Neurosurg. Rev. 4 (1981) 83-94

Introduction

The epineural mobilization of a divided nerve results in a two-fold interruption of its blood supply:

1. of the "extrinsic system" due to division of the regional afferent vessels along the mobilized length of nerve and

2. of the "intrinsic system" due to the transverse injury (12) and the resultant discontinuity.

The "intrinsic system" includes the deep intrafascicular as well as the superficial extrafascicular longitudinal vascular supply.

Mobilization studies on divided nerves have already been published by Smith (20, 21), Lundborg and Brånemark (13), Lundborg (12), Kline et al. (9) and Starkweather et al. (22). They included vital microscopic observations of the circulation in the sciatic nerve vessels of rabbits (13, 12), combined clinical, electrophysiological and histological investigations of monkey nerves (9, 22), and vessel perfusion studies in human corpses immediately post mortem, as well as in amputated limbs (20, 21). The results of these different methods can only provide information on some aspects of the effects of mobilization. The clinical relevance of the devascularisation due to the mobilization of an injured nerve that must be reapproximated by suture depends ultimately and exclusively upon the effect on the functioning and regenerative parenchyma in the proximal nerve segment. On the basis of these results, the following questions proved to be of primary interest and have been the subject of our experiments:

1. What is the critical mobilization length, i. e. the distance to which a nerve may be separated from its bed without resulting ischaemia?

2. What correlation exists between the separated nerve length and its ischaemic degeneration distance when the critical mobilization length is exceeded?

3. What quantitative relationship exists between parenchymal damage and the deficiency of the nerve's vascular system?

A Study on the Critical Mobilization Length of Peripheral Nerves

G. Orf, R. Schultheiss

Department of Neurosurgery (Dir.: Prof. Dr. Dr. R. Wüllenweber), and Institute for Neuropathology (Dir.: Prof. Dr. G. Kersting) of the University of Bonn, FRG

4. How is the revascularisation of the ischaemic nerve segment achieved?

Material and methods

Adult rabbits (14 males, 18 females) of the species "German giant greys" (Body weight $\approx 6.25 \pm 0.95$ kg) were used for the experiments on the sciatic, tibial and peroneal nerve of the left lower limb, i. e. a total of 64 nerves.

Neurotomy was performed without loss of substance by a clean cut with a pair of scissors. The level of the



Fig. 1. Schematic diagram of the arterial blood supply of the sciatic, tibial, and peroneal nerves in the thigh and lower leg.

The arrows indicate the respective positions where the nerves were severed and sutured. The figures indicate the proximal mobilization lengths associated with each neurotomy, as follows: 60 mm = 1; 80 mm = 2; 100 mm = 3; 120 mm = 4; 140 mm = 5; 160 mm = 6; 180 mm = 7; 200 mm = 8.

0344-5607/81/0042-0011 \$ 2.00 Copyright by Walter de Gruyter & Co.

84 G. Orf, R. Schultheiss

specimens examined		Proximal mobilization length		Vastular perfusion (Scriptol*)	Histological investigation	
Tibial nerves (n)	Peroneal nerves (n)	absolute (mm)	relative (%)	Number of nerves (n)	Number of nerves (n)	
	8	60	20.3	3	5	
8		80	27.0	3	5	
	8	100	33.8	3	5	
	8	120	40.5	3	5	
	8	140	47.3	3	5	
8		160	54.1	3	5	
8		180	60.8	3	5	
8		200	67.6	3	5	
32	32		nat dan Artin	24	40	
	64			64		
Т	otal			Total		

77 11	1	D	0 -1 - 1 - 1 -	_ £	T?		4.4
Tapie.	1.	Programme	Schedule	OL	EXI	berimen	us.
	_						

incision varied according to the proposed proximal mobilization length (Fig. 1). Beginning at the site of the neurotomy, epineural mobilization was performed in a proximal and distal direction. This included the circular interruption of all afferent vessels by bipolar microcoagulation or ligation throughout the whole mobilization lenght. In cases of extensive mobilization, the division of lateral branches of the nerves was unavoidable.

The retraction gap resulting from the neurotomy was closed without any tension by an end-to-end suture (Nylon monofil 10×0). In this way one was able to avoid the problems of distinguishing between the effects of tension and those of ischaemia resulting from mobilization. The procedure was carried out under nembutal anaesthesia.

The lengths of nerve mobilized were always assessed as a proportion of the total nerve length, as shown in Table 1. The total length was measured from the point of exit of the spinal nerve roots at the intervertebral foramen L7/S1, distally to where the nerve divided into its terminal branches, the tibial and peroneal nerves (metacarpal base) (29.6 \pm 1.25 cm). This necessitated the careful dissection of the nerve from its bed and the avoidance of any iatrogenic tension at the end of the experiments.

With regard to the aetiological relationship between devascularisation and parenchymal degeneration of the mobilized nerve, vascular perfusion studies and histological examinations were performed (Tab. 1). The main interest was devoted to the proximal nerve segment which plays an important role in the regeneration. The distal mobilization lengths of 10 mm to 130 mm were of only secondary importance. Of the nerves mobilized in a proximal and distal direction, one each was excluded from further evaluation because of a faulty Scriptol perfusion and two because of spreading inflammation. After the mobilization, the animals were allowed a survival period of one and three weeks before proceeding with investigation.

Twice filtered undiluted Scriptol* (Rotring No. 5915, Messr. G. Wagner) was used for vessel visualization. After flushing of the circulatory system with warmed Haemaccel^R, Scriptol was introduced into the vessels by intracardiac injection. Drugs were not administered.

The nerve preparations stained with Indian ink were embedded in celloidin. Their 200 μ thick longitudinal sections were transferred from 70 per cent into 100 per cent alcohol and then kept in xylol until they were completely transparent. The histological preparations were processed as follows: Embedding in paraffin, longitudinal sections of 8 μ , stained according to the methods of Bodian, Klüver-Barrera, Nissl, and Masson-Goldner. The non-mobilized nerves of the opposite side served as controls. They were also examined for possible length shrinkage resulting from fixation and embedding procedures. For celloidin preparations, the shrinkage was 9.5 per cent, for the paraffin preparations 4 per cent of the original length.

The ischaemic and degenerated distances were measured in the mobilized nerves. This was done microscopically and always from the suture line in a proximal and distal direction in the fascicular centre of the nerve or its main fascicle, to the endpoint of the vascular filling defect zone of the parenchymal disintegration area. In the histological sections, we only used the Bodian preprations for this purpose. * Indian ink Statistical methods were used to establish a correlation between the demonstrated ischaemic and degenerated distances and the free mobilization lengths of the nerves. The calculation of correlation and regression coefficients and regression straight line followed the instructions of Weber (24), the testing of the correlation coefficients for significance was performed with the aid of the z-transformation according to Fisher (2). The variability coefficient of Pearson (2) was used for comparison of the data variance of the quantitative histological findings with regard to the nerve vessels and parenchyma.

Results Nerve vessels:

None of the surgical mobilizations and resulting devascularisation caused necrosis of the nerve trunk. The resulting ischaemic defect zones showed two types of appearance which were almost equal in frequency, i. e. a circulatory failure which was solely intrafascicular and one which was a combination of intrafascicular and extrafascicular factors. In the case of pronounced intrafascicular circulatory failure, a transition zone was formed for a distance of 3 mm in the nerve between the normal angioarchitecture distal to the suture and the total block of the endoneural supply. This resulted initially in a less stained demonstration of the nerve vessels (vasa nervorum), which then became patchy and finally was completely lacking (Fig. 2 a-d). The function of the external longitudinal vessel was sometimes still maintained up to the suture line in some cases. At their entry into the nerve fibres, the branches of the extrafascicular arteries were occluded.

In the case of combined circulatory failure, the intrafascicular vascular system showed an abrupt interruption and the extrafascicular system only a short transition zone. In general, the capillary circulation in the mesoneural arcades still adhering to the mobilized nerve was the least disturbed. When an extrafascicular ischaemia was present, this was accompanied by a marked intrafascicular one. An intact deep vascular system was never associated with an ischaemic effect in the superficial system.

The revascularisation of the ischaemic nerve segments originated primarily from the vascular supply in the environment (Fig. 3). But even from epineural longitudinal arteries of the ischaemic nerve of fascicular segments, which were just still perfused newly formed capillaries emerged transversely to the outside into the organizing wound area (Fig. 4). With the restitution of the extrafascicular circulation, the intrafascicular also began to reorganize.



Fig. 2a-d. Transitional zone bordering on intrafascicular ischaemia progressing from proximally towards the suture line of a nerve segment mobilized proximally.

Mobilization length: 200 mm. Tibial nerve. Distance from the suture: 60 to 81 mm. Perfusion with Scriptol indian ink. 1st week. Original magnification $160 \times .$



Fig. 3. Revascularisation.

Anastomosis of blood vessels connecting the mobilized nerve and the neighbouring tissue with perfusion of longitudinal intrafascicular blood vessels.

Mobilization length: 160 mm. Tibial nerve. Distance from the suture: 43 mm proximal. Perfusion with Scriptol indian ink. 1st week. Original magnification $20 \times$.

Quantitatively, the extent of the ischaemic nerve distances proved to be divergent (Fig. 5, Tab. 2). Proximal mobilizations of 60 mm usually showed no loss of vascularisation. With mobilization of greater lengths up to 200 mm, some central nerve stumps similarly showed no ischaemic changes. Distal mobilizations up to 60 mm led to ischaemic damage in one of 14 nerves. According to the calculated correlation coefficient r = 0.51, a positive, although only average correlation exists between the mobilization lengths and ischaemic distances. Its significance level α is 0,01, its confidence range r = 0.12 to r = 0.76 (n = 40). The established regression equations (Fig. 5, 6) which define the ischaemic distance (y) as a function of the mobilized nerve length (x) read:

$$y_1 = 0.246x_1 - 5.9 \text{ (mm)}$$

 $y_2 = 0.246x_2 - 1.99 \text{ (\%)}.$

In absolute figures, the ischaemic as well as the remaining uninjured nerve lengths showed a steady increase as the mobilization length increases. At a mobilization length of 200 mm compared to that of 60 mm, the ischaemic distance increases by 8.7 per cent (Fig. 6). The gradient of the regression straight line, however, proves to be only 50 per cent of that of the degeneration distances (Fig. 6, 10). It follows that with increasing mobilization lengths the ischaemic distances do not increase at the same rate as the degenerative distances.

Nerve tissue:

Proximal mobilizations beyond 60 mm caused ischaemic parenchymal damage. This was identical with secondary Wallerian degeneration. The process of

Table 2. Survey of the Average Results of Proximal Nerve Mobilization.

		Vascular perfusion (Scriptol)				Histological investigation				
Proximal mobiliza- tion length	Number of nerves (n)	Ischaemic segment of mobilization length		Perfused segment of mobilization length		Number of nerves	Degenerated segment of mobilization length		Unaffected segment of mobilization length	
(mm)		absolute (mm)	relative (%)	absolute (mm)	relative (%)	(n)	absolute (mm)	relative (%)	absolute (mm)	relative (%)
60	2	0 ± 0	0± 0	60 ± 0	100 ± 0	5	4± 1	6± 2	56 ± 1	94 ± 2
80	3	17 ± 29	21 ± 36	63 ± 29	79 ± 36	5	21 ± 24	27 ± 30	69 ± 24	73 ± 30
100	3	10 ± 9	10± 9	90 ± 9	90 ± 9	5	8± 9	8± 9	92 ± 9	92 ± 9
120	3	15 ± 21	12 ± 18	105 ± 21	88 ± 18	4	6± 5	5 ± 4	114 ± 5	95 ± 4
140	3	9± 8	6 ± 6	131 ± 8	94± 6	5	19 ± 18	13 ± 13	121 ± 18	87 ± 13
160	3	63 ± 57	39 ± 36	97 ± 57	61 ± 36	5	78 ± 17	49 ± 11	82 ± 17	51 + 11
180	3	34 ± 33	19 ± 18	146 ± 33	81 ± 18	4	80 ± 19	44 ± 11	101 ± 19	56 ± 11
200	3	38±39	19 ± 20	162 ± 39	81 ± 20	5	78 ± 31	39±16	122 ± 31	61 ± 16



Fig. 4. Revascularisation.

Capillaries sprouting laterally from the longitudinal epineurial vessels into the surrounding tissue. Mobilization length: 100 mm. Peroneal nerve. Distance from the suture: 13 mm proximal. Perfusion with Skriptol indian ink. 1st week. Original magnification $40 \times .$

parenchymal disintegration and break-down was more pronounced in the third than in the first week, while the area of degeneration had not increased. The entire distal stump was in any case subject to traumatic degeneration. The proximal ischaemic degeneration area of the tibial and peroneal nerve (Fig. 7a-d) showed the same type of extension and always had a cone-like termination within the mobilization segment. The proximal nerve segment degenerated completely from the suture line to the cone base. Several intact subperineural neurites were seen only in the area of the cone base. Above the cone base, the degeneration area was successively restricted from the exterior nerve zone to the nerve centre (Fig. 8). The cone apex with some single axons with regressive changes in the nerve centre was directed centripetally. It was noted that the more distant from the suture the larger was the degeneration.

As a direct result of the neurotomy, a less frequent degenerative expansion area – usually 2 mm to 4 mm

above the neurorrhaphy – showed a relatively abrupt break-off.

In addition, the mobilized central nerve stumps demonstrate, morphologically, diffuse and focal signs of an "ischaemic collagenization". This fibrosis, due to traumatic secondary Wallerian degeneration and associated shrinkage of the sheath tissue, was even more pronounced in the distal nerve segments. The quantitative analysis of the proximal degenerative distances in relation to the lengths mobilization is summarized in Fig. 9 and Tab. 2.

The correlation coefficient r = 0.76 confirms a considerable relation between degeneration distances and mobilization lengths. The significance level α has a value of 0.01. The confidence range varies between r = 0.5 and r = 0.9 (n = 38). The reliability which allows one to deduce the resulting average degeneration distance y of the nerve from the mobilization length x is sufficiently great. The respective regression straight lines have the equations:



Fig. 5. Scattergram and regression line marking the relationship between the length of ischaemic nerve segment and the proximal and distal mobilization length.

1 measurements proximal \bullet and distal \bigcirc

- 2 measurements proximal \blacktriangle and distal \triangle
- 3 measurements proximal and distal

* % of mean total nerve length.

$$y_1 = 0.60x_1 - 41.6 \text{ (mm)}$$

 $y_2 = 0.60x_2 - 14.05 \text{ (\%)}.$

The data of the degeneration distances did not vary to the same extent as the ischaemic distances in the vascular perfusion studies. This is confirmed by the variability coefficients according to Pearson (2), as their mean value of 63.7 ± 42.1 is definitely lower compared to that of 111.2 ± 32.7 .

The gradient of the regression straight line is twice as great as that of the ischaemic distance. With increasing mobilization length, extent and rate of increase of the degeneration distance evidently exceed that of the ischaemic distance (Fig. 6, 10). With a nerve exposure of 200 mm it reaches almost twice the ischaemic distance. With a mobilization length of 80 mm to 200 mm, it increases by 31.5 per cent. Compared to the ischaemic one, the degenerative distance is increased up to 77 per cent, the intact mobilized nerve segment decreased up to 22 per cent, which corresponds to a total of up to 17 per cent of the total length mobilized.

Based on the established morphological findings and their statistical evaluation, the nerve mobilization results in a critical length at 70 mm, i. e. 24 per cent of the total nerve length, beyond which ischaemic parenchymal damages occur. Beyond a freely mobilized length of 140 mm, i. e. 47 per cent of the total nerve length, a degeneration distance of at least 40 mm, i. e. 14 per cent of the total nerve length, will develop in any case.

Discussion

In mobilized nerves, the parenchymal lesion represents an ischaemic effect and shows a greater extent



Fig. 6. Diagram and regression line of the ischaemic nerve segment. The bars indicate the mobilization length; the lengths of the ischaemic segments to be expected are indicated below, those of the segments of unaffected vascularisation are indicated above the straight line.

* % of mean total nerve length.

on average than the perfusion failure distance. Thus, ischaemic degeneration also occurs in the transition zone with its impaired circulation. This is already manifested during the first week. A residual circulation at the margin of the irrigation area is apparently not sufficient to guarantee a maintainance supply to the parenchyma.

A transition zone with stagnation of the circulation distal to the neurotomy or suture site has also been observed microscopically intra vitam by Lundborg (12). Three zones of intravascular filling – a normal zone distal to the suture, a transition zone, and a

filling defect close to the suture – were also confirmed by Smith (20, 21). The already apparent failure of the intrafascicular vascular system proximally as compared to the circulation usually continuing in the extrafascicular blood vessels is explained by an endoneural oedema. According to Ducker et al. (5) it can persist for one week or longer. Due to the associated increase of the intrafascicular pressure, the vessels entering the endoneural space in an oblique and transverse direction show a break off due to the compression and occlusion.

Complete compensation of the "extrinsic system" by



d

Fig. 7a-d. Ischaemic degeneration of the neural parenchyma spreading quantitatively and gradually towards the suture from proximally.

Mobilization length: 200 mm. Tibial nerve. Distance from the suture: 110 to 140 mm proximal. Staining of axons by Bodian's method. 3rd week. Original magnification 128. the "intrinsic system" is still guaranteed at a mobilization length up to 24 per cent of the total nerve length. When this limit is exceeded, only partial compensation is possible distal to the suture above the transition zone. In the mobilization segment close to the suture, compensation of the extrinsic by the intrinsic supply system and vice versa is no longer possible. For the mobilized nerves, this implies a dependence of the deep nerve vessels upon the superficial blood vessels, which confirms the experimental results of Durward (6). Even with extensive mobilization, the missing ischaemic effect apparently depends upon individual variations in the blood supply. Circulatory and nerve tissue failure patterns which are sometimes seen after the isolated interruption of nutritional arteries (1, 3, 4) could not be found.

That the restitution of the intraneural circulation originates from two particular points has also been reported by Tarlov and Epstein (23), Hiller (8), Nobel and Black (14), as well as by Nobel et al. (15).

With regard to neurosurgical practice, the failure of the circulation in the proximal nerve stump which determines the regeneration, is of less importance than the consecutive ischaemic parenchymal degeneration. It is found as a cone-shaped field in chronic nerve extension (16) as well as after any traction on the nerve vessels. The disintegration distance of the nerve depends upon the free mobilization length. An exposure beyond 70 mm, i. e. 24 per cent of the total nerve length always results in ischaemic degeneration. This figure represents the critical mobilization length for the proximal nerve segment. Further mobilization results in an analogous and more extensive disintegration distance. Thus, the margin of the parenchymal in the proximal nerve stump is being shifted centripetally closer to the trophic cell centres in the anterior horn or spinal ganglia. This results in an increase of the nerve segment to be regenerated and the prognosis for clinical recovery becomes more unfavourable. In any case, the speed of regeneration is decreased in nerve segments with impaired nutrition (19, 25). An additional barrier to regeneration is the "ischaemic collagenisation" (10, 11, 22). Even excessive mobilization lengths of 200 mm, i. e. 68 per cent of the total nerve length, however, never led to the nerve's functioning as a free graft. Nevertheless mobilization will remain justified as a "manipulative" procedure to obtain a tension-free end-to-end suture of injured nerves as long as 24 per cent of the total nerve length, i. e. the critical length, is not exceeded. With the aid of the regression equations (p. 7) and straight lines in the diagram in Fig. 9 and 10, and the known mobilization length x, the average degenera-



Fig. 8. Central degeneration field.

Intact nerve fibres in the periphery of the fascicle. Mobilization length: 80 mm. Tibial nerve. Distance from the suture: 18 mm proximal. 3rd week. Original magnification 64.

tion ,,distance y" can be predicted with fair reliability. The percentage of an individual nerve length can be determined in patients in absolute figures by indirect measurements (17, 18). Further experiments are necessary in order to determine whether there is a uniform, relative critical mobilization length for nerves in each extremity. The permissible or critical

Summary

The transverse section of the sciatic, tibial and peroneal nerves in rabbits was followed by mobilization in a proximal and distal direction and a tension-free end-to-end suture of the cut surfaces. The proximal mobilization was performed up to eight different levels between 60 mm and 200 mm, i. e. 20.3% to 67.7% of the total nerve length. Histological findings and their statistical analysis indicated that the critical mobilization length, beyond which ischaemic parenchymal damages occur, is 70 mm or 24% of the total nerve length. Up to this level, the vascular

mobilization lengths for nerves in the upper extremities and for nerves of other species are still to be determined. As regards the degree of tension tolerated by an end-to-end suture, a comparison remains to be made between the use of mobilized and non – mobilized nerves.

"extrinsic system" of the mobilized nerve segments is completely compensated for by the "intrinsic system". When the mobilization length is increased, the degeneration distances were more pronounced than those of the ischaemic nerve distances. Some nerves showed no effects from the ischaemia.

Key words:

Nerve mobilization – Critical mobilization length – Nerve devascularisation – Nerve ischaemia.

92 G. Orf, R. Schultheiss



Fig. 9. Scattergram and regression line marking the relationship between the length of degenerated segment and the proximal mobilization length.

1 measurement

2 measurements

- 3 measurements
- * % of mean total nerve length.

Zusammenfassung

An den Nn. ischiadici, tibiales und peronaei von Kaninchen wurden nach transversaler Durchtrennung proximo-distale Mobilisationen vorgenommen und anschließend die freien Stümpfe spannungslos End-zu-End genäht. Die ascendierende Mobilisation erfolgte in 8 verschiedenen Dimensionen zwischen 60 mm und 220 mm, das sind 20,3% bis 67,7% der Nervengesamtlänge. Die kritische Mobilisationslänge eines Nerven, oberhalb welcher ischämische Parenchymschäden auftreten, wurde nach den histologischen Befunden und deren statistischer Auswertung bei 70 mm, entsprechend 24% der Nervengesamtlänge, gefunden. Bis dahin wiesen die mobilisierten Nervensegmente eine vollständige Kompensation der ausgeschalteten äußeren Blutzuflüsse durch das innere Gefäßsystem auf. Mit zunehmender Mobilisationslänge stiegen die Degenerationsstrekken stärker als die ischämischen Nervenstrecken an. Einzelne Nerven blieben ohne Ischämieeffekte.

Schlüsselwörter:

Nervenmobilisation – Kritische Mobilisationslänge – Nervendevascularisation – Ischämie peripherer Nerven.



Fig. 10. Diagram and regression line of the degenerated nerve segments. The bars indicate the mobilization lengths; the lengths of the degenerated segments to be expected are indicated below, the lengths of the intact segments of parenchyma are indicated above the straight line.

* % mean total nerve length.

References

1. Adams, W. E.: The blood supply of nerves. II. The effects of exclusion of its regional sources of supply on the sciatic nerve of the rabbit. J. Anat. (Lond.) 77 (1943).

2. Clauss, G., H. Ebner: Grundlagen der Statistik für Psychologen, Pädagogen und Soziologen. Deutsch, Frankfurt-Zürich 1972.

3. Denny-Brown, D., C. Brenner: Paralysis of nerve induced by direct pressure and by tourniquet. Arch. Neurol. Psychiat. (Chic.) 51 (1944) 1–26.

4. Denny-Brown, D., C. Brenner: Lesion in peripheral

nerve resulting from compression by spring clip. Arch. Neurol. Psychiat. (Chic.) 52 (1944) 1-19.

5. Ducker, M. B., L. G. Kempe, G. J. Hayes: The metabolic background for peripheral nerve surgery. J. Neurosurg. 30 (1969) 270–280.

6. Durward, A.: The blood supply of nerves. Postgrad. med. J. 24 (1948) 11–14.

7. Glees, P., W. J. H. Nauta: A critical review of studies on axonal and terminal degeneration. Mschr. Psychiat. Neurol. 129 (1955) 74–91. 8. Hiller, F.: Nerve regeneration in grafts. J. Neuropath. clin. Neurol. 1 (1951) 5–25.

9. Kline, D. G., E. R. Hackett, G. D. Davis, M. B. Myers: Effect of mobilization on the blood supply and regeneration of injured nerves. J. surg. Res. 12 (1972) 254–266.

10. Krücke, W.: Erkrankungen der peripheren Nerven. In: Lubarsch, O., F. Henke, R. Rössle: Handbuch der speziellen pathologischen Anatomie und Histologie. Vol. XIII/5, pp. 1–248. Springer, Berlin-Göttingen-Heidelberg 1955.

11. Krücke, W.: Pathologie der peripheren Nerven. In: Olivecrona, H., W. Tönnis, W. Krenkel: Handbuch der Neurochirurgie. Vol. VII/3, pp. 1–267. Springer, Berlin-Heidelberg-New York 1974.

12. Lundborg, G.: Ischaemic Nerve Injury. Scand. J. Plast. Reconstr. Surg., Suppl. 6 (1970).

13. Lundborg, G., P.-I. Brånemark: Microvascular structure and function of peripheral nerves. Vital microscopic studies of the tibial nerve in the rabbit. Adv. Microcirc. 1 (1968) 66–88.

14. Nobel, W., D. Black: The microcirculation of peripheral nerves: Techniques for perfusion and microangiographic, macrophotographic, and photomicrographic recordings in animals. J. Neurosurg. 41 (1974) 83–91.

15. Nobel, W., D. Black, P. Johnson, C. Kase: Effect of chronic compression on the microcirculation and function of peripheral nerves. In: Cervós-Navarro, J.: Pathology of cerebral microcirculation. Proc. Int. Symposium, Berlin, Sept. 1973. Walter de Gruyter & Co., Berlin-New York 1974.

16. Orf, G.: Critical resection length and gap distance in peripheral nerves. Acta neurochir. (Wien), Suppl. 26 (1978).

17. Orf, G., H. D. Gerhards: Correlation between External Body Dimension and the Length of Peripheral Nerves. Z. Morphol. Anthropol. 71 (1980) 259–267.

18. Orf, G., H. D. Gerhards: Determining the Length of Peripheral Nerves Indirectly by Regression Analysis. Z. Morphol. Anthropol. 1981 (in press).

19. Sanders, F. K., J. Z. Young: The degeneration and re-innervation of grafted nerves. J. Anat. (Lond.) 76 (1942) 143–166.

20. Smith, J. W.: Factors influencing nerve repair. Arch. Surg. 93 (1966) 335–341.

21. Smith, J. W.: Factors influencing nerve repair. II. Collateral circulation of peripheral nerves. Arch. Surg. 93 (1966) 433–437.

22. Starkweather, R. J., R. J. Neviaser, J. P. Adams, D. B. Parsons: The effect of devascularisation on the regeneration of lacerated peripheral nerves. An experimental study. J. Hand Surg. 3 (1978) 163–167.

23. Tarlov, I. M., J. A. Epstein: Nerve grafts: The importance of an adequate blood supply. J. Neurosurg. 2 (1945) 49–71.

24. Weber, E.: Grundriß der biologischen Statistik. Fischer, Jena 1961.

25. Weddell, G.: Axonal regeneration in cutaneous nerve plexuses. J. Anat. (Lond.) 77 (1942) 49-62.

PD. Dr. G. Orf, Neurochirurgische Universitäts-Klinik, Sigmund-Freud-Straße 25, D-5300 Bonn-Venusberg.