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THE CONDUCTION VELOCITY OF REGENERATED PERIPHERAL NERVE FIBRES

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During the early stages of regeneration after Wallerian degeneration, peripheral myelinated nerve fibres have a reduced conduction velocity (Berry, Grundfest & Hinsey, 1944; Erlanger & Schoepfle, 1946; Sanders & Whitteridge, 1946) and possess small axons, thin myelin sheaths and short internodes (Sanders, 1948; Vizoso & Young, 1948). Following nerve section and suture, Berry *et al.* (1944) found that conduction velocity increased progressively, as did fibre diameter, but that even at 466 days after operation maximal velocity did not exceed 80 % of the normal value. This finding was not surprising, as reconstitution of the normal fibre diameter distribution was known to be defective after nerve section and suture (Gutmann & Sanders, 1943). On the other hand, after regeneration following a localized crush lesion, Sanders & Whitteridge (1946) stated that full recovery of conduction velocity occurred, this being associated with restoration of fibre diameter (Gutmann & Sanders, 1943), internodal length remaining short (Hiscoe, 1947; Vizoso & Young, 1948). However, a seemingly contradictory result was reported by Erlanger & Schoepfle (1946), who noted that at 343 days after a crush injury, velocity had only returned to 62 % of the normal value, and it was suggested that a normal value might never be achieved by regenerated nerve fibres.

In view of the increasing use of measurements of nerve conduction velocity in the investigation of neuropathies in man (see Gilliatt, 1961; Thomas, 1961; Lambert, 1962), it seemed necessary to re-examine the question of conduction velocity in regenerated nerve fibres. Localized crush lesions have therefore been made on the peroneal nerves of rabbits, and conduction velocity examined distal to the crush after survival periods of 12 and 16 months. Measurements of nerve fibre diameter and internodal length were also made. Fibre diameter is known to be correlated with conduction velocity, but it is not known whether a reduction in internodal length would influence conduction velocity. Rushton (1951) has presented theoretical arguments to show that any reduction in velocity

would be small if the adult internodal length was optimal for conduction velocity, or a little longer.

METHODS

Experiments were performed on twelve male albino rabbits. The peroneal nerve on one side was crushed for 10 sec with smooth-tipped watchmaker's forceps 1 cm below the sciatic notch with full aseptic precautions, employing pentobarbital sodium (Nembutal, Abbott Laboratories) and ether anaesthesia. The opposite nerve was left untouched and used for control observations.

In order to ensure a long period of survival it was found necessary to limit the amount of pellet food provided and to supplement it with hay (as suggested by Mr D. J. Short of the National Institute for Medical Research), so that only a slow increase of body weight occurred. In earlier experiments, with unlimited pellet feed, an excessive obesity developed and sudden deaths were common late in the survival period.

At biopsy the sciatic nerve trunks were removed from the two sides under pentobarbital sodium anaesthesia between the levels of the sciatic notch and the knee and the peroneal nerves separated from the tibial nerves under a dissecting microscope. Conduction velocity was measured in both peroneal nerves in ten rabbits. After being washed in oxygenated saline, the nerve trunk was blotted on filter paper and arranged on stainless-steel electrodes placed at intervals of 5 mm in a bath of mineral oil maintained at 38° C. Rectangular stimulating pulses of 0·1 msec duration were delivered through an isolating transformer to the electrodes at one end of the nerve trunk. After adjusting the stimulus voltage to elicit a maximal A fibre potential, the recurrence rate was set at 1/sec, and the stimulus parameters were not changed thereafter. The cathode-follower input leads were connected to electrodes at the other end of the nerve, and after each photograph had been taken these leads were moved to electrodes 5 mm nearer to the stimulated end of the nerve. One of the electrodes between the positions of stimulation and recording was earthed.

The oscilloscope traces were photographed at full size on recording paper; each photograph contained a 10 kc/s marker that was locked both to the trace and to the stimulator, so that successive traces and markers could be superimposed. The latency of a point half way up the rising face of the action potential spike was plotted against conduction distance, and the conduction velocity was determined from the best straight line drawn by eye. The half-rise latencies were found to be more accurately proportional to conduction distance than the point of inflexion or the peak of the action potential, which were less easily defined.

The nerves were washed in saline after removal from the paraffin bath and then blotted on filter paper. Short lengths from the middle of the nerve and from the distal and proximal ends were attached to card frames and fixed in Flemming's solution, embedded in paraffin wax and cut transversely at 5 μ, to be stained by the modified Weigert method described by Gutmann & Sanders (1943). The remaining lengths of nerve trunk were attached to card frames and fixed in 10% neutral formol-saline. Some pieces of this material were later stained with osmium tetroxide and teased apart in glycerine by the method described by Thomas (1955) in order to examine the internodal lengths of single nerve fibres.

Measurements of internodal length were made on the isolated fibres at a magnification of 100 times, by means of a microscope with an ocular micrometer. Five measurements of diameter were then made along each internode at a magnification of 1000 times with an oil-immersion objective, the mean of these readings being taken.

In the Weigert-stained transverse sections measurements of diameter were made on the twenty largest fibres at the three levels in the operated and control nerves. The measurements were obtained by projection on to a screen at a magnification of 750 times, with a projecting microscope. The outer diameters of the myelin sheaths were matched against circles scored on a Perspex sheet, the fibres being marked with a wax pencil on the screen

after counting. Spherical aberration was not measurable over the portion of the screen on which the measurements were made.

Two rabbits were killed 3 weeks after operation in order to test the efficacy of the crush in producing Wallerian degeneration distal to the lesion. A segment was removed from the operated nerve in the lower thigh in each animal and fixed in Flemming's solution for Weigert staining of transverse sections as detailed above. No intact myelinated nerve fibres were present. It was therefore evident that the operation was effective in producing a lesion that interrupted all myelinated fibres.

RESULTS

Electrical observations

Biopsy was carried out on five rabbits at about 12 months, and on another five at 16 months after operation. In all cases the plot of latency against conduction distance was linear in the 70–80 mm below the crush. Figure 1 shows an example of the records obtained from the crushed and control peroneal nerves after 486 days survival. The amplitudes and durations of the action potential spikes were always similar on the two sides, and no excessive dispersion occurred in the spike of the regenerated nerve over the distance available for conduction. In some experiments the operated nerve was placed in the mineral oil first, while the control nerve was left in oxygenated saline, but in other experiments the order was reversed without apparently affecting the relative conduction velocities. Gutmann & Holubář (1950) found a reduction in conduction velocity in nerves kept *in vitro*, but this was only apparent after 24 hr, whereas the measurements on any one nerve in the present experiments were completed in 15 min.

The consistency of the measurements of latency is shown by the graphs in Fig. 2. The position of the latency scale is arbitrary, since the measurements were made from the time marker without correction for the small latency of the stimulus. There was no consistent difference in the extrapolated latency for zero distance of conduction between the two sides in the nerves studied. The latency graphs clearly indicated a reduced velocity in the regenerated nerves, and Table 1 summarizes the results for the two groups of rabbits.

The mean value for the conduction velocity of the control nerves was 87·4 m/sec. Conduction velocity in these nerves showed a positive correlation with nerve-fibre diameter ($r = 0{\cdot}7$; $P < 0{\cdot}05$), but not with the weight of the animal. The values for fibre diameter are given in Table 2. When the two groups of survival times were compared, conduction velocity was observed to be slightly greater in the longer-survival group, although the difference was not statistically significant ($P = 0{\cdot}1$ by Student's t test).

The mean value for conduction velocity in the regenerated nerves was 66·1 m/sec, this being 21·3 m/sec less than that obtained in the control nerves, representing a reduction of approximately 25 %. The reduction was statistically significant ($P < 0{\cdot}05$). As in the control nerves, conduction

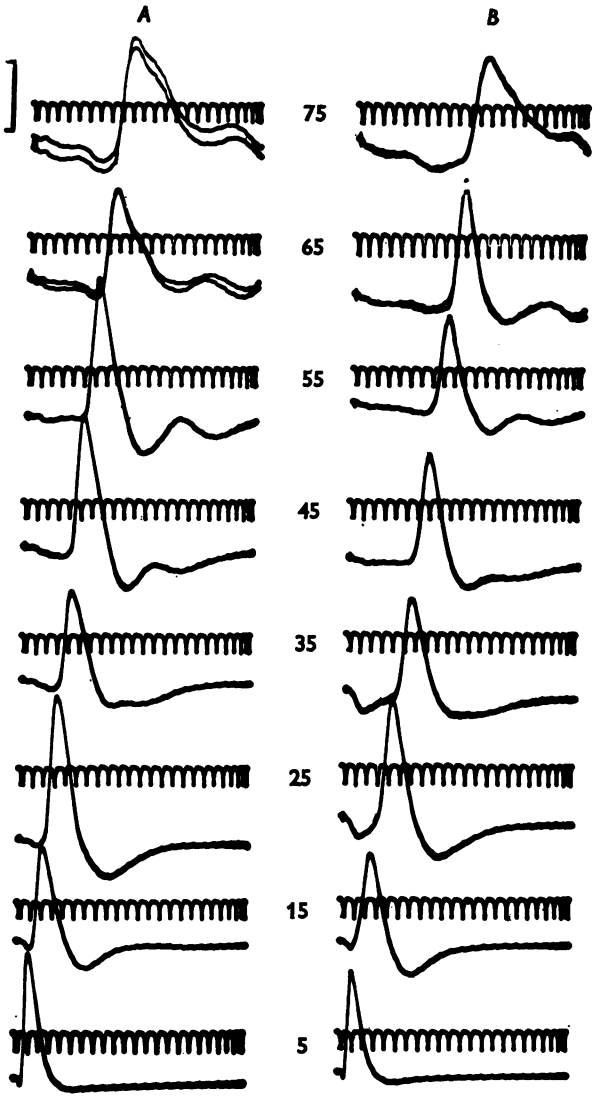

Fig. 1. Action potentials from the peroneal nerve of a rabbit 486 days after a proximal crush (*B*) compared with those from the contralateral control side (*A*). Conduction distances in millimetres; time-marker 10 kc/s; vertical bar represents 1 mV.

velocity was slightly greater in the longer-survival group, but this difference was not significant ($P = 0{\cdot}1$). When the mean values for the ratios of the velocities of the regenerated and control sides, expressed as percentages, were compared, the values for the two groups were found to be remarkably close (75·3 and 76·0 % respectively). Thus no further recovery in conduction velocity occurred after 1 year following crushing.

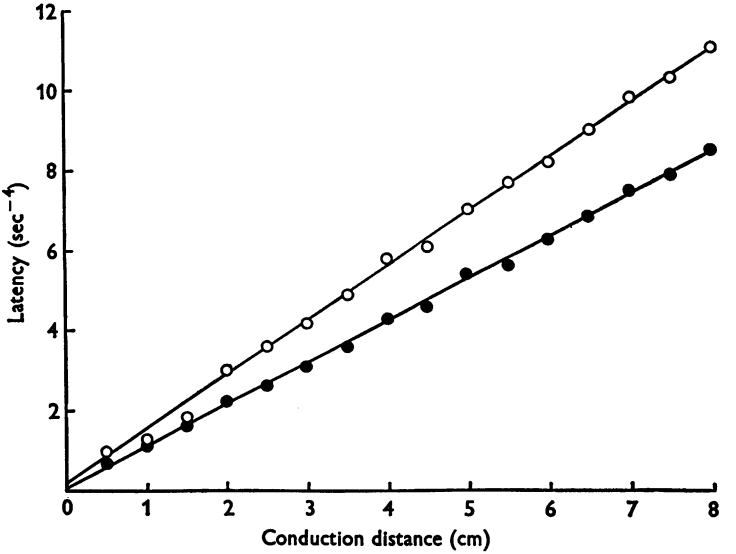

Fig. 2. Plot of conduction distance against latency of points half way up the rising faces of the action potentials shown in Fig. 1, together with measurements taken from similar records after reversing the nerve on the electrodes. The conduction velocity of the control nerve (●) is 96·7 m/sec, that of the regenerated nerve (○) 74·4 m/sec, or 76·9 % of the control side.

TABLE 1. Conduction velocity in the control nerves (C) and the regenerated nerves (R) and their ratios expressed as percentages for the two groups of survival periods

Rabbit no.	Survival period (days)	Conduction velocity (m/sec) C	R	$R \times 100/C$	Weight (kg)
1	364	82·5	60·0	72·7	3·1
2	368	73·5	57·3	78·0	3·5
3	385	94·0	71·4	75·5	2·7
4	388	88·0	65·0	73·8	3·8
5	430	80·0	61·0	76·3	3·8
Means		83·6	62·9	75·3	3·38
6	485	84·9	65·2	76·8	2·95
7	485	91·8	66·7	72·7	3·0
8	486	93·7	66·7	71·2	3·05
9	486	96·7	74·4	76·9	3·7
10	486	89·0	73·0	82·2	2·7
Means		91·2	69·2	76·0	3·08
Over-all means		87·4	66·1	75·6	3·23

TABLE 2. The diameter (μ) of the twenty largest myelinated nerve fibres in the control and regenerated nerves at the proximal (P), middle (M) and distal (D) levels, and for the three levels combined (T), for the two groups of survival periods

Rabbit no.	Survival period (days)	Control nerves				Regenerated nerves				$T_r \times 100/T_c$
		P	M	D	T_c	P	M	D	T_r	
1	364	18·7	18·8	18·6	18·7	17·9	16·1	16·2	16·7	89·3
2	368	—	18·8	19·8	19·3	21·2	16·1	16·0	17·8	92·2
3	385	20·8	20·3	19·1	20·1	20·4	17·3	16·3	18·0	89·6
4	388	18·3	18·3	18·5	18·4	16·9	16·3	16·6	16·6	90·2
5	430	16·5	16·0	16·1	16·2	14·2	13·7	14·0	14·0	86·4
Means		18·6	18·4	18·4	18·5	18·2	15·9	15·8	16·6	89·6
6	485	22·1	20·5	19·1	20·6	20·2	19·3	16·6	18·7	90·8
7	485	22·1	20·7	20·2	21·0	21·2	16·3	17·1	18·2	86·7
8	486	22·1	20·2	20·0	20·8	22·2	18·1	17·5	19·3	92·8
9	486	23·5	21·8	22·2	22·5	18·3	20·1	20·2	19·5	86·7
10	486	20·9	20·3	20·6	20·6	18·2	18·4	18·9	18·5	89·8
Means		22·1	20·7	20·4	21·1	20·0	18·4	18·1	18·8	89·4
Over-all means		20·6	19·6	19·4	19·8	18·8	17·2	16·9	17·7	89·4

Histological observations

The results obtained for the measurements of the size of the twenty largest myelinated nerve fibres in the regenerated and control nerves from the ten animals in which measurements of conduction velocity were made are given in Table 2. The specimen from the proximal level of the control nerve of animal 2 was unfortunately lost during histological preparation.

When the regenerated and control nerves were compared, the mean values were less in the regenerated nerves at all three levels. At the proximal level, the mean fibre diameter for the regenerated nerves was 18·8 μ, as compared with 20·6 μ in the control nerves. This difference of 1·8 μ, however, did not reach the level of statistical significance ($P > 0{\cdot}1$). At the middle and distal levels, mean values of 17·2 and 16·9 μ were obtained as compared with 19·6 and 19·4 μ in the control nerves. Both these differences were significant ($P < 0{\cdot}01$). These results suggested a distal tapering of the regenerated nerve fibres. This was tested by comparing the mean values for the distal and proximal levels, and the difference just reached the level of significance ($P = 0{\cdot}05$). The degree of tapering was somewhat variable, but it did not differ between the early and late survival groups. In the control nerves there was no significant difference between the mean values for the proximal and distal levels ($P > 0{\cdot}1$).

A comparison was also made between the shorter- and longer-survival period groups. For the control nerves the mean diameter for the shorter-survival group, combining the results from all three levels in the nerves, was 18·5 μ, as compared with 21·1 μ for the longer-survival group. This

difference was statistically significant ($P < 0{\cdot}01$). A similar difference was evident in the regenerated nerves, where the corresponding values were 16·6 μ and 18·8 μ; this was also significant ($P < 0{\cdot}05$). Thus both the control and the regenerated nerves showed small increases of fibre diameter between the two survival times. However, as was found for con-

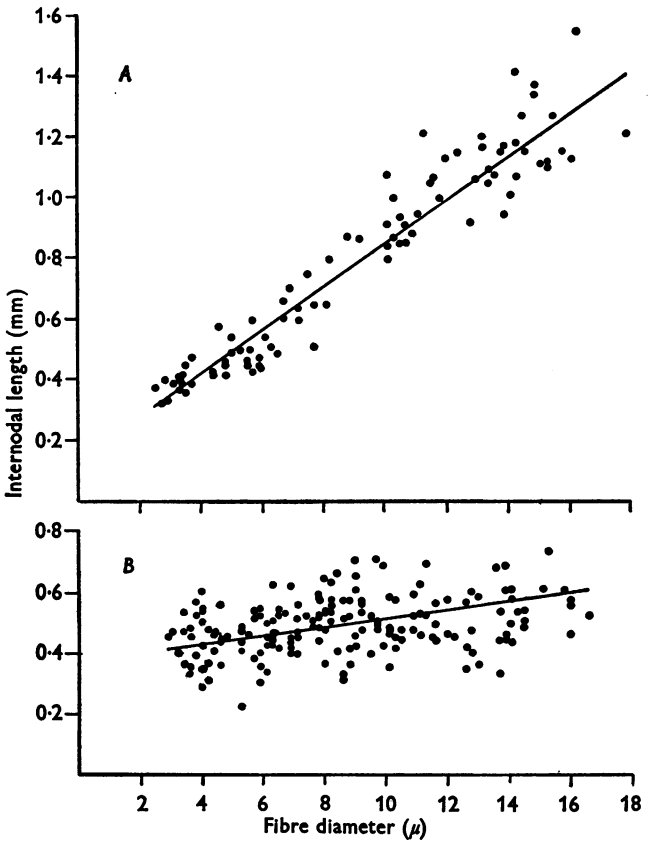

Fig. 3. Relationship between internodal length and fibre diameter for individual internodes from the control (*A*) and regenerated (*B*) nerves of a rabbit operated 364 days previously. Calculated regression lines have been fitted.

duction velocity, the mean percentage ratios for the two groups were extremely close (89·5 and 89·4 %).

Measurements of internodal length were made on the regenerated and control nerves of a rabbit examined 364 days after operation, and the results obtained are shown in Fig. 3. The nodes were usually easily recognized, except on very small fibres, and were of normal appearance in the regenerated nerves. There was a positive correlation between inter-

nodal length and fibre diameter in both nerves, the relation being approximately linear. Calculated regression lines have been fitted. For the control nerve, the slope was 0·072 mm/μ, the largest fibres isolated having an internodal length of about 1·3 mm. The slope was considerably less in the regenerated nerve, being only 0·014 mm/μ, the largest fibres having an internodal length of about 0·6 mm. Internodal length was also measured in the regenerated nerve of a second rabbit, examined 368 days after operation, with similar findings. The results obtained here correspond very closely to those previously reported by Vizoso & Young (1948), who also examined the rabbit peroneal nerve after regeneration following a crush lesion, and confirm that nerve regeneration is not accompanied by recovery of internodal length.

DISCUSSION

The present investigation has revealed a consistent reduction in conduction velocity in regenerated nerve fibres after crush lesions, amounting to approximately 25 % of the normal value. There was no significant increase in conduction velocity between the observations at 12 months and those at 16 months. Although our earlier- and later-survival groups were separated by 4 months, it might be argued that had a longer interval been allowed, a significant improvement might have been found. Against this is the fact that the percentage recovery of velocity (75·3 % at 12 months, 76·0 % at 16 months) was nearly static. This result therefore supports the suggestion made by Erlanger & Schoepfle (1946) that a fully normal conduction velocity is never regained by regenerated nerve fibres.

In seeking an explanation for the disparity between our results and those of Sanders & Whitteridge (1946), who did not detect a significant reduction in conduction velocity of regenerated rabbit peroneal nerves 16 months after crush lesions, it is important that Sanders & Whitteridge based their conclusion on measurements made on four nerves from two rabbits in which bilateral operations had been performed. The average maximal conduction velocity for these nerves was 63·3 m/sec, which is close to the mean value for the operated nerves in the present study, namely 66·1 m/sec. The average conduction velocity for the unoperated nerves in our series was 87·4 m/sec, whereas that for twelve normal nerves examined by Sanders & Whitteridge was only 68·6 m/sec. It is possible that the latter were from young or recently acquired animals, as Quilliam (1958) found increases in fibre diameter in the sural nerves of rabbits kept for prolonged periods as compared with those more recently acquired, and this was even apparent between the longer- and shorter-survival groups in the present study. Little detailed information is available about the factors regulating post-natal growth in the rabbit, but it is clear that

changes of this nature must be considered in long-term experiments on the peripheral nerves of this species. The situation may differ between laboratory animals, for Birren & Wall (1956) detected no significant change in conduction velocity in the sciatic nerves of rats between 350 and 650 days of life.

In the present experiments the comparisons were made between the operated and contralateral unoperated nerves in the same animals, thus eliminating the complication of age changes. There is a close symmetry in fibre size between the nerve to the medial head of the gastrocnemius muscle in the rabbit on the two sides, and the differences existing between sides are considerably less than those between animals (Causey, 1948; Quilliam, 1956; Aitken & Thomas, 1961). This is also true of the sural nerve (Quilliam, 1956). Although no comparable anatomical data are available for the peroneal nerve, there is no reason to believe that the situation would be different, and Cragg & Thomas (1961) found that conduction velocity in this nerve is normally closely similar on the two sides. A possible objection to using the unoperated contralateral nerve for control purposes is the observation by Greenman (1913) that following crushing the peroneal nerve on one side in rats, the contralateral nerve showed a diminution in fibre diameter. But such an effect was not confirmed in rabbits by Quilliam (1958) or by Aitken & Thomas (1961) and seems improbable.

The absence of a significant difference between the diameter of the largest fibres of the regenerated and control nerves at the proximal level in the present experiments is in agreement with the observations of Gutmann & Sanders (1943). They reported that 1 cm below a crush lesion of the peroneal nerve in a rabbit examined 250 days after operation, fibre size was fully restored, although more distally it was still reduced. But in another animal, examined 300 days after operation, fibre diameter was only slightly less distally than it was 1 cm below the lesion, where it had fully recovered. The present results showed a certain amount of variability as to the degree of tapering exhibited by the largest fibres. In the sural nerve of the rabbit Quilliam (1958) observed that after a crush lesion, few if any of the larger fibres in the peripheral stump ever attain the diameter of those in the central stump, and that the deficit is greater distally.

The reason for the deficit in fibre diameter distally is uncertain. Shrinkage of the endoneurial tubes occurs during Wallerian degeneration (Holmes & Young, 1942; Sunderland & Bradley, 1950) and it is possible that subsequent re-expansion is limited by endoneurial fibrosis (Sanders & Young, 1944; Thomas, 1964). It seems unlikely that loss of the larger fibres in the central stump because of retrograde degeneration of the parent neurones

could be responsible: neither Gutmann & Sanders (1943) nor Quilliam (1958) found a reduction in the number of myelinated fibres central to crush lesions in the thigh.

The distal reduction in fibre diameter noted in the present experiments is small and seems insufficient to account for the whole of the reduction in conduction velocity, which amounts to an average value of 21·3 m/sec. Hursh (1939) found that for the cat there was a linear relation between conduction velocity and fibre diameter, the slope of the line indicating a coefficient of 6·0 m/sec/μ. The coefficient for the rabbit will be less, since although the diameter of the largest myelinated nerve fibres in the peroneal nerve is similar in the two species (*ca.* 20 μ), maximal conduction velocity is less in the rabbit. In the control nerves of the present study the mean diameter of the twenty largest fibres was 19·8 μ and maximal conduction velocity 87·4 m/sec. Assuming a linear relation between conduction velocity and fibre diameter, this would indicate a slope of 4·4 m/sec/μ. As recordings were not made from the extreme ends of the excised portions of nerve, it would seem most justifiable to take the diameter values obtained from the middle of the nerve for comparison with velocity. The mean reduction in fibre diameter at this level was 2·5 μ, which would correspond to a reduction in velocity of only 11·0 m/sec. If all levels are combined, the calculated reduction amounts to 8·8 m/sec. It is clear, however, that these values can only be approximate, as it is not certain whether the measurements of conduction velocity and fibre diameter correspond exactly to the same fibres in the nerve. But it nevertheless seems probable that some factor other than fibre diameter is also contributing to the reduction in conduction velocity. Although the nodal gaps are wider during the earlier stages of regeneration (Sanders, 1948), they were of normal appearance in the present nerves. Furthermore, the axon-diameter:myelin-thickness ratio of regenerated nerve fibres is very nearly normal at this stage (Sanders, 1948). Internodal length, on the other hand, is reduced by slightly more than 50 % on the largest fibres. Yet it is doubtful whether the reduced internodal length contributes to the reduced conduction velocity. J. E. Thomas & Lambert (1960) showed that in human nerves conduction velocity reaches the adult value at about 5 years, at which time adult values of fibre diameter are also attained, whereas internodal length continues to increase until growth in length of the nerves ceases (Vizoso, 1950). The full explanation of the slowing of conduction in regenerated nerves is therefore unknown.

SUMMARY

1. The peroneal nerve was crushed in the upper thigh in twelve adult rabbits and allowed to regenerate. Maximal conduction velocity was

measured *in vitro* in the operated and unoperated peroneal nerves of ten animals, 12 and 16 months after injury.

2. Conduction velocity was found to be consistently reduced in the regenerated nerves below the crush, being approximately 75 % of that in the unoperated nerves. There was no significant difference between the degree of reduction present at 12 and 16 months after operation.

3. The diameter of the largest myelinated nerve fibres showed a small reduction in the distal portions of the operated nerves, which was not seen in the control nerves. Internodal length was measured in the operated nerves from two animals and was found to be short on fibres of all diameters.

4. The explanation of the reduction in conduction velocity is discussed.

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