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THE OPTIC NERVE. PROPERTIES OF A CENTRAL TRACT*

P. O. BISHOP, D. JEREMY AND J. W. LANCE†

From the Brain Research Unit, Departments of Surgery, Anatomy and Physiology, University of Sydney

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In attempting to reconstruct the compound spike potential of the cat's optic nerve from the fibre-calibre spectrum (Bishop, Glenn and Gye, to be published) information was required concerning the properties of the fibres. For this purpose a technique of antidromic activation of the nerve *in situ* was developed. This preparation has provided a unique opportunity for determining the properties of the fibres in a tract in the central nervous system, for developmentally and structurally the optic nerve must be considered as a central tract (Cone & Macmillan, 1932). The systematic study of the properties of mammalian peripheral nerves by Gasser & Grundfest (1936, 1939), Grundfest & Gasser (1938) and Grundfest (1939) demonstrated that the fibres fall into three major groups (A, B and autonomic C) with the possibility of a fourth (afferent C). Gasser (1950) has recently confirmed the existence of a distinct afferent mammalian C group. By contrast, very little information of a similar nature has as yet been obtained about the fibres in the central pathways. The scattered data available are almost entirely restricted to aspects of conduction velocity (e.g. G. H. Bishop, 1933; Grundfest & Campbell, 1942). It is generally assumed that central fibres will have the same properties as those in the periphery, but hitherto it has not been possible to identify any of the major peripheral groups in the central nervous system. As a result of the investigation to be reported here it has been demonstrated that all the fibres in the cat's optic nerve belong to the A group. A similar study (Bishop, Jeremy & Lance, 1953) on the pyramidal tract indicates that all the myelinated fibres in that tract probably also belong to the A group.

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† National Health and Medical Research Council Fellow.

METHODS

Adult cats were used in all the experiments, and were anaesthetized with intraperitoneal Dial (Ciba) (0·6 ml./kg) with occasionally additional small amounts (0·5 ml.) of pentobarbitone sodium (Nembutal) intravenously to control the level of anaesthesia. Throughout the course of the experiment the cat's body temperature, read with a rectal thermometer, was controlled within normal limits (38–39° C) with the aid of an electric heating blanket.

The preparation of the optic nerve for stimulation or recording has been described elsewhere (Bishop, 1953; Bishop, Jeremy & McLeod, 1953). The eyeball contents were removed and the bulb wall collapsed and tied around a small silver rod with expanded end which served as a lead from the crushed end. The major portion of the bulb wall distal to the tie was now cut away. Up to 10 mm of optic nerve enclosed in its dural sheath may be suspended clear of orbital tissue by means of a thread tied to the silver rod. A second silver electrode in the form of an inverted 'U' was placed astride the nerve at a distance of 8 mm from the eyeball. For antidromic monophasic recording the nerve was crushed at its exit from the eyeball, the crushed portion being about 2 mm wide; the active electrode is then 6 mm from the proximal portion of the crush. The problem of obtaining a satisfactory monophasic lead has been discussed recently by Gasser (1950). The optic nerve does not lend itself to the use of the cocaine block, and the simple procedure of crushing the nerve, though by no means reliable, was adequate for the purposes of the present investigation. In most cases a monophasic record was obtained but some diphasicity usually became apparent later, so that recrushing was necessary to maintain the monophasic lead.

The cortex of the opposite cerebral hemisphere above the optic tract was exposed. The head was firmly clamped in a stereotaxic instrument and the electrodes directed through the intact cortex into the optic tract by means of a micro-manipulator using Horsley-Clarke planes of reference. The most suitable Horsley-Clarke vertical was found to be centred about 7·5 mm lateral and 12·5 mm anterior. When introduced into the optic tract along this vertical the stimulating electrodes gave an antidromic conduction distance to the electrode astride the nerve of about 22·5 mm. This conduction distance only varied by $\pm 1{\cdot}0$ mm in the majority of the experiments. At the position selected the optic tract is a compact bundle running at right angles to the Horsley-Clarke vertical. More posteriorly the tract begins to curve upwards in a broad sweep round the cerebral peduncles, becoming almost vertical and very much broader as the lateral geniculate body is approached. At the end of the experiment a needle was inserted and left in the brain to mark the stimulation site. The animal was perfused with normal saline and formalin-saline and the brain and optic nerves removed intact. By subsequently dissecting away the temporal lobe to expose the needle transfixing the optic tract the apparent conduction distance was measured by placing a fine cotton thread along the nerve and tract from eyeball to needle.

The bipolar stimulating electrodes were prepared by electrolytically pointing steel beading needles (Bishop & Collin, 1950; Grundfest, Sengstaken, Oettinger & Gurry, 1950). They were mounted about 1 mm apart and insulated to the tip with Bakelite varnish. The cathode of the bipole was directed along the appropriate Horsley-Clarke vertical with the anode orientated so that it would enter the tract on the geniculate side of the cathode. The stimulating pulses were about 50 μsec in duration and up to 100 V in amplitude, delivered to the preparation through a shielded transformer. With the apparatus and techniques available it was not found possible to stimulate the optic tract maximally. Increasing the amplitude and duration of the stimulating pulse beyond that used in these experiments is probably undesirable because of the difficulty of controlling current spread and the consequent uncertainty regarding site of stimulation. For orthodromic recording a steel microelectrode with a tip diameter less than 10 μ was used with stimulation applied via the electrodes on the optic nerve.

RESULTS

The compound spike potential of the optic nerve

The determination of the compound spike potential of the optic nerve as a whole is beset with difficulties which have not, as yet, been satisfactorily

surmounted. Insufficient optic nerve can be mobilized to provide an adequate conduction distance. It will be seen below that the method of antidromic stimulation of the tract used in this investigation does not provide a satisfactory alternative. The optic nerve distributes fibres to both optic tracts, and although the form of the compound spike potential of each component separately, crossed and uncrossed, seems fairly clear, difficulty is experienced in attempting their combination because of lack of precise information concerning the separate conduction distances. The fibre-calibre spectrum of the cat's optic nerve has been studied in this laboratory. A preliminary statement of this work (Bishop, 1953) will be followed by a detailed account later (Bishop & Walshe, to be published). The nerve contains about 120,000 fibres, all of which are myelinated and range in axonal diameter from 1 to 8·5 μ. The fibre-size spectrum shows evidence of grouping about axonal diameters of 1–1·5 μ and 4–4·5 μ, although the modes are rather ill defined. About 80% of the fibres have axon diameters of 3 μ or less.

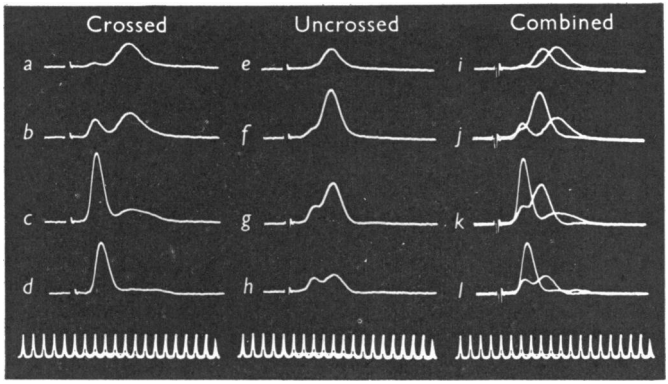

Fig. 1. The antidromic compound spike potentials from the optic nerve of the opposite and the same side using a common stimulation site in the tract for each pair of records *a* and *e*, *b* and *f*, etc. In the third column these have been superimposed. Conduction distance (crossed) 19·5 mm. Time intervals 0·2 msec.

If the fibres in the one tract are stimulated, records from the optic nerve of either side reveal the presence of two distinct fibre groups (Fig. 1). Such a clear separation of peaks, especially in the crossed fibres, would not be expected from a consideration of the fibre spectrum of the optic nerve. The presence of two fibre groups is clearly suggested, however, by an inspection of a suitably stained cross-section of the optic tract at the site of stimulation (Fig. 2A, B). The fibres show a well-marked segregation according to size with only small fibres present in the upper part of the tract and the large fibres concentrated into the lower portion. Although the upper part contains small fibres only, there are, however, small fibres scattered amongst the large. This

segregation according to size may easily be demonstrated by inserting a stimulating bipole from above downwards through the optic tract at intervals of 250 μ recording the nerve response at each step. Fig. 2D shows the result of a typical experiment recording from the contralateral optic nerve. It will be seen that record (*a*) contains only a small fibre spike with the large fibres becoming apparent after the bipole has been moved on a further 0·5 mm or so.

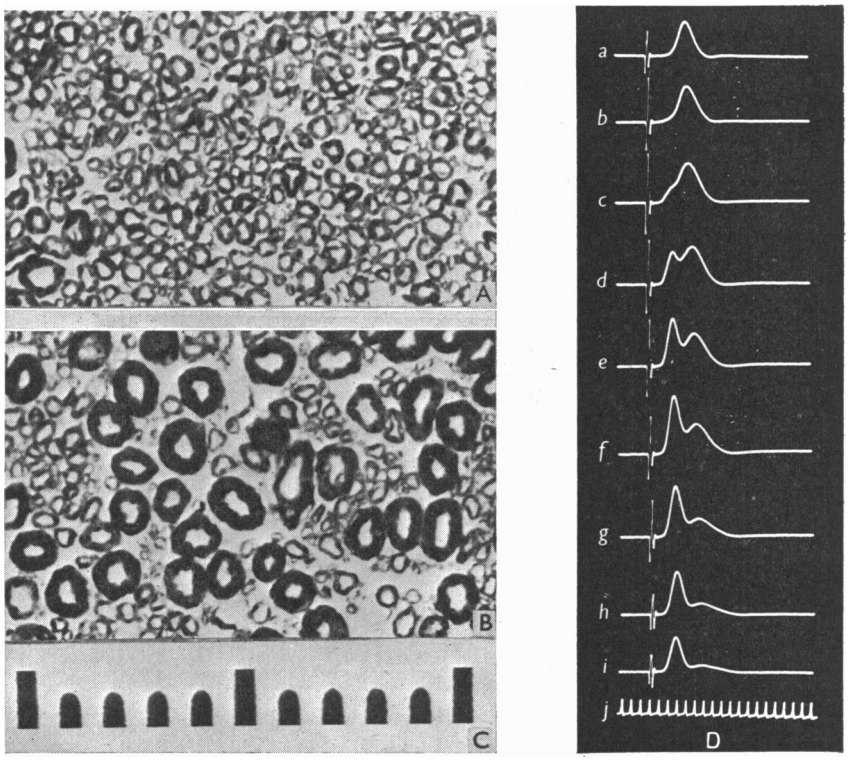

Fig. 2. A and B, fields from cat's optic tract stained with osmium tetroxide. A, field from the upper part of the tract containing small fibres only. B, field from the lower part of the tract containing both large and small fibres. C, scale, 10 and 50 μ. D, antidromic records from contralateral optic nerve with the stimulating bipole descending through the tract at intervals of 0·25 mm. Time intervals 0·2 msec. Conduction distance 19·5 mm.

Though the small fibres make a recognizable contribution to the compound potential throughout the whole extent of the bipole traverse it becomes relatively small in the lower records. A similar segregation of fibres is apparent if records are taken from the homolateral nerve (Fig. 1*e–h*), the small and large fibres of each side being grouped together in the tract. In the case of the homolateral response the amplitude of the small fibres at each stimulation site approximates that given by the small fibres in the other nerve, but the

amplitude of the large is very much smaller. Thus the homolateral tract fibres are predominantly small. The response from the homolateral nerve may occasionally reveal a distinct peak due to large fibres (e.g. Fig. 1*h*), but it is more usual for them to produce little more than a hump on the foot of the rising limb of the small fibre spike (e.g. Fig. 1*f*).

A rather unexpected finding is revealed when the time relations of the peaks of the spike potentials are compared by superimposing the responses from a common stimulation site in the tract and with recording electrodes placed at equal distances from the eyeball in each case (Fig. 1*i–l*). Records were taken at intervals of 250 μ as the stimulating bipole was inserted down through the optic tract. Those selected for Fig. 1 were at intervals of *a* to *b* 0·25 mm, *b* to *c* 0·5 mm, and *c* to *d* 0·5 mm. The homolateral small fibre spike falls in the trough between the two contralateral spikes (Fig. 1*j*) with the homolateral large fibre peak very slightly earlier than its fellow of the opposite side. Although the homolateral small fibres have been consistently faster than the contralateral small group the low amplitude of the homolateral large fibre potential makes a similar relationship for the large fibres difficult to determine. The homolateral large fibres have never been slower than the contralateral. To what extent this finding reflects a difference in the fibre-size spectra of the two components in the tract it is difficult to say because of the uncertainty regarding the true conduction distances in each case. The chiasm in the cat is 'H'-shaped rather than 'X'-shaped with the cross-member of the 'H' measuring about 2·5 mm. Presumably this would make the conduction distance of the crossed fibres longer than the uncrossed by the same amount. Such a difference in conduction distance is, however, insufficient to account for the observed difference in peak times. If the fibre spectra of the two pathways are different it would explain why the groupings in the spectrum of the nerve are ill-defined. These alternatives are at present under investigation.

Conduction velocities

Using a fine cotton thread for measuring conduction distances (see Methods) the conduction velocity of the peaks of the various fibre spikes has been determined. That of the contralateral large fibres ranged from 30 to 40 m/sec (mean 34) and the homolateral large fibres appear to be faster in every instance by about 0·5–1·0 m/sec. The velocity range of the contralateral small fibres was 15–20 m/sec (mean 18) and for the homolateral small, 17–23 m/sec (mean 21). Thus, as a group, the homolateral small fibres are faster than the contralateral small by about 3 m/sec. The velocity of the foot of the compound spike potential averaged from many records in five separate experiments was 70 m/sec. Taking 8·5 μ as the axonal diameter of the largest fibre making a significant contribution to the fibre-spectrum, and therefore to the action potential this gives a factor of 8·2 for converting axon diameter to conduction

velocity. This agrees well with the figure of 8·7 given by Gasser & Grundfest (1939) for peripheral A fibres. In the fibre spectrum of one optic nerve there were no fibres having axonal diameters greater than 8·0 μ giving, in this case, a factor of 8·7 for a velocity of 70 m/sec. These estimations have not taken account of shock-response time and the slowing of conduction in the small portion of exposed optic nerve so that the figures given for conduction velocities are probably somewhat too low. Spread of stimulus was probably not a significant factor in these experiments.

Recovery of excitability

In order to test the fluctuations of excitability of the fibres following a single impulse two stimuli were delivered to the tract from the one bipole at varying intervals between conditioning and test shock. Because it is possible at any position of the bipole to stimulate only a fraction of the total number of fibres in the optic tract, it is essential that the second or testing shock be delivered via the same electrodes as the first, otherwise the testing shock may activate fibres which were not previously stimulated by the conditioning shock. For this reason also the second shock must not be of such a strength that on its own it would give rise to a spike of larger amplitude than that due to the conditioning shock. The stimulus situation in a volume of conducting tissue is complex. Thus, for any one stimulus strength above the threshold for a perceptible response, the fibres in the immediate vicinity of the cathode of the bipole can be regarded as being subjected to a stimulus which is well above threshold. Gradually merging with, and surrounding this zone, is another in which the fibres are receiving a just-threshold stimulus, and farther out again the stimulus will be subthreshold. The situation is further complicated by the fact that the fibres do not possess uniform excitabilities. For any one stimulus strength the zone of effective stimulation will be greater for the larger than for the smaller fibres.

Recovery of excitability can be measured by the variation in amplitude of the response to a stimulus of constant strength (Graham & Lorente de Nó, 1938) because, over the limited range considered, the amplitude of the unconditioned response varies linearly with stimulus strength (Fig. 3). Fluctuations in excitability were, however, usually expressed as a percentage change in amplitude of the testing response when preceded by a conditioning response. About 4 sec before each combination of conditioning and test shock a control 'test' response was recorded on its own. In this way allowance was made for any variation which might occur in the amplitude of the control 'test' response. During the whole experiment the tract was stimulated as infrequently as possible, the time-base recurrence being once every 4 sec or longer with consecutive sweeps being recorded.

Absolutely refractory period. The absolutely refractory period was deter-

mined in the usual way by progressively decreasing the interval between conditioning and test shock (Fig. 4 A and B). The second spike is seen to suffer a progressive reduction in velocity and amplitude, the latter being caused partly by a reduction in the number of fibres activated and partly by a reduction in the amplitude of the individual spikes of the axons which are still responding.

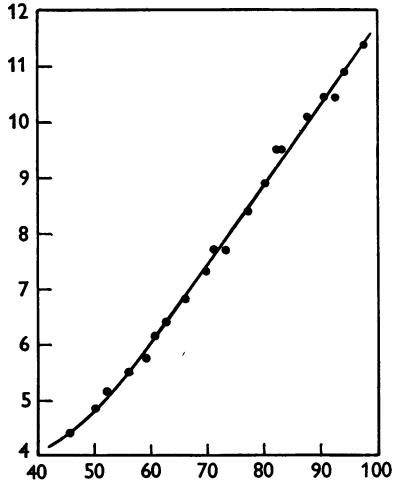

Fig. 3. Variation in height of unconditioned response with strength of stimulus. Ordinate, amplitude of response in arbitrary units. Abscissa, amplitude of stimulating pulse in volts (duration 50 μsec).

The fibres situated on the periphery of the stimulated zone will fall out of the test response first as the interval is decreased; there then follows a progressive falling out from the periphery inwards towards the cathode of the bipole. The last to fall out are those which have been subjected to a stimulus which is well above threshold. Thus the conditioning shock–test shock interval at which a test response just ceases to be perceptible may be taken as the absolutely refractory period. In one case (Fig. 4 B) the absolutely refractory period was measured by orthodromic stimulation of the optic nerve, recording the response by means of a microelectrode inserted into the tract. This method gave figures comparable to those obtained by the antidromic method, but it is less satisfactory because it is difficult to maintain the optic nerve at body temperature. With the rectal temperature maintained at a normal level it was considered that, using the antidromic method, the temperature at the point of stimulation in the optic tract would approximate to normal.

The above methods indicated that the absolutely refractory period of the large fibres ranged from 0·35 to 0·6 msec (mean 0·47 msec) and of the small fibres 0·4 to 0·7 msec (mean 0·53 msec). In nearly all cases the period for the

small fibres was slightly longer than that for the large. Observations taken at the end of an experiment usually did not differ significantly from those taken at the beginning, although in some cases a slight lengthening of the absolutely refractory period was noted presumably due to previous activity and general deterioration of the preparation. In the later preparations the figures tended to be lower than those obtained in the earlier ones so that the mean figures given above are probably too large.

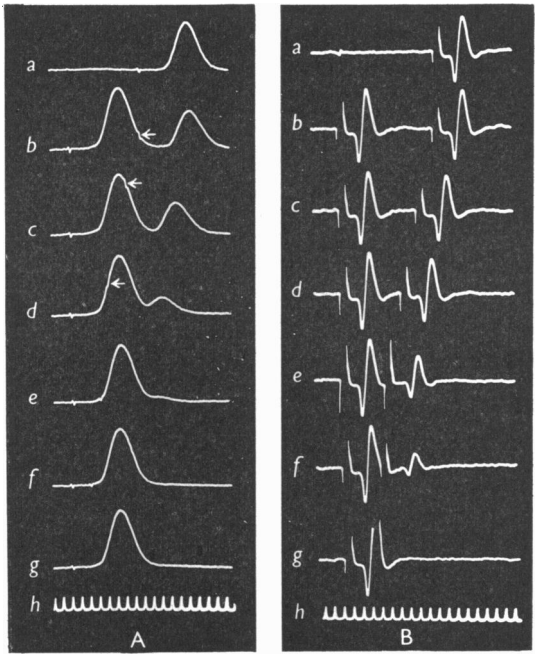

Fig. 4. Absolutely refractory period. A, antidromic response—small fibres. B, orthodromic response—large fibres. Arrows (A: *b*, *c*, *d*) indicate shock artifact. A: *a* and B: *a*, unconditioned test responses alone. A: *g*, conditioning response alone. A: *f* and B: *g*, failure of test responses. Time intervals 0·2 msec.

Relatively refractory period. The relatively refractory period may be estimated by using a test response which alone has the same amplitude as that of the conditioning response, and by measuring the interval between conditioning and test shock at which the test response first suffers a reduction in amplitude. As pointed out above, the fibres first to leave the test response are those which have been provided by a just-threshold stimulus by the conditioning shock as well as by the test shock on its own. This method indicates that the relatively refractory period extends up to about 6 msec after the conditioning shock. Fig. 5 shows the form of the curve of recovery of responsiveness, the response

returning to normal at 5·5 msec. When the testing response on its own is smaller than the conditioning response supernormality becomes apparent after 2–3 msec. This indicates that the relatively refractory period of individual fibres in the group varies from less than 2 msec to as much as 6 msec.

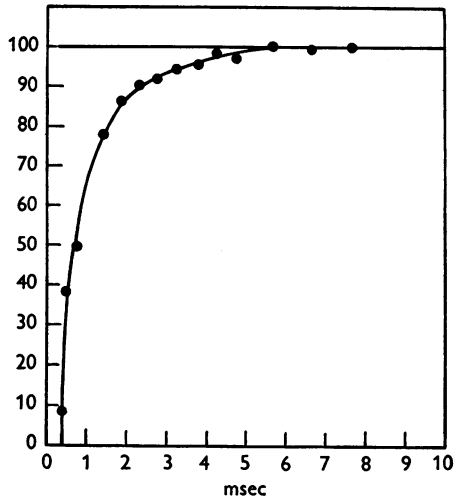

Fig. 5.

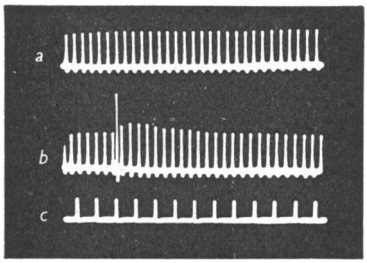

Fig. 6.

Fig. 5. Recovery of responsiveness—small fibres. Ordinate, amplitude of test response as percentage of unconditioned amplitude. Abscissa, shock interval in milliseconds.

Fig. 6. Supernormality—small fibres. *a*, unconditioned tetanus at 230 stimuli per second. *b*, effect of interpolation of conditioning response. *c*, time intervals, 10 msec.

Supernormal period. When the test shock is smaller than the conditioning shock supernormality cannot lead to an amplitude of test response greater than that of the conditioning response. In order to avoid this limiting condition in all the investigations into the supernormal and subnormal phases of recovery care was taken to use a testing shock of such a strength that the supernormal amplitude never reached that of the conditioning response amplitude. A phase of supernormality may be clearly demonstrated (Fig. 6) by interpolating a conditioning shock among a train of smaller test shocks (Gasser & Grundfest, 1936). Supernormality was consistently detected following a single conditioning shock (Fig. 7). The increase in amplitude of the test response was usually between 10 and 20% of the unconditioned response, gradually subsiding to normality at about 40 msec. Three typical curves for the later phases of recovery of excitability, both for the large and small fibres, are shown in Fig. 7. The end of the supernormal period varied from 20 to 45 msec (mean 33 msec) for the large fibres and 30–45 msec (mean 40 msec) for the small fibres, the curve becoming supernormal in both cases at 2–3 msec.

The peak of the supernormality of the large fibres (4–6 msec) usually occurred earlier, and was of lower amplitude than that of the smaller fibres (6–7 msec).

Subnormal period. After the long supernormal period the subnormal phase is hard to detect, being usually within the range of variation of the individual unconditioned responses. Estimates of its duration must necessarily be somewhat uncertain, but the end of subnormality ranged from 65 to 90 msec after

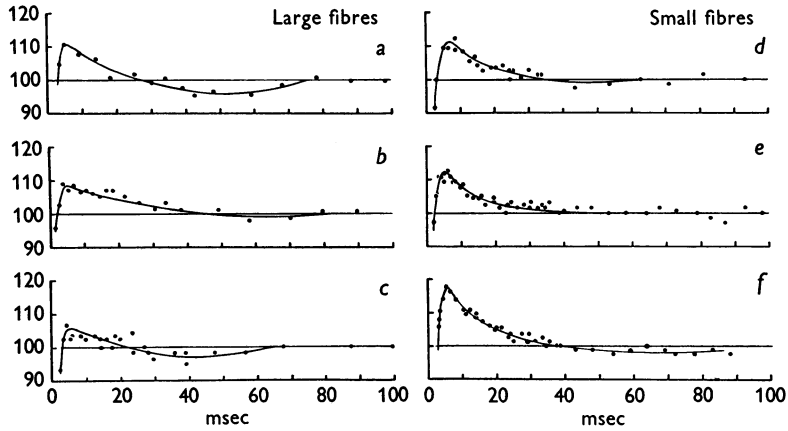

Fig. 7. Variations in excitability following single conditioning impulse. Typical cycles showing variation in amplitude of test response of large fibres (*a–c*) and small fibres (*d–f*). Ordinate, amplitude of test response as percentage of unconditioned amplitude. Abscissa, shock interval in milliseconds.

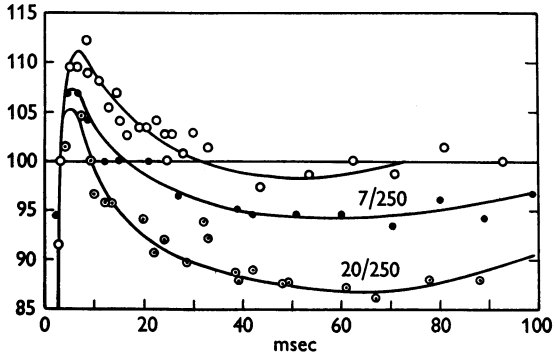

Fig. 8. Recovery cycle of optic nerve fibres following tetanic stimulation—small fibres. The uppermost curve, following a single conditioning response, the lower ones following 7 and 20 stimuli respectively at 250 stimuli per second. Ordinate, amplitude of test response as percentage of unconditioned amplitude. Abscissa, shock interval in milliseconds.

the stimulus artifact. Subnormality is well demonstrated by conditioning the fibres with a train of impulses (Fig. 8). Following tetanic stimulation the curve of recovery is characterized by a reduction in absolute supernormality and by increased subnormality. The tendency to supernormality in the early part of

the recovery cycle always remained with the stimulation frequencies used, and even with bursts of twenty stimuli at 250/sec absolute supernormality of the small fibres still prevailed (Fig. 8). In the case of the large fibres with a burst of 20/250 the tendency to supernormality is still marked, but the response remains subnormal throughout the recovery cycle. Such a conditioning train of stimuli causes the later part of the recovery cycle of the small fibres to display a deeply augmented phase of subnormality (Fig. 8), but in the case of the large fibres this augmentation of subnormality, although present, is not nearly so marked.

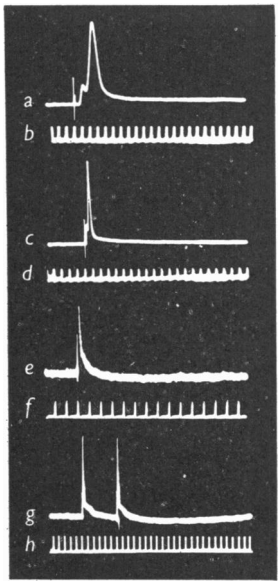

Fig. 9. Negative and positive after-potentials—small fibres. Time intervals: *b*, 0·5 msec; *d*, 2·0 msec; *f*, 5 msec; *h*, 5 msec.

After-potentials

Because the small fibres are always stimulated to some extent the after-potentials associated with the large fibres could not be satisfactorily recorded. In the case of the small fibres, however, a monophasic action potential may be obtained which is always characterized by a well-marked negative after-potential (Fig. 9*a*, *c*).

The positive after-potential which follows is, however, of small amplitude and requires high amplification to be demonstrated (Fig. 9*e*). The positive after-potential is more easily seen when two or more suitably timed impulses are superimposed (Fig. 9*g*). The amplitude of the negative after-potential measured at the point where the slope in the decline of the action potential begins sharply to decrease was about 10% of the spike height after some 2 cm

of conduction. The actual percentage would be less because temporal dispersion reduces the spike height without greatly altering the amplitude of the after-potentials. The duration of the negative after-potential may be measured in suitable cases and is of the same order (30–40 msec) as the duration of the supernormal period. The duration of the negative after-potential shown in Fig. 9*c* is 32 msec. No rising phase was ever observed nor did rhythmic after-potentials occur.

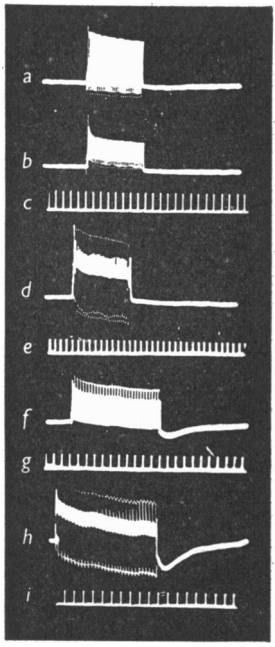

Fig. 10.

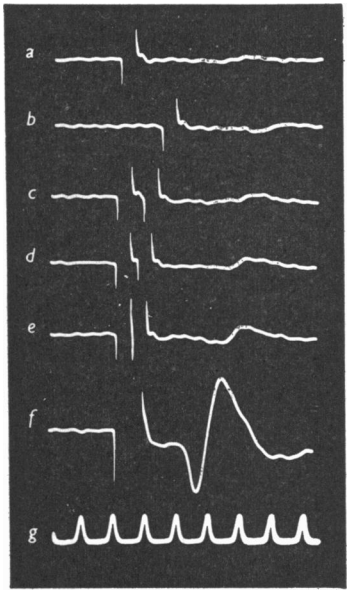

Fig. 11.

Fig. 10. Modification of after-potentials by tetanic stimulation—small fibres. *a*, 480 stimuli per sec; *b*; 1000 stimuli per sec, *c*, time intervals 10 msec; *d*, 1100 stimuli per sec; *e*, time intervals 2·0 msec; *f*, 800 stimuli per sec; *g*, time intervals 50 msec; *h*, 1100 stimuli per sec; *i*, time intervals 50 msec.

Fig. 11. Latent addition, *a*, *b*, just subthreshold orthodromic stimuli; *d*, *e*, *f*, latent addition; *g*, time intervals 0·2 msec.

Tetanic stimulation. Modification of the after-potential sequence by tetanic stimulation of high frequency resulted in summation of the negative after-potentials during the tetanus with an immediate overswing to positivity at the cessation of stimulation (Fig. 10). 'Staircasing' of the negative after-potentials (Fig. 10*d*) and augmentation of the afterpositivity (Fig. 10*h*) becomes marked only after a relatively long tetanus at fairly high frequency. Reversion to negativity or rhythmicity of after-potentials was not observed. That the negative after-potential process remained during the augmented positivity is

evident from an examination of the curve of recovery of excitability following bursts of stimuli (Fig. 8).

Latent addition

The period of latent addition for the large fibres was tested by using two just subthreshold shocks delivered to the optic nerve and observing the optic tract response. It was found to be of the same order (0·13 msec) as that for A and B peripheral fibres (0·2 msec; Gasser & Grundfest, 1936; Grundfest, 1939). A typical experiment for its determination is illustrated in Fig. 11.

The properties of the two groups of fibres may now be summarized, and this is done in Table 1.

TABLE 1. Properties of two groups of optic nerve fibres

Function	Units	Large fibres	Small fibres
Axonal diameter (all myelinated)	μ	3–8·5 (4·5)	1–2 (1·5)
Peak conduction velocity (crossed fibres)	m/sec	30–40 (34)	15–20 (18)
Peak conduction velocity (uncrossed fibres)	m/sec	—	17–23 (21)
Absolutely refractory period	msec	0·35–0·6 (0·47)	0·4–0·7 (0·53)
Relatively refractory period	msec	<2–4	<2–6
Duration of supernormal period	msec	18–43 (31)	28–43 (38)
Negative after-potential			
Amount	% of spike	—	<10%
Duration	msec	—	30–40
Period of latent addition	msec	0·13	—

DISCUSSION

Although it has long been recognized that the fibres in the optic nerve are of graded size, data such as the fibre-calibre spectrum of the nerve as a whole and of its crossed and uncrossed components, the number of fibres of each size that cross in the chiasm and the spatial distribution of the fibre groups in the various parts of the pathway are almost entirely lacking from the literature. The only systematic account of the range and frequency of fibre sizes appears to be Chacko's (1948) analysis of the human optic nerve. Many investigators, including particularly Arey and his collaborators, have made total fibre counts of the optic nerves of various animals. The finding of Bruesch & Arey (1942), that there were no unmyelinated fibres in the cat's optic nerve, is confirmed in the present investigation by the absence of any potential wave which could be attributed to such slowly conducting fibres. Furthermore, counts in this laboratory have shown that the number of ganglion cells in the retina closely approximates to the number of fibres in the optic nerve that may be stained with osmium tetroxide. The early study by G. H. Bishop (1933) showed that, on the basis of conduction velocity and threshold for electrical stimulation, the frog optic nerve contains three main groups of fibres. His experiments on the rabbit optic nerve were less satisfactory, but antidromic activation of the crossed pathways revealed two groups similar to the first two of the frog and probably a third whose identification was less certain. Although many references to possible fibre groups in the optic nerve have been made since

then, there has been no systematic attempt to confirm or extend these original observations. As far as the cat is concerned rather divergent views have been held by different investigators and by the same investigator at different times (e.g. G. H. Bishop & O'Leary, 1938, 1940, 1942; Chang, 1952). Chang's recent support for a trichromatic theory of colour vision (Chang, 1951) is based on a triple conducting pathway from the retina.

In the present study no evidence has been obtained for the presence of more than two groups of fibres in either of the two components, crossed and uncrossed, of the optic nerve of the cat. The two waves of the compound spike potentials of the crossed and uncrossed fibres are, however, different in respect to amplitude and conduction velocity. Even after allowance is made for a probable increase in conduction distance for the crossed fibres at the chiasm they still remain significantly slower, group for group, than the uncrossed fibres. Investigations are at present in progress to determine whether the fibre-size spectra of the two components of the nerve show corresponding differences. The presence of such a difference gains support from the fact that the fibre-size spectra of the nerve as a whole that have been carried out in this laboratory have not shown the clear evidence for fibre grouping that might be suspected from the action-potential studies. This finding is, however, to be expected if the potential waves of the one side fall partly in the troughs of the other. This difference between the crossed and uncrossed fibres is rather surprising because, except for numbers, the two pathways are generally assumed to be essentially the same. It is possibly related to the asymmetrical arrangement of the fibres in the retina due to the fact that the area centralis is lateral to the fundus. This would provide a relatively shorter retinal path for the fibres destined to cross in the chiasm. Compensation for this asymmetry might then be provided partly by the increased conduction distance at the chiasm and partly by the alteration in the fibre-size spectrum suggested above.

The meaning of the pattern of fibre groupings in peripheral nerve is still obscure (Gasser, 1950). As far as the visual system is concerned fibres from both groups synapse in the lateral geniculate body (Bishop & McLeod, to be published), and work in progress in this laboratory indicates that they are also distributed to the superior colliculus and pretectal regions. It is probable, therefore, that neither group is wholly responsible for either the reflex aspects of visual function or those aspects such as pattern discrimination (object vision) which in the cat and higher forms depend almost entirely on the occipital cortex. The present investigation has provided no evidence for the triple nervous pathway which might be used to support the trichromatic theory of colour vision (Chang, 1951).

The time relations of the curve of recovery of excitability following a single shock for peripheral mammalian A fibres tested *in situ* (Gasser & Grundfest, 1936; Graham & Lorente de Nó, 1938; cf. also Grundfest, 1952) are very

similar to those reported here. The after-potentials have been found to be in keeping with the excitability curve. All the fibres in the optic nerve must therefore be classified among the A group. This demonstration implies that the fibres in the central nervous system have the same properties of conduction and recovery as do peripheral nerve fibres.

Gasser & Grundfest (1939) found that the delta fibres have an absolutely refractory period of 0·6–0·7 msec in the cat despite the fact that the spike duration appeared to remain constant at 0·4–0·45 msec. This increase in absolutely refractory period for the smallest fibres of the A group is confirmed by the present study and by the previous study in the pyramidal tract (Bishop, Jeremy & Lance, 1953). Unfortunately, no information is available about the spike duration of individual fibres in the optic nerve, although attempts at the reconstruction of the compound spike potential suggest a slightly longer duration for the smaller fibres.

The excitability curves of isolated peripheral nerve are characterized by a greater negative after-potential and therefore supernormal period than the same nerves tested *in situ* (Gasser & Grundfest, 1936). It is somewhat surprising, therefore, that the supernormal period for optic nerve fibres is both greater in degree and lasts longer than the corresponding phase for isolated peripheral A fibres. The negative after-potential is also of correspondingly longer duration and probably somewhat greater in amplitude. By contrast, the subnormal phase is frequently hard to detect and is of relatively short duration. These observations are supported by the fact that residual supernormality occurs following a brief tetanus (Fig. 8), since supernormality after a conditioning tetanus never remains in peripheral nerve following stimulation frequencies used in these observations (Gasser, 1935; Gasser & Grundfest, 1936; Graham & Lorente de Nó, 1938). Lloyd (1951) has studied the after-potentials, after-currents and recovery of excitability of the intra- and extramedullary axons of spinal motoneurones. Comparing the intramedullary axon cycle with that for the extramedullary axon the rate of transition from refractoriness to maximum supernormality is higher, the peak being reached in 2–4 msec, whereas the peak of enhancement of extramedullary axons occurs at 7–8 msec. Transition from supernormality to subnormality again is earlier, being at 10–12 msec, rather than 30–40 msec. At about 40 msec and onwards the subnormality of intramedullary axons is relatively less and of somewhat shorter duration. The recovery cycle of the smaller fibres in the optic nerve is almost identical with that of the extramedullary axons of spinal motoneurones. Lloyd attributes the differences he found between the two portions of the axon to aftercurrents associated with the cell body, but in view of the differences found in the present study between the large and small fibres under constant environmental conditions fibre diameter as a factor must be considered.

Graham & Lorente de Nó (1938) regarded their excitability curve of type D, which resembles so much those obtained in this series, as abnormal. In a similar manner they regarded the excitability curve in one cat preparation which retained an absolute supernormality during recovery from a tetanus as abnormal. Although the present experiments were carried out under conditions as closely approximating to normal as possible the stimulating bipole must have occasioned some damage to the fibres in the optic tract. Flows of demarcation current associated with this injury may have played some part in enhancing excitability. Despite this the excitability curve is strikingly similar to that for the extramedullary axons which Lloyd regarded as more characteristic than the intramedullary curve. Post-tetanic potentiation of supernormality (Amberson, Parpart & Sanders, 1931; Graham & Lorente de Nó, 1938; Lloyd, 1949) cannot be a factor because the curve was of the same form at the commencement of the experiment after no more than the stimuli needed to determine the excitability cycle. The degree of supernormality found in this series is therefore probably a normal property of optic nerve fibres.

SUMMARY

1. All the fibres of the optic nerve of the cat are myelinated and range in axonal diameter from 1 to 8·5 μ with very occasional larger fibres ($< 0{\cdot}15\%$).

2. Antidromic activation of the optic nerve by means of bipolar needle electrodes inserted into the optic tract demonstrates a segregation of fibres according to size in the tract, the smaller fibres being situated uppermost. This segregation is also clearly seen in histological preparations.

3. The monophasic action potentials of both the homolateral and contralateral optic nerves following stimulation of an optic tract display two peaks. Despite a correction for the possible increase in conduction distance caused by the chiasm the peak conduction velocities of the homolateral response remain significantly faster than those for the contralateral nerve. The peak conduction velocity of the homolateral small fibre group is about 3 m/sec faster than the corresponding contralateral group.

4. The ratio of conduction velocity (m/sec) to axonal diameter (μ) for the largest fibres is 8·2.

5. The curve of recovery of excitability following activation for both the large and the small fibres shows a well-marked phase of supernormality followed by a shallow phase of subnormality. Supernormal excitability remained for 30–40 msec, following the spike potential. Tetanic stimulation caused the recovery cycle to display deeply augmented subnormality but a tendency to supernormality always remained in the early part of the recovery cycle with the stimulation frequencies used.

6. The monophasic action potentials display well-marked negative after-potentials with durations comparable to the phase of supernormal excitability.

The positive after-potential which follows is of relatively small amplitude. Tetanic stimulation leads to summation of negative after-potentials with augmentation of the positive after-potential at the cessation of stimulation.

7. The periods of absolute and relative refractoriness suggest that these fibres have a spike duration similar to that of peripheral A fibres. The absolutely refractory period of the small fibres is longer than that of the larger fibres.

8. Except for the duration of the supernormal period in the recovery of excitability following stimulation, all the properties studied of the optic nerve fibres are similar to those which are characteristic of peripheral A fibres.

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