontaneous waves was 6·2 c/s.

Spontaneous slow oscillations of potential also occur in the lateral geniculate nucleus, both when the synapses are blocked by LSD and during post-tetanic delayed depression (PTDD). In one experiment (Table 4*B*) the mean frequency of the spontaneous rhythms was 8·7 c/s, and some hours later repeated injections of relatively large doses of LSD were given

but the spontaneous rhythms still continued though at a slightly reduced frequency (6·9 c/s) despite the synaptic block. In the same experiment the duration of the first cycle of the response to orthodromic volleys remained virtually unchanged during subsequent LSD block and during PTDD.

A detailed comparison was made in one experiment during PTDD between the spontaneously occurring slow waves, as recorded in the nucleus (Fig. 8, *i*), and the slow waves following orthodromic volleys recorded both in the nucleus (Fig. 8, *j*) and in the optic radiation (Fig. 8, *k*). The time base used allowed 4 or 5 waves to be measured in each sweep.

TABLE 4. Relationship between spontaneous and evoked potentials in two preparations (*A* and *B*)

		Mean interval (msec)	Frequency (c/s)
A	Spontaneous rhythms*	145	6·9
	After-potentials† following a		
	Contralateral volley	155	6·5
	Ipsilateral volley	140	7·1
	Antidromic volley	160	6·3
B	Spontaneous rhythms*		
	Before LSD	115	8·7
	After LSD	145	6·9
	Responses† to orthodromic stimulation		
	Normal after-potential	125	8·0
	LSD synaptic potential	115	8·7
	PTDD synaptic potential	115	8·7

* Measured between peaks of rhythmic oscillations.
† Measured from stimulus artifact to peak of the first late negative wave.

The mean frequency of the spontaneous rhythms was 6·3 c/s, while the mean frequency of the evoked slow waves was 7·1 c/s, both in the nucleus and in the optic radiation. It can be seen from records *i* and *j* of Fig. 8 that the nuclear and optic radiation waves remain approximately 180° out of phase throughout almost the whole duration of the sweep. It is probable therefore that the optic radiation slow waves arise as a result of electrotonic propagation of oscillations of membrane potential from the cell body and dendrites of the geniculate neurones.

DISCUSSION

General considerations

In order that the electrical records obtained from cell populations may be correlated with histological structure a detailed knowledge is required of the geometrical arrangement of the functionally distinct portions of the neurone both within the individual neurones themselves and as between one neurone and another. The relevant information that is

available in relation to the lateral geniculate nucleus has already been discussed in earlier analyses of this kind (G. H. Bishop & O'Leary, 1942; Bishop, 1953; Bishop & McLeod, 1954; Vastola, 1957). To assist the present discussion it is desirable to emphasize some of the structural features of the principal cells, i.e. those giving rise to axons that proceed upwards in the optic radiation. The dendritic shafts arborize extensively in all directions away from the cell body (Taboada, 1927). This predominantly radial arrangement of the dendrites with respect to the individual neurone is however obscured in the population by the extensive intertwining of the dendrites from adjacent neurones. The general orientation of the dendrites is therefore essentially random. As far as the axons are concerned, from whatever part of the cell they arise they quickly achieve a roughly parallel orientation as they pass towards the upper surface of the nucleus and beyond into the optic radiation.

Considering the events in synaptic transmission it may be supposed that the impulses in the presynaptic endings lead to local depolarizations of the post-synaptic membrane. The presence of local sources and sinks of current on dendrites which are randomly arranged with respect to one another would not produce a potential field effective for recording. A post-synaptic potential would not therefore be recorded until the depolarization of the dendritic tree and cell body had become sufficiently widespread for significant currents to be drawn from the portions of the radiation axons towards the upper surface of the nucleus and beyond into the optic radiation. In this region the separation of axons on the one hand from cell bodies and dendrites on the other leads to a corresponding separation of sources and sinks of current and hence of positive and negative fields. Even considering a single cell the radial arrangement of the dendrites means that the initial portion of the axon will be electrically hidden, so that net outward flow of membrane current will be found only as the axon approaches and leaves the limits of the field of distribution of the cell's dendrites.

Several important consequences follow from the above considerations. It will not be easy to distinguish between dendritic and cell-body potentials, since both are recorded only by reason of electrotonic current spread up the radiation axons and the recording of dendritic potentials must necessarily involve the cell body. Furthermore, although there is reason to believe from a study of single-unit records that the initial segment of the axon has properties which distinguish it from other parts of the neurone (Freygang, 1958; P. O. Bishop, W. Burke and R. Davis, unpublished; cf. Eccles, 1957), it is likely that under the present recording conditions its contribution will be swamped by the potentials of dendritic and cell-body origin. Neither can the records described above provide any direct evidence for

conduction along dendrites, although it has been suggested on other evidence (Bishop, 1953; Bishop & McLeod, 1954; Freygang, 1958) that dendritic conduction probably does not occur in the lateral geniculate nucleus. Since the volume conductor records of dendritic and cell-body potentials are obtained because of the difference in potential which exists between the surface of the dendritic-cell body membrane and that of the axon, any potential changes that occur in the axon will have a distorting effect on the former potentials. These effects will be minimal when synaptic transmission is blocked but the presence of a spike and after-potentials in the axons will lead to important modifications of the dendritic and cell-body potentials.

The above considerations do not, of course, necessarily apply to extracellular recordings made with micro-electrodes having tip diameters of $0{\cdot}5\,\mu$ or less, since with their aid it is possible to record local intensities of membrane current associated with particular parts of individual cells to the exclusion of the general field potentials. For this reason extracellular records obtained with such micro-electrodes differ markedly from those obtained with the micro-electrodes used in the present study (Freygang, 1958; Bishop, Burke & Davis, 1958).

Synaptic potentials

It will be convenient to discuss the synaptic potentials first, since changes in the membrane potential of the post-synaptic axons are not directly involved in this case. Analysis of the spatial distribution of the sources and sinks of current indicates that the axonal changes occur only as a result of electrotonic spread of current from the cell body and dendrites. The currents arise as a result of oscillation in the membrane potential of the cell body and dendrites. As was pointed out above, our records do not allow separate cell-body and dendritic components to be distinguished, although it is highly probable that both structures are actively involved (cf. Fatt, 1957*a*, *b*; Curtis & Eccles, 1959; Rall, 1959). An initial depolarization (negative) phase, lasting about 12 msec, is followed by a hyperpolarization (positive) phase which has a trough at about 25 msec after the onset of post-synaptic activity. The base line is crossed again at about 60 msec, the late negative wave (peak at about 100 msec) leading on to further oscillations of potential which are difficult to distinguish from background slow-wave activity. It should be emphasized again that the mean values for the duration of the positive and the late negative waves given above are underestimates (p. 520).

Bishop (1953) and Bishop & McLeod (1954) noted that the initial post-synaptic negativity is followed by a long-lasting positivity, but the phenomenon was not further investigated at that time. The synaptic

potentials studied by Bishop & McLeod (1954) had a much faster time course than those examined here, presumably because they were produced by a test shock timed to coincide with the trough of the positive after-potential that follows a brief high-frequency train of conditioning shocks. Under these circumstances the synaptic potentials are distorted by the currents associated with the after-potentials in the cell bodies and axons. In other studies from this laboratory the synaptic potentials reported here have been shown to be capable of summation, so that transmission becomes possible through synapses which were otherwise blocked (Bishop *et al.* 1959*a*, *b*). Extracellular synaptic potentials can also be recorded from single geniculate cells (Freygang, 1958; Bishop, Burke & Davis, 1958) though it is clear that they must bear a rather indirect and complex relationship to the potentials recorded from populations of geniculate cells.

The geniculate synaptic potentials reported here are very similar to the so-called 'focal synaptic potentials' recorded with extracellular micro-electrodes from motoneurone pools in the spinal cord (Renshaw, 1946; Brooks & Eccles, 1947; Lloyd & McIntyre, 1949; Brooks, Downman & Eccles, 1950*a*, *b*). The spatial distribution of the current flow (Eccles, Fatt, Landgren & Winsbury, 1954) in relation to the dendrites, cell bodies and axons of the motoneurones also resembles that found in the lateral geniculate nucleus. The motoneurone focal synaptic potential has a duration of about 10·5 msec (Fig. 9 of Brooks & Eccles, 1947) and a late after-positivity of up to 50 msec was noted by Brooks *et al.* (1950*b*). The late after-positivity is much more clearly seen in the ventral-root recording after electrotonic spread along the intramedullary motor axons. With ventral-root recording maximum positivity is reached at 40 msec and the base line is regained at 100 msec (Brooks *et al.* 1950*b*). It is to be expected that the time course of the early phases of the ventral root recordings will be distorted by passive membrane electrotonus (Brooks & Eccles, 1947), though the late positive phase should be relatively unaffected. It is rather surprising that Frank & Fuortes (1955) did not find any evidence of these slow potentials in their intracellular records from intramedullary motor axons (cf. also Coombs, Curtis & Eccles, 1957). Similar focal and ventral-root synaptic potentials have been recorded from the frog's spinal cord (Brookhart & Fadiga, 1960).

The focal synaptic potentials can be clearly correlated with the intracellularly recorded synaptic potentials (Brock, Coombs & Eccles, 1952; Fatt, 1957*b*). The intracellular excitatory synaptic potential, however, reaches its summit more slowly and has a longer time of decay. Interest has centred around the time constant of decay of the intracellular excitatory synaptic potential and no systematic measurements seem to have

been made of the duration of the phase of depolarization. Published records, indicating a value between 15 and 20 msec, are very variable, presumably reflecting differences in the geometrical distribution of the synaptic depolarizations with respect to the locus of the micro-electrode and the distorting effect of the cable properties of the dendrites (cf. Frank & Fuortes, 1956; Fatt, 1957*b*; Rall, 1959). The initial depolarization is followed by a long-lasting hyperpolarization (Brock *et al.* 1952; Coombs, Eccles & Fatt, 1955). Maximum hyperpolarization, in one instance, was reached at about 35 msec and the total duration was about 100 msec, both values being similar to the positivity of the ventral root recordings. No later fluctuations of membrane potential appear to occur in the motoneurone. The phase of depolarization is clearly correlated with Lloyd's (1946) heterosynaptic facilitation curve and the late after-positivity can be correlated with a phase of depression both in respect to the amplitude of a testing focal synaptic potential and the level of reflex excitability (Brooks *et al.* 1950*b*). Coombs *et al.* (1955) suggest that the hyperpolarization phase of the synaptic potential is probably a true after-potential developing as an immediate consequence of the preceding depolarization, and therefore a partial activation of a process more intensively activated by the spike.

After-potentials

The after-potentials which follow the discharge of the geniculate cells resemble the synaptic potentials. The initial negative after-potential, lasting about 7·3 msec, is followed by an after-positivity which has a trough at 22 msec. The base line is crossed again at 80 msec, the late negative wave (peak at about 100 msec) leading on to further oscillations of potential that are again difficult to distinguish from background slow-wave activity. The orthodromic after-potentials recorded in the optic radiation are of reverse polarity to those recorded in the nucleus, and the antidromic after-potentials closely resemble those obtained at the same site by orthodromic stimulation (cf. Vastola, 1957, 1959; Bishop, Burke, Davis & Hayhow, 1958). Vastola (1957) reported that the negative portion of the post-synaptic response to orthodromic stimulation was 2–10 msec in duration. Later (1959) he gave values of 10–15 msec for the initial negative phase and 100–200 msec for the total duration of the after-positivity. Vastola also described the late negative or negative-going wave.

The focally recorded after-potentials in the motoneurone pools in the spinal cord following antidromic activation (Brooks *et al.* 1950*a*) have been studied, but the comparable after-potentials following orthodromic activation do not appear to have been investigated. The orthodromic after-potentials have, however, been described, using ventral root leads

(Brooks *et al.* 1950*b*). With intracellular recording both negative and positive after-potentials may be recorded following the spike discharge (Brock *et al.* 1952; Araki, Otani & Furukawa, 1953; Coombs *et al.* 1955). These after-potentials have nearly always been produced by antidromic activation of the motoneurone but the potentials are reported to be the same following both antidromically and orthodromically evoked spikes (Brock *et al.* 1952). Very little attention has been paid to the intracellularly recorded negative after-potential, but from published records it rarely seems to have a post-spike duration of more than about 2 msec. It rapidly decays to a large positive after-potential that reaches a maximum after 10–15 msec and has a duration of about 100 msec. By contrast the spike in the Renshaw cell may be followed by a negative after-potential lasting more than 40 msec (Eccles, Fatt & Koketsu, 1954). The after-potential mechanisms in the frog spinal motoneurones appear to be different from those in the cat (Machne, Fadiga & Brookhart, 1959).

With antidromic activation the components of the motoneurone spike may be readily separated (cf. Eccles, 1957) and these are found to have distinctive after-potentials. Although no detailed studies have yet been made it is clear that, taking the axon, initial segment and cell body in sequence, there is a progressive reduction in the duration of the negative after-potential, with finally a marked augmentation of the positive after-potential. Both the axonal and 'initial segment' spikes (IS spike of Coombs *et al.* 1957; A spike of Fuortes, Frank & Becker, 1957) are followed by a prominent negative after-potential, the positive after-potential being practically absent in each case. By contrast the 'soma-dendritic' spike (SD spike of Coombs *et al.* 1957; B spike of Fuortes *et al.* 1957) has a very brief negative after-potential and a deep and prolonged positive after-potential. The excitability of these components is in keeping with the pattern of their after-potentials (Coombs *et al.* 1957; Fuortes *et al.* 1957). The interpretation of motoneurone after-potentials is complicated by the generation of synaptic potentials by activation of recurrent motor-axon collaterals (Eccles, Fatt & Koketsu, 1954). The importance of this factor in the lateral geniculate nucleus is not clear (Vastola, 1959; Bishop & Davis, 1960). It is curious that the antidromic spike in the Betz cells in the motor cortex is not usually followed by any after-polarizations (Phillips, 1959).

Relationship between after-potentials and synaptic potentials

Comparing the geniculate synaptic potentials and the corresponding spike after-potentials (Table 3; Figs. 9, 10) the principal differences are the marked reduction in the duration of the initial negative phase when a spike is discharged and the marked augmentation of the amplitude of

the succeeding positivity (Figs. 9, *b*, *c* and 10). The duration of both the positivity and the subsequent oscillations of potential remain practically unchanged. A strikingly similar change can be seen to occur in the ventral-root synaptic potentials when a motoneurone discharge takes place (Brooks *et al.* 1950*b*). This change is to be expected in view of the pattern of the after-potentials that follow the cell-body spike (see above). The after-positivity which follows the motoneurone excitatory synaptic potential has the same duration as the after-positivity which follows the spike,

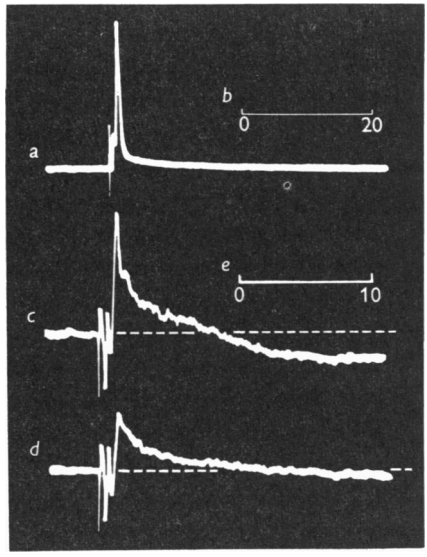


Fig. 9. *a*, Monophasic action potential and prolonged negative after-potential obtained from the optic nerve following antidromic activation of the contralateral optic tract. Time scale *b* (20 msec) refers to trace *a*. *c* and *d*, taken from Figs. 1 and 5, show the after-potential and LSD synaptic potential respectively, recorded at the same intrageniculate site following maximal shocks applied to the contralateral optic nerve. Time scale *e* (10 msec) refers to traces *c* and *d*.

i.e. 100 msec. Another factor has to be taken into consideration, namely the after-potentials of the post-synaptic axons. Assuming that the properties of the radiation axons resemble those of the optic nerve axons they will have a prolonged negative after-potential and practically no positive after-potential (Fig. 9*a*; Bishop, Jeremy & Lance, 1953; cf. Lloyd, 1951; Rudin & Eisenman, 1954; Coombs *et al.* 1957). This negativity in the axons will have the effect of reducing the amplitude and shortening the duration of the post-spike negativity recorded in the lateral geniculate nucleus.

Probably much more important than the axon, however, is the effect of potentials originating in the dendrites and the extent to which they survive the spike and thereby impose a continuing influence on the spike-

generating mechanism (Tasaki *et al.* 1954; Eyzaguirre & Kuffler, 1955*a*, *b*). The first component (IS or A) of an intercurrent antidromic spike is incapable of abolishing the motoneurone synaptic potential (Brock, Coombs & Eccles, 1953), presumably because it does not invade that region of the neurone where synaptic activity occurs. By contrast the second component (SD or B) largely removes the synaptic potential, because the spike now invades membrane involved in synaptic activity. The small residual potential that remains in the latter case has been variously interpreted as due to prolonged action of the synaptic transmitter (Curtis & Eccles, 1959) or to the fact that part of the dendritic surface does not have its synaptic potential wiped out by antidromic invasion, and that this residual synaptic potential then spreads electrotonically to the soma (Rall, 1959; cf. also Fatt, 1957*b*). Both factors may well be of importance in the one cell. The degree of antidromic invasion of the dendrites probably varies from one type of neurone to another and also in the one neurone under different conditions. Freygang (1958) has put forward the view that a large part of the soma-dendritic membrane of the geniculate neurone, though excited synaptically to produce a synaptic potential, is not invaded by a propagating spike. Even if the large dendrites are invaded it is likely that there will still be a significant spread of charge from uninvaded finer dendritic branches into the cell body and axon. In the crayfish isolated neurone (Eyzaguirre & Kuffler, 1955*a*) a persisting generator potential in the distal dendritic processes can lead to the initiation of impulses at a site along the axon at a distance of 0·5 mm from the cell body (Edwards & Ottoson, 1958). The long-continued depolarizing potential and repetitive discharge in the Renshaw cell have been attributed to the persistence of the transmitter substance as a result of the presence of diffusional barriers intimately related to the synaptic terminals on the cell (Eccles, Eccles & Fatt, 1956). Such a diffusional barrier might also lead to the accumulation of potassium released during activity and hence to the production of a negative after-potential such as occurs in the axon (cf. Narahashi & Yamasaki, 1960).

It is possible that the cell-body after-potentials of both motoneurones and geniculate neurones are, in fact, very similar, consisting of a very brief after-negativity and a deep and prolonged after-positivity without further oscillations of potential. The longer negative after-potential of the geniculate neurone and the later oscillations of potential would then be due to continuing spread of charge from the dendrites. One can regard the properties of geniculate neurones as being intermediate between those of the Renshaw cells on the one hand and of the motoneurones on the other. Part of the brief negative after-potential that follows the motoneurone SD spike may also be due to residual dendritic activity.

Relationship between after-potentials and recovery of responsiveness

The extent to which the after-potentials determine the recovery of responsiveness of geniculate synapses (Bishop & Evans, 1956; Bishop & Davis, 1960) following their discharge may now be briefly discussed. The curve of recovery of responsiveness of the r_1 spike is shown as the continuous line in Fig. 10, the ordinates representing the degree of supernormality and subnormality as a percentage of the unconditioned r_1 spike (for details see Bishop & Davis, 1960). The two early phases of supernormality have

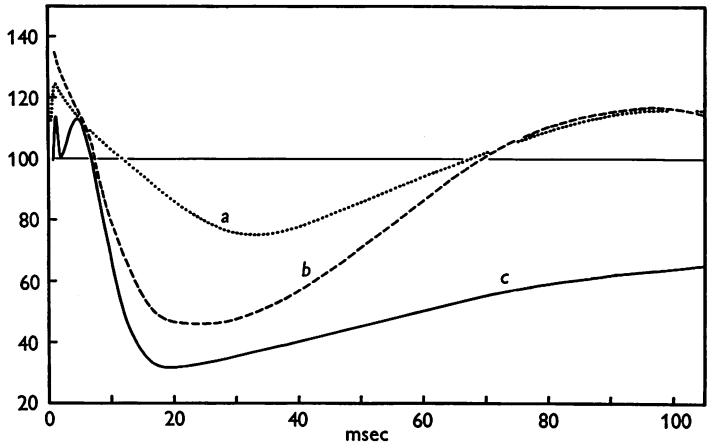


Fig. 10. *a* and *b*, mean time courses of the synaptic potentials and the after-potentials of geniculate neurones respectively; ordinates in arbitrary units. *c*, mean curve of recovery of responsiveness of geniculate synapses following a maximal conditioning shock applied to the optic nerve expressed as a percentage of the unconditioned test response (from Bishop & Davis, 1960).

a total duration of 6·7 msec. The following phase of depression is maximal at 19 msec and the recovery, which is still continuing at 100 msec, is not actually complete until about 2 sec. The geniculate after-potentials are shown as the broken line in Fig. 10, the ordinates being in arbitrary units. The negative after-potential has a duration of 7·3 msec and the positive after-potential has a trough at 22 msec. Thereafter the curve of recovery of responsiveness soon diverges widely from the after-potential pattern. The close correspondence that obtains, however, during the first 30–40 msec indicates that the after-potentials probably play a dominant part in determing the excitability of the neurone during this interval. Bishop & Davis (1960) considered the two early phases of supernormality as though they were of largely separate origin. It is more likely, however, that they represent two phases of the same process determined by the summed effect of the negative after-potentials of the cell body and axon and the residual

soma-dendritic synaptic activity. The brief intervening phase of depression or relative depression is possibly due to the brief interval of reduced or reversed current flow between cell body and axon that occurs when the radiation axons are occupied by the spike process.

The later stages of the recovery cycle are probably largely determined by presynaptic changes. An indication of the importance of presynaptic changes is the fact that, with heterosynaptic testing, the prolonged phase of depression of geniculate synapses lasts only 350 msec in contrast to the depression following homosynaptic testing which lasts about 2 sec (Bishop, Burke, Davis & Hayhow, 1958). Brooks *et al.* (1950*b*) have produced similar evidence which indicates that the late phase of the depression (i.e. after about 150 msec) in the recovery cycle of motoneurones following orthodromic activation is due to a presynaptic change.

Slow rhythms

It is becoming increasingly evident that the slow waves recorded from the cerebral cortex depend upon the rhythmic fluctuations of the membrane potential of the apical dendrites, whether the waves are spontaneously occurring or evoked (cf. particularly Clare & Bishop, 1955; Li *et al.* 1956) and the homology has been suggested of these potential waves to the post-synaptic potentials that may be recorded in the spinal cord and lateral geniculate nucleus (cf. Bremer, 1958). The results of the present study also indicate that the dendrites of the principal cells may be responsible for the slow rhythms that are recorded in the lateral geniculate nucleus. Moreover, the principal cells are almost certainly the only neurones present in the nucleus whose morphological arrangement would permit of the generation of the recorded potential fields. The geniculate rhythms continue even though the afferent input from the retina has been removed. The rhythms do not therefore depend upon the spontaneous discharge of retinal receptors. Whether the dendrites of the geniculate neurones are actually capable of continuous self-excitation or not, it is clear that, under normal circumstances, they must be conceived as being at least only just below this self-excitatory level, because they may be triggered into prolonged rhythmic activity by synaptic potentials that are well below the level necessary to initiate a propagated discharge. This occurs when the synapses on the principal geniculate neurones are blocked by large doses of LSD. If one accepts the view that LSD acts by competitive antagonism to the normal transmitter it is likely that all the excitatory synapses on the principal cells will be blocked by its action. This places a clear restriction on the mechanism used to explain the neuronal synchronization that must occur in the production of these rhythmical potentials. One explanation of neuronal synergy (cf. Bremer, 1958) involves

intricate synaptic connexions between the active cells by short axon interneurones and by recurrent collaterals of long axons. Although LSD may be without effect on the short axon interneuronal circuits in the lateral geniculate nucleus, it is unlikely that the principal neurones themselves can be included in the recurrent loops but must be regarded as non-propagating off-shoots.

Prolonged rhythmic waves, indistinguishable from those arising spontaneously, may be triggered off both orthodromically by synaptic potentials and antidromically by optic radiation volleys. The first cycle of the triggered activity, however, is different from the later slow waves. The first cycle always commences with a relatively brief negative phase followed by a positive phase which has a greater amplitude and is more prolonged than those that succeed it (Figs. 8, 10). The general conclusion suggested as a result of this study is that the geniculate neurones are capable of generating three kinds of non-propagating potentials, namely: (1) slow rhythmic oscillations due to fairly prolonged or possibly sustained self-excitation; (2) synaptic potentials due to orthodromic volleys, having an initial negative phase, lasting somewhat longer than 12 msec, followed by a prolonged after-positivity; and (3) after-potentials that arise as a result of the generation of a cell-body spike, having a very brief initial negativity (less than 7 msec), followed by a deep and prolonged after-positivity. It is not possible to assign these potentials to either dendrites or cell-body with any certainty. The possible restriction of the spike to the cell body would restrict the after-potentials to the cell body also. The other two potentials possibly involve both structures with the prolonged rhythmic activity being largely of dendritic origin. It is likely that the mechanisms responsible for all these potentials may operate concurrently so that the potentials actually recorded result from their complex interaction.

SUMMARY

1. The lateral geniculate neurones have been activated, orthodromically and antidromically respectively, by means of electrical stimulation of the optic nerve in the orbit and the optic radiation terminals in the cerebral cortex of the cat. The geniculate responses have been recorded extracellularly by means of a micro-electrode inserted down through the intact cerebral cortex.

2. Measured from the onset of post-synaptic activity, the initial negative after-potential of the orthodromic response has a mean duration of 7·3 msec; the subsequent positive after-potential has a sharp decline to a maximum at about 22 msec and returns to the base line at about 80 msec. There is a late negative peak soon after 100 msec. The subsequent oscil-

lations of potential, which have a mean frequency of 9 c/s, cannot be distinguished from the spontaneous slow rhythms.

3. The orthodromic after-potentials increase in amplitude with increasing afferent stimulation but are not significantly altered in time course. Recorded in the optic radiation the after-potentials have a similar time course but are of opposite polarity to those recorded in the nucleus. By using iron deposition to label the recording site the level at which the potentials reversed sign was found to be the upper surface of the nucleus.

4. Except for the initial after-negativity the antidromic after-potentials are the same as those evoked at the same site by orthodromic volleys.

5. Synchronous repetitive firing of geniculate neurones was frequently associated with the negative after-potential and occasionally unit repetitive firing was observed in relation to the negative peaks of the later slow rhythms. The repetitive firing ceased with the onset of the positive after-potential.

6. Synaptic potentials were recorded while the geniculate synapses were blocked by lysergic acid diethylamide and as a result of post-tetanic delayed depression, the level of block being monitored by recording from the cerebral cortex.

7. The time course of the synaptic potentials was the same in either case. Measured from the onset of post-synaptic activity the initial negativity has a duration of about 12 msec, the succeeding positivity reaches its maximum at about 25 msec, the base line is recrossed soon after 60 msec and the late negative wave has a peak at about 100 msec. The subsequent oscillations of potential are indistinguishable from the spontaneous slow rhythms which also occur in the nucleus during synaptic block.

8. In the temporary absence of spontaneous rhythmic activity long-continued oscillations of potential may be triggered off both by orthodromic and antidromic volleys.

This study was aided by grants from the National Health and Medical Research Council of Australia, from the Ophthalmic Research Institute of Australia and from the Consolidated Medical Research Funds of the University of Sydney. Sandoz Limited, Basle, Switzerland, kindly supplied the lysergic acid diethylamide. We are grateful for the skilled assistance given by the technical staff of the Physiology Department, particularly by Mr J. Stephens, Mr D. Larnach and Mr B. McGee, and we wish to express our thanks to Miss J. Kearsey for considerable secretarial assistance.

REFERENCES

ARAKI, T., OTANI, T. & FURUKAWA, T. (1953). The electrical activities of single motoneurones in toad's spinal cord, recorded with intracellular electrodes. *Jap. J. Physiol.* **3**, 254–267.

BAUMGARTEN, R. & JUNG, R. (1952). Microelectrode studies on visual cortex. *Rev. Neurol.* **87**, 151–155.

BISHOP, G. H. & O'LEARY, J. L. (1942). Factors determining the form of the potential record in the vicinity of the synapses of the dorsal nucleus of the lateral geniculate body. *J. cell. comp. Physiol.* **19**, 315–331.

BISHOP, P. O. (1953). Synaptic transmission. An analysis of the electrical activity of the lateral geniculate nucleus in the cat after optic nerve stimulation. *Proc. Roy. Soc.* B, **141**, 362–392.

BISHOP, P. O., BURKE, W. & DAVIS, R. (1958). Synapse discharged by single fibre in mammalian visual system. *Nature, Lond.*, **182**, 728–730.

BISHOP, P. O., BURKE, W., DAVIS, R. & HAYHOW, W. R. (1958). Binocular interaction in the lateral geniculate nucleus—a general review. *Trans. ophthal. Soc. Austr.* **18**, 15–35.

BISHOP, P. O., BURKE, W. & HAYHOW, W. R. (1959*a*). Repetitive stimulation of optic nerve and lateral geniculate synapses. *Exp. Neurol.* **1**, 534–555.

BISHOP, P. O., BURKE, W. & HAYHOW, W. R. (1959*b*). Lysergic acid diethylamide block of lateral geniculate synapses and relief by repetitive stimulation. *Exp. Neurol.* **1**, 556–568.

BISHOP, P. O. & DAVIS, R. (1960). The recovery of responsiveness of the sensory synapses in the lateral geniculate nucleus. *J. Physiol.* **150**, 214–238.

BISHOP, P. O. & EVANS, W. A. (1956). The refractory period of the sensory synapses of the lateral geniculate nucleus. *J. Physiol.* **134**, 538–557.

BISHOP, P. O., FIELD, G., HENNESSY, B. L. & SMITH, J. R. (1958). Action of D-lysergic acid diethylamide on lateral geniculate synapses. *J. Neurophysiol.* **21**, 529–549.

BISHOP, P. O., JEREMY, D. & LANCE, J. W. (1953). The optic nerve. Properties of a central tract. *J. Physiol.* **121**, 415–432.

BISHOP, P. O., JEREMY, D. & MCLEOD, J. G. (1953). The phenomenon of repetitive firing in lateral geniculate nucleus of cat. *J. Neurophysiol.* **16**, 437–447.

BISHOP, P. O. & MCLEOD, J. G. (1954). Nature of potentials associated with synaptic transmission in lateral geniculate of cat. *J. Neurophysiol.* **17**, 387–414.

BREMER, F. (1958). Cerebral and cerebellar potentials. *Physiol. Rev.* **38**, 357–388.

BROCK, L. G., COOMBS, J. S. & ECCLES, J. C. (1952). The recording of potentials from motoneurones with an intracellular electrode. *J. Physiol.* **117**, 431–460.

BROCK, L. G., COOMBS, J. S. & ECCLES, J. C. (1953). Intracellular recording from antidromically activated motoneurones. *J. Physiol.* **122**, 429–461.

BROOKHART, J. M. & FADIGA, E. (1960). Potential fields initiated during monosynaptic activation of frog motoneurones. *J. Physiol.* **150**, 633–655.

BROOKS, C. MCC., DOWNMAN, C. B. B. & ECCLES, J. C. (1950*a*). After-potentials and excitability of spinal motoneurones following antidromic activation. *J. Neurophysiol.* **13**, 9–38.

BROOKS, C. MCC., DOWNMAN, C. B. B. & ECCLES, J. C. (1950*b*). After-potentials and excitability of spinal motoneurones following orthodromic activation. *J. Neurophysiol.* **13**, 157–176.

BROOKS, C. MCC. & ECCLES, J. C. (1947). Electrical investigation of the monosynaptic pathway through the spinal cord. *J. Neurophysiol.* **10**, 251–273.

CLARE, M. H. & BISHOP, G. H. (1955). Properties of dendrites; Apical dendrites of cat cortex. *Electroenceph. clin. Neurophysiol.* **7**, 85–98.

COOMBS, J. S., CURTIS, D. R. & ECCLES, J. C. (1957). The interpretation of spike potentials of motoneurones. *J. Physiol.* **139**, 198–231.

COOMBS, J. S., ECCLES, J. C. & FATT, P. (1955). Excitatory synaptic action in motoneurones. *J. Physiol.* **130**, 374–395.

CURTIS, D. R. & ECCLES, J. C. (1959). The time courses of excitatory and inhibitory synaptic actions. *J. Physiol.* **145**, 529–546.

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

ECCLES, J. C. (1957). *The Physiology of Nerve Cells.* Baltimore: The Johns Hopkins Press.

ECCLES, J. C., ECCLES, R. M. & FATT, P. (1956). Pharmacological investigations on a central synapse operated by acetylcholine. *J. Physiol.* **131**, 154–169.

ECCLES, J. C., FATT, P. & KOKETSU, K. (1954). Cholinergic and inhibitory synapses in a pathway from motor-axon collaterals to motoneurones. *J. Physiol.* **126**, 524–562.

ECCLES, J. C., FATT, P. LANDGREN, S. & WINSBURY, G. J. (1954). Spinal cord potentials generated by volley in large muscle afferents. *J. Physiol.* **125**, 590–606.

EDWARDS, C. & OTTOSON, D. (1958). The site of impulse initiation in a nerve cell of a crustacean stretch receptor. *J. Physiol.* **143**, 138–148.

ERLANGER, J. & GASSER, H. S. (1937). *Electrical Signs of Nervous Activity*, p. 29. Philadelphia: University of Pennsylvania Press.

EVARTS, E. V. & HUGHES, J. R. (1957*a*). Relation of post-tetanic potentiation to subnormality of lateral geniculate potentials. *Amer. J. Physiol.* **188**, 238–244.

EVARTS, E. V. & HUGHES, J. R. (1957*b*). Effects of prolonged optic nerve tetanization on lateral geniculate potentials. *Amer. J. Physiol.* **188**, 245–248.

EVARTS, E. V., LANDAU, W., FREYGANG, W., JR. & MARSHALL, W. H. (1955). Some effects of lysergic acid diethylamide and bufotenine on electrical activity in the cat's visual system. *Amer. J. Physiol.* **182**, 594–598.

EYZAGUIRRE, C. & KUFFLER, S. W. (1955*a*). Processes of excitation in the dendrites and in the soma of single isolated sensory nerve cells of the lobster and crayfish. *J. gen. Physiol.* **39**, 87–119.

EYZAGUIRRE, C. & KUFFLER, S. W. (1955*b*). Further study of soma, dendrite, and axon excitation in single neurons. *J. gen. Physiol.* **39**, 121–153.

FATT, P. (1957*a*). Electrical potentials occurring around a neurone during its antidromic activation. *J. Neurophysiol.* **20**, 27–60.

FATT, P. (1957*b*). Sequence of events in synaptic activation of a motoneurone. *J. Neurophysiol.* **20**, 61–80.

FRANK, K. & FUORTES, M. G. F. (1955). Potentials recorded from the spinal cord with microelectrodes. *J. Physiol.* **130**, 625–654.

FRANK, K. & FUORTES, M. G. F. (1956). Stimulation of spinal motoneurones with intracellular electrodes. *J. Physiol.* **134**, 451–470.

FREYGANG, W. H., Jr. (1958). An analysis of extracellular potentials from single neurons in the lateral geniculate nucleus of the cat. *J. gen. Physiol.* **41**, 543–564.

FUORTES, M. G. F., FRANK, K. & BECKER, M. C. (1957). Steps in the production of motoneuron spikes. *J. gen. Physiol.* **40**, 735–752.

GRANIT, R., PASCOE, J. E. & STEG, G. (1957). The behaviour of tonic α and β motoneurones during stimulation of recurrent collaterals. *J. Physiol.* **138**, 381–400.

HUGHES, J. R., EVARTS, E. V. & MARSHALL, W. H. (1956). Post-tetanic potentiation in the visual system of cats. *Amer. J. Physiol.* **186**, 483–487.

LI, C-L., CULLEN, C. & JASPER, H. H. (1956). Laminar microelectrode analysis of cortical unspecific recruiting responses and spontaneous rhythms. *J. Neurophysiol.* **19**, 131–143.

LLOYD, D. P. (1946). Facilitation and inhibition of spinal motoneurons. *J. Neurophysiol.* **9**, 421–438.

LLOYD, D. P. C. (1951). After-currents, after-potentials, excitability, and ventral root electrotonus in spinal motoneurons. *J. gen. Physiol.* **35**, 289–321.

LLOYD, D. P. C. & MCINTYRE, A. K. (1949). On the origins of dorsal root potentials. *J. gen. Physiol.* **32**, 409–443.

MACHNE, X., FADIGA, E. & BROOKHART, J. M. (1959). Antidromic and synaptic activation of frog motor neurons. *J. Neurophysiol.* **22**, 483–503.

NARAHASHI, T. & YAMASAKI, T. (1960). Mechanism of the after-potential production in the giant axons of the cockroach. *J. Physiol.* **151**, 75–88.

PHILLIPS, C. G. (1959). Actions of antidromic pyramidal volleys on single Betz cells in the cat. *Quart. J. exp. Physiol.* **44**, 1–25.

RALL, W. (1959). Dendritic current distribution and whole neuron properties. Research Report NM 01 05 00.01.02, Vol. 17, 479–526, Naval Medical Research Institute, Bethesda, Md.

RENSHAW, B. (1941). Influence of discharge of motoneurons upon excitation of neighbouring motoneurons. *J. Neurophysiol.* **4**, 167–183.

RENSHAW, B. (1946). Observations on interaction of nerve impulses in the gray matter and on the nature of central inhibition. *Amer. J. Physiol.* **146**, 443–448.

RUDIN, D. O. & EISENMAN, G. (1954). The action potential of spinal axons in vitro. *J. gen. Physiol.* **37**, 505–538.

TABOADA, R. P. (1927). Note sur la structure du corps genouillé externe. *Trab. Lab. Invest. biol. Univ. Madr.* **25**, 319–329.

TASAKI, I., POLLEY, E. H. & ORREGO, F. (1954). Action potentials from individual elements in cat geniculate and striate cortex. *J. Neurophysiol.* **17**, 454–474.

VASTOLA, E. F. (1957). Antidromic action potentials in lateral geniculate body. *J. Neurophysiol.* **20**, 167–185.

VASTOLA, E. F. (1959). After-positivity in lateral geniculate body. *J. Neurophysiol.* **22**, 258–272.