EFFECTS OF STRETCHING THE TIBIAL NERVE OF THE RABBIT
A Preliminary Study of the Intraneural Circulation and the Barrier Function of the Perineurium

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An injury of a peripheral nerve often involves a break in its structural and functional continuity. A small gap may be closed by suture without significant tension. In the case of a more extensive gap, however, the surgeon has to choose between two methods of treatment, namely, suture under tension, to avoid an excessive amount of which often requires extensive mobilisation, or bridging by a graft. The point at which attempts to achieve end-to-end union should be abandoned in favour of grafting must depend on the extent to which a nerve can be stretched without interfering with the process of repair.

A research programme has been designed in order to provide an experimental basis for the treatment of such peripheral nerve lesions. As a first step the present study was initiated for the purpose of introducing an experimental model and of making some essential observations on the response of the intraneural vasculature to stretching. Qualitative and quantitative changes in microvascular flow as well as an increase in permeability are early indications of tissue injury. Accordingly, these parameters were chosen for analysis.

Both clinical experience and experimental studies have made it clear that nerve trunks are highly resistant to stretching (Mitchell 1872, Denny-Brown and Doherty 1954, Leffert and Seddon 1965). The functional changes in a stretched nerve are highly dependent on the magnitude and character of the deforming force, as well as on the length of time during which it operates. The internal topography of the nerve trunk also plays an important role. The nerve fibres are distributed throughout the intrafascicular endoneural space. The fascicles—each surrounded by perineurium—are embedded in the epineurial tissue. The number, size and arrangement of the fascicles, together with the amount of epineurium present, influence both the severity and site of the stretch lesions (Sunderland 1968).

The stress-strain properties of peripheral nerve trunks have been extensively investigated (Sunderland and Bradley 1961, 1968, Haftek 1970), and it is obvious that peripheral nerves possess a high degree of elasticity. However, with increasing traction force a point is reached when conduction is blocked though no significant morphological change can be detected (Sunderland 1968). A further increase leads to disruption of axons and fascicles.

Under tension the cross-sectional area of the fascicles is reduced, causing an increase in intrafascicular pressure and consequent interference with intrafascicular nutritive blood flow. The importance of a sufficient blood supply for the function of the nerve is well known (Koch 1926, Gerard 1930, Bentley and Schlapp 1943, Porter and Wharton 1949, Sunderland 1968, Lundborg 1970). Therefore stretching sufficient to interfere with the intraneural microcirculation can be expected to impair nerve function.

The present study is based upon the method of in vivo observation of intraneural microcirculation described by Lundborg (1970) and his findings on the structure and function of the microvessels. Interest has also been focused on the perineurial sheath surrounding the nerve fascicles. Normally the perineurium constitutes an effective diffusion barrier for several substances including proteins (Sunderland 1968), and so helps to preserve a constant and favourable intrafascicular environment. However, in various types of injury this selective function could be modified. Stretching might damage the perineurium and affect its permeability. Leakage of proteins into the fascicles from injured extraneurial and epineurial blood vessels could lead to fibrosis and jeopardise the return of normal structure and function.
Therefore, as a natural part of the present study, special interest was paid to the perineurium and its function as a diffusion barrier.

MATERIAL AND METHODS

Thirty rabbits of both sexes, weighing 1.4 to 1.8 kilograms, were used. The animals were anaesthetised with 1.5 grammes of urethane per kilogram body weight, injected in physiological saline into an ear vein. The studies were performed on the tibial nerve, which is easily exposed near the ankle by the method described by Lundborg (1970) (Fig. 1).

Traction was applied to the nerve in the following way. After dissection over some 2 centimetres, two epineurial sutures of 6-0 silk were applied 1.5 centimetres proximal to the ankle joint. The nerve was then cut immediately distal to the sutures, about 6 centimetres of which were retained. These free ends were fastened to equipment enabling continuous and controlled stretching of the nerve (Fig. 2). The aim was not to measure the tension force applied, but rather to follow the movement of the proximal end of the nerve distally. This "stretching distance" was used to estimate the true elongation of the nerve expressed as a percentage. During the stretching period the exposed nerve was continuously irrigated with Tyrode's solution at room temperature.

Four series of experiments were performed in connection with the stretching procedures.

Series A: Observation of intraneural microvascular flow patterns—The microcirculation was observed about 0.5 centimetre proximal to the cut end. The nerve was transilluminated by a quartz glass rod and viewed with a modified Leitz intravital microscope (Bränemark 1962, 1963). Low voltage xenon lamps provided the sources of light. Heat-absorbing filters were used to reduce the increase in local tissue temperature. In addition, the studies were performed in the green part of the spectrum at about 5,500 Å.
During the controlled stretching the light conductor was continuously moved distally so that the same field was in focus throughout. The analysis involved observations of intravasal rheological phenomena as well as quantitative changes in flow in arterioles, capillaries and venules. In every experiment the results were based on observations made on six to eight epineurial venules, two to four arterioles and two or three intrafascicular nutritive units (arteriole-capillary-venule).

The aim was not to achieve continuous numerical data on the reduction in microvascular flow with stretching. However, two well-defined "critical limits" could easily be recognised. At a certain degree of tension ("lower tension limit") the first intravasal evidence of tissue injury could be noticed: granulocytosis and the occurrence of microthrombi and emboli, generally accompanied by detectible reduction of flow in more than 50 per cent of the venules. Further stretching caused increasing impairment of the microcirculation until no flow could be seen. This degree was called the "upper stretching limit" and was maintained for half an hour. Then the traction was released and the return of blood flow was observed in the completely slack nerve for at least half an hour (the "relaxation period").

**Series B: Analysis of changes in microvascular permeability**—Evaluation of permeability was performed during the "relaxation period". Changes were demonstrated by tracing serum albumin tagged with Evan's blue. This tracer solution, here called EBA, was prepared by mixing 5 per cent bovine serum albumin with Evan's blue, following the method of Steinwall and Klatzo (1965) and Olsson (1966). The conjugate was carefully filtered before use through a Sephadex column for removal of free tracer. The standard dose was 1 millilitre of 5 per cent labelled albumin solution per 100 grammes body weight. This solution was slowly injected into an ear vein immediately the nerve became slack. The animals were killed thirty minutes later. Specimens of nerve taken 1 centimetre from the cut end were fixed in 5 per cent formalin for twenty-four hours. Frozen longitudinal sections 10 μ thick were mounted in 50 per cent aqueous glycerine and immediately examined in a Leitz fluorescence microscope equipped with a dark field condenser and an Osram HBO 200 watt high pressure mercury lamp. The light was directed through a Schott BG 12/3 millimetre filter, the emitted light passing in the tubes through a K 510 filter. Under these conditions, Evan's blue-albumin emits a bright red fluorescence, and can easily be traced.

**Series C: Analysis of changes in the function of the perineurial barrier**—Analyses of this function were performed during the relaxation period, the same tracer being used. Two cubic centimetres were injected around the nerve and its epineurial sheath in situ. After two hours of contact, specimens were taken from the nerve 1 centimetre proximal to the cut end. Analyses of the distribution of the tracer were performed in ultra-violet light by the method described above.
Series D: Estimation of the true elongation of the nerve—When stretching was applied to the proximal nerve segment, the change of position of this end in a distal direction ("stretching distance") could be exactly measured with the aid of the equipment described in Figure 2. Because of the elastic properties of the nerve, points situated more proximally in the nerve did not, of course, move distally to the same extent. In order to estimate the true elongation of the nerve segment, a dissection was performed about 4 centimetres proximal to the cut end and a fine epineurial suture was applied (Fig. 3). The change in the position of this point in relation to the cut nerve end was continuously measured and the elongation of the nerve could then be expressed as a percentage (Fig. 7).

![Fig. 3](image)

A schematic drawing showing the tibial nerve with normal tension (above) and under increasing tension. Normally the regional nutrient vessels are coiled and tortuous. When slight tension is applied to the nerve their "reserve in length" is taken up and the coiled appearance is lost. Stronger tension results in stretching of the regional vessels (below). Due to the elastic properties of the nerve, the point (a) situated proximally does not move distally to the same extent as the more distal point (b) where the epineurial sutures are applied. The change in the position of (b) is called the stretching distance (△b). An epineurial suture applied at (a) allows measurement of the movement distally of this point (△a). The elongation of the nerve expressed as a percentage is \( \frac{△b - △a}{ab} \times 100 \). (After Lundborg 1970. By kind permission of the Editor of the Scandinavian Journal of Plastic and Reconstructive Surgery.)

RESULTS

Series A: Intraneurial microvascular flow patterns—There were fifteen experiments (Table I and Fig. 6). Prior to stretching, the intraneurial vasculature presented the architecture and flow patterns reviewed by Lundborg in 1970. The plexuses in the epineurium, perineurium and endoneurium could easily be recognised, the numerous anastomoses making a characteristic design. The intrafascicular, endoneurial capillary bed consists of vessels mostly parallel to the long axis of the nerve but sometimes oblique or perpendicular (Fig. 4). These intrafascicular vessels communicate freely with the extrafascicular vessels by numerous anastomoses penetrating the perineurial sheath.

When stretching was first applied to the nerve there was initially no detectible interference with the blood flow in any layer. As the tension increased, however, there was a slight slowing down in venular flow in epineurium and perineurium, and complete stagnation in some of the transverse anastomoses. At this stage the first intravascular evidence of tissue injury was generally noticed: slight granulocytosis and the appearance of microthrombi and emboli.
These first signs of a change in flow patterns, involving a definite reduction in more than 50 per cent of the observed venules, generally occurred at a stretching distance of 4 to 7 millimetres (Table I), which corresponded to elongation of the nerve by 5 to 10 per cent, the mean value of 8 per cent being obtained from the curve presented in Figure 7. This degree of stretching will be referred to as the "lower stretching limit".

No interference with arteriolar flow or intrafascicular capillary flow could be detected at this stage. With increasing tension, however, there was a continuous general decrease in intraneural blood flow and a considerable increase in the number of granulocytes rolling along the vessel walls.

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Mean value 7.9±8 Mean value 15.4±15

After a short interval of rapid impairment of arteriolar and capillary flow, a complete standstill in all intraneural vessels became evident at a stretching distance of 8 to 12 millimetres, corresponding to elongation by 11 to 18 per cent with a mean value of 15 per cent. This tension, the "upper stretching limit", was maintained for thirty minutes. After complete relaxation of the nerve the intraneural vessels regained their flow and a marked hyperaemia occurred. In some cases the return of flow was incomplete, a few venules remaining occluded. However, in all cases the arteriolar flow and the capillary flow appeared to be completely restored. For some time after the release of tension there was a persistent granulocytosis and numerous microthrombi and emboli were observed.

**Series B: Microvascular permeability**—There were eight experiments. The studies were performed on nerves at the "upper stretching limit" for half an hour, after which they were completely relaxed. The vital microscopic studies had revealed that the intraneural blood

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flow was restored after the stretching period. Consequently, injection of tracer solution (EBA) during the relaxation phase resulted in perfusion of the vascular bed.

Microscopic examination revealed a good filling of endoneurial vessels, with the tracer invariably confined to the vessel lumen. In no case was there any indication of leakage of albumin through the vessel walls (Fig. 5).

**Series C: The function of the perineurial barrier**—There were eight experiments. The analyses of permeability were performed during the relaxation period. The tracer solution (EBA) was left around the nerve for two hours before the specimens of nerve were removed.

In no case was any fluorescence observed in the endoneurial space (Fig. 5). However, a bright red fluorescence was clearly visible throughout the epineurium, showing that the

**Fig. 4**

A schematic drawing of the intrafascicular microvascular architecture. (P) perineurium, (a) arteriole, (v) venule, and (c) capillary. Note the capillary loops, sometimes arranged in planes perpendicular to the long axis of the nerve. Arrows indicate the direction of flow. (From Lundborg and Brånemark 1968. By kind permission of the Editor of *Advances in Microcirculation*.)
To show the distribution of Evan's blue-albumin (EBA) in the endoneurial space under different experimental conditions. Before the relaxation of tension the nerve had been kept at the "upper stretching limit" for half an hour. In ultra-violet light the EBA fluoresced bright red (here white) and the nerve tissue green (here black). Above (a), the distribution of EBA following intravenous injection half an hour previously. * Endoneurial capillary, ** Perineurial layer. The EBA is strictly confined to the capillary lumen. Below (b), the distribution of EBA following local application to the nerve for two hours, starting immediately after the release of tension. * Diffuse, extrafascicular fluorescence in the epineurium. ** Perineurium surrounding a fascicle. No endoneurial fluorescence can be observed. Note the increased concentration of tracer immediately outside the perineurial layer.
**EFFECTS OF STRETCHING THE TIBIAL NERVE OF THE RABBIT**

**FIG. 6**
Graphic representation of the effects of stretching on intraneural microvascular flow, based upon the mean values presented in Table I. The flow velocity in the different components of the vascular bed was estimated by sight.

**FIG. 7**
Graphic representation of the correlation between stretching distance and true elongation of the nerve. Data obtained from this curve were used to prepare Table I and Figure 6.
tracer solution had penetrated this layer thoroughly. The perineurium thus presented a sharp contrast between the bright-red epineurium and the intrafascicular space, green from autofluorescence of the nerve fibres (Fig. 5). Obviously, under the prevailing experimental conditions, the perineurium still maintained its function as a barrier to proteins, as further evidenced by an increase in the concentration of red fluorescence immediately outside that layer.

**Series D: Estimation of the true elongation of the nerve**—There were ten experiments (Table I and Fig. 7). This part of the study was performed in order to find an adequate expression for elongation. The experiments in Series A, involving observations of intraneural blood flow, did not permit the dissection required for simultaneous estimation of elongation of the nerve. This special series of ten experiments was performed for the purpose of analysing the correspondence between the “stretching distance” and true elongation of nerve in the experimental model. The results, based on the mean values obtained from the ten experiments, were represented graphically (Fig. 7). This curve, showing almost linear correlation between “stretching distance” and elongation, was then used for estimating the elongation of the nerve during the observations made in Series A, B and C.

**DISCUSSION**

In most studies of the effects of stretching of peripheral nerve trunks, interest has been focused on the mechanical properties of the nerve tissue. A nerve trunk comprises several tissues with varying degrees of resistance to stretch, and different suggestions regarding the elastic properties of the various components have been put forward (Sunderland 1968, Haftek 1970). From those investigations it is obvious that a peripheral nerve trunk, besides having a high degree of elasticity, is a very strong structure. Thus Sunderland and Bradley (1961) reported that loads ranging from 18 to 165 kilograms are required to rupture the median and ulnar nerves. Concerning the point at which stretching leads to detectible damage there is profound disagreement. Denny-Brown and Doherty (1945) reported 100 per cent elongation without damage in the cat, compared with 25 to 50 per cent given by Hoen and Brackett (1956) as the critical limit in dogs. Sunderland and Bradley gave a figure of 20 to 32 per cent from experiments on cadavers. According to Mitchell (1872) failure of conduction occurred in rabbit’s sciatic nerve after an elongation of 25 per cent. On the basis of histological examination severe damage was observed after elongation by 11 per cent (Hight and Sanders 1943) and 4 per cent (Liu, Bend and Lewey 1948). Recently Haftek reported the mean elongation at the “limit of elasticity” to be 69.3 per cent, and at the point of rupture 73.3 per cent.

In the present study attention has been paid not so much to the mechanical properties of the nerve trunk as to changes in the intraneural microcirculation. It is reasonable to assume that stretching, which involves changes in the normal intraneural topography, interferes with microvascular structure and function in the nerve, thus jeopardising the nutrition of the nerve fibres. It is a well-established fact that peripheral nerves require an adequate and continuous supply of oxygen to function properly. Accordingly, all procedures interfering with intraneural blood flow can be expected to induce disturbances of nerve function.

The anatomy of the intraneural blood vessels has been analysed in numerous investigations (Adams 1942, Sunderland 1945 a, b, c, Roberts 1948, Richards 1951, Blunt 1957, Lang 1962, Lundborg and Brånemark 1968, Lundborg 1970). Lundborg observed in vivo the intrafascicular microcirculation in the tibial nerve of the rabbit under various experimental conditions such as extensive mobilisation and sectioning.

According to Lundborg the vascular bed of a nerve can be separated into an extrinsic and an intrinsic system. The extrinsic system consists of regional vascular branches of varying dimensions, approaching the nerve at different levels along its course, penetrating the nerve, branching and anastomosing with the intraneural microvascular bed. The intrinsic system consists of vascular plexuses in the epineurium, the perineurium and the endoneurium. In the endoneurial space the vessels are mainly capillaries and so constitute the nutritive vascular...
bed for the nerve fibres (Fig. 4). These intrafascicular vessels communicate with the extra-
fascicular vessels by numerous anastomoses piercing the perineurial layer.

In the present study the tibial nerve of the rabbit was chosen as an experimental model. This nerve has a well-defined extrinsic supply, mainly concentrated in the areas around the ankle and knee. In the present study the nerve was divided about 2 centimetres proximal to the ankle and the blood vessels were observed at the proximal end close to the section. Such division has proved not to impair the intrafascicular vascular supply, the intraneural micro-
circulation being well preserved even close to the point of section (Lundborg 1970).

When tension is applied to a nerve both the extrinsic and the intrinsic systems are affected. Normally the regional nutrient vessels present a coiled and tortuous appearance (Fig. 3). When the nerve is stretched the coils disappear and the “reserve in length” is lost. Stronger tension stretches the regional vessels and the blood flow in them is obstructed. It was shown by Lundborg that the exclusion of the external regional vascular supply per se had little or no effect on the nutritive intrafascicular microcirculation in the sciatic-tibial nerve of the rabbit, even over a length of 15 centimetres. However, tension of a nerve also reduces the cross-
sectional area within the epineurium, implying—among other things—an increase in intra-
fascicular pressure. In this way the intraneural vessels may be occluded and the nutrition of nerve fibres impaired.

The first vessels to be affected are the venules. With elongation of 5 to 10 per cent there was a slowing down in venular flow, though no reduction of flow could be observed in arterioles and capillaries. At this stage the first intravasal evidence of tissue injury—
granulocytosis, microthrombi and emboli—was observed. It is reasonable to expect also a reduction or stagnation of flow in the epineurial lymph spaces. Accordingly, this degree of stretching—if maintained over a long period as in the case of end-to-end nerve suture—would probably give rise to a continuous impairment of intraneural microvascular flow, jeopardising the nutrition of nerve fibres. This degree is here called the “lower stretching limit” and is of great interest. This limit, however, might still be lower than the degree of tension at which the first recognisable alterations in intraneural morphology could be observed. The “upper stretching limit” when all microcirculation ceases, of 11 to 18 per cent, is already far beyond the real critical limit as far as long-term viability of the nerve is concerned, and is therefore of less importance.

The axons possess a remarkable resistance to ischaemia (Lundborg 1970), but if ischaemia by stretching is maintained over a long period of time definite damage can be expected. The vessels themselves may also suffer from the anoxia, especially when combined with mechanical trauma. Long-lasting stretching may so damage the vessel walls that when the nerve is relaxed and restoration of blood flow taking place, this may result in leakage of proteins and the development of an intrafascicular oedema (Lundborg 1970). Such a distribution of proteins in the endoneurium might bring about the formation of a scar interfering with the healing and restoration of nerve function. In the present study, however, no transvascular passage of proteins into the endoneurium could be observed, not even after half an hour at the “upper stretching limit”. Obviously, the endothelium is highly resistant to this kind of trauma. This might be expected in view of previous findings that the endothelial cells seem to remain impermeable to proteins even after six hours of ischaemia (Lundborg 1970).

Each fascicle is surrounded by perineurium, which seems to be impermeable to proteins even after long periods of ischaemia (Lundborg, Nordborg, Olsson and Rydevik 1972). In the present study no change in this property could be observed even after half an hour at the “upper stretching limit”. This is rather astonishing because the perineurium under these conditions is subject to considerable mechanical forces. The results obtained in this study indicate that the tibial nerve of the rabbit tolerates elongation of about 8 per cent before a definite impairment of the intraneural microvascular flow is noticed. At about 15 per cent of elongation the vessels are totally occluded and the nerve tissue suffers complete ischaemia.
These results are obtained from an ideal experimental situation, the nerve being cut but otherwise undamaged. However, nerve surgery often concerns heavily damaged nerve trunks with the vascularity already impaired. Under such unfavourable conditions the data obtained from this study may of course be misleading, the “lower stretching limit” being still lower.

Following relaxation of the nerve after traction at the “upper stretching limit” the intraneural circulation recovered well. For some time there was granulocytosis, with numerous microthrombi and emboli. The arterioles and capillaries regained their flow immediately but the venules took five to ten minutes. In these experiments stretching lasted half an hour. This period must be of great importance as regards the restitution of flow. When the deforming force is gradually applied and just enough to obliterate the vessels, it could probably be maintained for several hours without causing irreversible damage to the blood vessels or to the nerve tissue itself, but only when the “upper stretching limit” is not high enough to damage essential non-vascular structures of the nerve trunk. According to Lundborg (1970) ischaemia per se can be maintained for four to six hours and still be followed by a good return of blood flow and nerve function. However, he also showed that the addition of mechanical deformation led to considerably greater damage to the nerve. In the clinical situation a nerve may be sutured under tension that is continuous. Moreover, a blood-free field for an hour or longer might increase the risk of inadequate restitution of blood flow in the already damaged nerve trunk. The conclusion is that the experimental model used here does not correspond to a clinical situation involving a complex injury to a limb. However, the model should be regarded as adequate for analysing basic mechanisms, and various limits are of interest when stretching per se is applied to a peripheral nerve accidentally cut and subjected to partial resection and suture.

One might ask to what extent the figure of around 8 per cent obtained for the “lower stretching limit” is valid for other nerves and for humans. This result was obtained from a rabbit’s nerve, lying free between muscle bundles and cut distally. There was no contact with bone or curving around a joint in the immediate neighbourhood. The experimental situation could so far well correspond to a clinical state. Theoretically, if all peripheral nerves consisted of the same homogeneous substance, the stretching limits might have a certain general applicability. In reality, however, things are far more complicated. A nerve trunk consists of several fascicles, each surrounded by perineurium and embedded in epineurium. Each layer of the nerve possesses different elastic properties. Nerves vary in the proportion between number of fascicles and volume of epineurium, a fact which has been systematically investigated by Sunderland (1968). The architecture of the intraneural vasculature, though generally following the same basic principles, exhibits varying proportions between the three plexuses. Moreover, a larger nerve trunk has larger vessels, capable of resisting compression better than those of smaller nerves.

Obviously it is impossible to define lower and upper stretching limits valid for nerves in general. However, it may prove to be of value to have some basic data obtained from the ideal experimental situation. Together with clinical experience, a knowledge of the basic pathophysiological mechanisms involved in this very special circumstance might contribute to the successful treatment of peripheral nerve injuries by end-to-end suture under some degree of tension.

**SUMMARY**

1. Stretching of the tibial nerve cut 2 centimetres above the ankle has been the subject of an experimental study in rabbits.
2. The effects on intraneural microcirculation, on vascular permeability, and on the barrier function of the perineurium have been analysed with the aim of determining the extent to which a divided nerve can be stretched without interfering with the process of repair.
3. The results obtained may prove valuable for understanding basic mechanisms and for establishing certain important limitations when end-to-end suture of a nerve trunk is performed under some degree of tension in man.
EFFECTS OF STRETCHING THE TIBIAL NERVE OF THE RABBIT

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