Efferent and afferent fibres in human sacral ventral nerve roots: basic research and clinical implications [1]

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Abstract

Single extracellular action potentials have been recorded at 2 sites from human S4 ventral nerve roots, and their amplitude, duration and conduction time measured. Conduction velocity frequency distribution histograms have been constructed. Three classes of α-motoneurons could be identified, which had mean conduction velocity values of 61.5 (α_1), 49 (α_2) and 37.5 m/sec (α_3) for a young adult at about 37 °C. The conduction velocity of single motor fibres has been correlated with its action potential amplitude and duration. The action potential amplitude increased and the duration decreased with the conduction velocity. Touch-stimulated and other afferences have been identified in these motor roots. The fastest afferents had about the same conduction velocity as the α_1-motoneurons and the touch-stimulated afferents had conduction velocities of between 20 and 41 m/sec at about 34 °C. Also the amplitude of the afferent single unit potentials increased and the duration decreased with the conduction velocity.

The electrophysiologically measured roots have been removed and morphologically analysed with the light and electron microscope. Nerve fibre diameter frequency distribution histograms have been constructed with respect to 4 myelin sheath thickness ranges. In the diameter histograms 3 α-motoneuron peaks with mean values of about 12.5 (α_1), 10.3 (α_2) and 8.3 μm (α_3) and 1 peak of touch stimulated afferences with a mean value of 11.2 μm could be identified for myelin sheath thicknesses between 1.8 and 2.3 μm. A teased fibre dissection gave a factor of 100 between the internode length and the nerve fibre diameter.

The electrophysiologic parameters have been correlated with the morphologic parameters. Approximate factors between the mean conduction velocities and the mean nerve fibre diameters of the α-motoneuron classes were 5.1 (α_1), 4.85 (α_2) and 4.4 m/sec/μm (α_3) at about 37 °C. Comparable approximate conversion factors for group I and fastest touch-stimulated ventral root afferents were 4.5 (gr. I) and 3.5 m/sec/μm (touch).

By comparing the number of nerve fibres of each class of motoneurons with the number of spontaneously occurring action potentials, it was found that the α_3-motoneurons, most likely supplying the slow fatigue resistant muscle fibres, had the highest activation at rest.

The existance of ventral root afferents has been discussed with respect to pain treatments by deafferentation and ventral root stimulation to improve the bladder function in paraplegia. The nerve fibre composition of the S4 ventral roots and the identification of afferent functions can be used to improve the efficiency of nerve anastomoses from intercostal nerves to the sacral nerve roots in lower spinal cord lesions. By measuring the α- and γ-motoneuron activity in ventral roots a specific intraoperative diagnosis of the function of these neuron cell bodies in the spinal cord is possible.

Key-words: Human — single unit potentials — conduction velocities — ventral root afferents — nerve fibre diameters — restorative neurology.

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Introduction

To treate lower spinal cord lesions by nerve anastomoses (12, 93) or to improve the results of sacral ventral nerve root stimulation for the control bladder function in paraplegia (9), detailed morphologic and electrophysiologic knowledge of the human sacral nerve roots is needed. For the understanding and treatment of deafferentation pain it would be desirable to know for definite wether there are afferent fibres in the human ventral roots (22, 33, 83, 90), since they exist in the cat (13, 40, 65) and dog (74).

Nerve fibres have been counted in nerve roots. For literature see Ref. 66 and 92. Some anatomical aspects are known about the lower human spinal canal (12, 20, 49, 66). Substantial knowledge about the innervation of the urinary bladder is available for animals, but these data are mostly not discussed in respect to human validity and applicability. From sacral nerve blockages and rhizotomies there exist indirect knowledge about the innervation of the human bladder (2, 20, 29, 49, 51, 61, 94, 103).

In this paper, an attempt will be made to start to analyse the nerve fibre composition of human lower sacral ventral nerve roots to offer human data to improve treatments like the ones listed above. By recording spontaneous efferent single unit potentials (67) from ventral sacral nerve roots and calculating conduction velocity distribution histograms, the electrophysiologic composition of active nerve fibres with large single unit potential amplitudes of some sacral ventral roots will be obtained. By constructing nerve fibre diameter distribution histograms from cross-sections of the electrophysiologically measured nerve roots the morphologic composition of myelinated nerve fibres of the same roots is obtained. Comparing the mean conduction velocity values of different peaks with the mean fibre diameter values of different peaks, 3 α-motoneuron classes can be identified. The analysis of the thick nerve fibres of the lower ventral sacral roots is complicated by the finding that ventral root afferent also exist in humans and alter more or less the efferent nerve fibre distribution.

Clinical material and method

Measurements were collected from 1 human cadaver, 3 brain dead human cadavers (HT's) and one patient (intraoperative diagnosis). There was no known neurological disease. It was attempted to keep the blood pressure up by administration of Dopamin according to those standards used in kidney removals. Elevated brain pressure, subarachnoidal bleeding, disturbance of coagulation and consumptive coagulopathy complicated the trials.

The measurements were done in accordance with the Declaration of Helsinki, on the same principle as in kidney removals (to reconstract urinary tract function) and according to medical tradition, to take information from human cadavers for the benefit of patients (see clinical implications).

Electrophysiology and electrodiagnosis

Extracellular action potentials were recorded from nerve roots or subdivisions of roots (92) (called root filaments) from the HT's and the one patient (Pat) with 2 platinum wire electrode pairs (electrode distance in each pair 4 mm) at 2 sites, preamplified and displayed on a digital storage oscilloscope (Vuko Vks 22-16) as shown in Figure 1 (67). For the HT3, the recordings were stored (5, 26) with a PCM-processor (Digital Audio Processor PCM-501ES) and a video recorder (JVC-Kassettenrecorder, Modell-Nr. Hr-D250EG) (Fig. 1). Conduction velocities were calculated from the conduction time

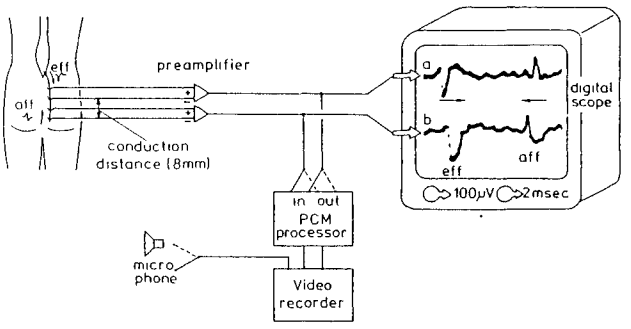

Fig. 1. — Arrangements for recording extracellular action potentials with 2 electrode pairs at 2 sites. aff = afference, eff = efference.

(Fig. 2) and conduction distance (Fig. 1). Action potential amplitude and duration were measured according to Figure 3B, C (inset) and Figure 5C for efferent potentials from trace *a* and for afferent potentials from trace *b*.

For an intraoperative diagnosis with the single unit potential recording method about 1/2 an hour is needed. The pure recording time varies between 0.5 and 10 minutes, depending on how many functions are measured on how many roots. Reversible pressure symptoms of the nerve fibres as alteration of the action potential wave form or partial conduction block between the electrode pairs of some fibres increase with the time the root is lying on the electrodes; a rest from pressure symptoms may have to be given every few minutes to the root, by removing it from the electrodes. The temperature of the root changes with increasing measuring time from those of the fluid in the spinal canal. Recordings *a* and *b* (Table 1) of the HT3 have been performed at the beginning (a) and at the end of the measurement (b) after about 10 minutes with removing the root from the electrodes once in between. The conduction velocity values changed with the temperature; the relative features were stable.

Morphology and morphometry

Root pieces of a few cm were removed from the HT's after recording the single unit potentials and from the cadaver (Cad), fixated in 4% glutaraldehyd in cacodylate buffer, after-fixated in 1% OsO_4 for 2 hours and dehydrated and embedded in Araldite according to standard techniques. Pictures were made from semi-thin sections stained with thionin and acridine orange (75) with the light microscope ($\times$ 1000). Nerve fibre diameters $\varnothing = 1/2(\varnothing_1 + \varnothing_2)$ ($\varnothing_1$ and $\varnothing_2$ the larger and smaller diameter of non-roundshaped fibres) and the mean myelin sheath thickness d were measured by hand (Figs. 8, 9). A shrinkage correction of 8% (19) was taken into account. The measured nerve fibre diameters were divided up in 4 myelin sheath thickness classes ($d = 0.25-0.75\,\mu m$, $d = 0.8-1.25\,\mu m$, $d = 1.3-1.75\,\mu m$, $d = 1.8-2.3\,\mu m$, d-values were measured in steps of $0.05\,\mu m$ so that there are no gaps between the d-ranges) and for each myelin sheath thickness range a nerve fibre diameter distribution histogram was constructed. The sum of the 4 distributions gives the usual diameter distribution histogram. A second root piece from the HT1 was embedded in soft Araldite and was used for a teased fibre dissection. Internode length and the diameter of the nerve fibres were measured under a Wild M8 stereomicroscope ($\times$ 100). Thin sections were viewed under the electron microscope, $\times$ 2000 and $\times$ 4000 pictures made and pictures of the whole cross section (Figs. 6, 7) constructed. The electron micrographs were only used for the control of the light microscope pictures, not for the actual measurements.

Results

Measurements were done on one human cadaver, 3 brain dead human cadavers (HT's) and one patient. On the cadaver only nerve fibre diameter spectra could be determined. Diameter and conduction velocity spectra were determined and correlated for the 3 HT's. Only conduction velocity spectra could be obtained from the intraoperative measurement.

Electrophysiology

Single efferent extracellular action potentials

Efferent extracellular single unit potentials were recorded at 2 sites from ventral roots. The recording arrangements are shown in Figure 1.

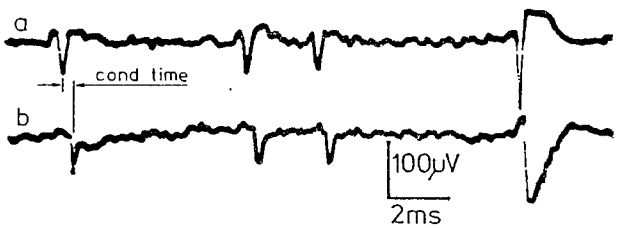

Fig. 2. — Typical sweep piece of a recording of efferent single unit potentials from the HT1. The largest action potential on trace *b* has a prolonged duration, compared to its corresponding potential on trace *a*, due to mechanical alteration.

The sacral ventral roots were identified from their position in the cauda equina (66), their ventral rootlets from the spinal cord and their thicknesses (66).

A typical piece of a recorded 800 msec long sweep from the HT1 is shown in Figure 2. The whole sweep was quantitatively analysed with respect to conduction velocity (= conduction distance/conduction time), amplitude and duration of the spontaneously occurring action potentials. Only those action potentials were used, which did not show double or deformed action potential form; some potentials travelling in the opposite direction were also not taken into consideration (32, 37, 46, 59, 62, 63, 80, 81, 88, 89) (see also section ventral root afferents).

The calculated conduction velocities were plotted in a histogram in Figure 3A in 2 ways. Firstly, all conduction velocity values were counted (open plus hatched histogram). This way of counting ensures that all spontaneously firing efferent fibres were taken into account; repeated firing fibres will have been counted several times according to their frequency of firing. Secondly, each velocity value was used only once (hatched histogram). This counting ensures that repeatedly firing nerve fibres were counted only once, but different nerve fibres conducting at the same velocity will have been left out. The true situation, that the conduction velocity of each spontaneously active nerve fibre is counted only once, lies between the 2 ways of counting shown in Figure 3A. But since conduction velocities of nerve fibres are seldom exactly the same, the plotted conduction velocity histogram "each value taken only once" (hatched) will have a similar characteristic shape to the real conduction velocity spectrum of spontaneously active nerve fibres.

Three peaks of the most regularly occurring conduction velocities can be identified in Figure 3A. Mean conduction velocity values of 22.5, 32.5 and 50 m/sec are obtained for the HT1. Another conduction velocity histogram of efferent nerve fibres, showing the 3-peak distribution (HT3), is shown in Figure 5A. The values for all measured cases are summarized in Table 1 (section correlation between electrophysiology and morphology).

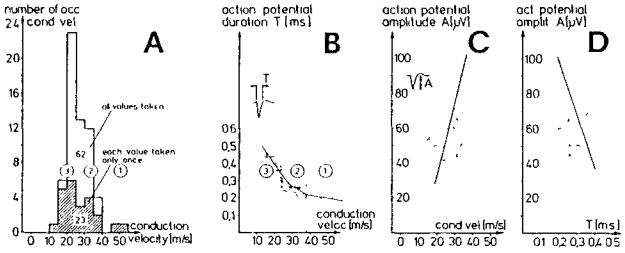

Fig. 3. — Analysis of a 800 ms recording of efferent action potentials from the HT1 (s = sec).

A. Conduction velocity histogram. Hatched part, each conduction velocity value taken only once; open plus hatched part, all values are taken. Numbers mark peaks.

B. Duration of action potentials in relation to their conduction velocities. Correlation line drawn by eye. Long dashed line parts indicate possible overlapping of these lines. Short dashed vertical lines mark possible borders of classes of fibres.

C. Amplitude of action potentials in relation to their conduction velocities. Correlation line drawn by eye.

D. Amplitude of action potentials in relation to their durations. Line drawn by eye.

In Figure 3B the velocity of conduction of all single action potentials were plotted against their duration (HT1). 3 straight lines with different steepnesses were fitted by eye into these correlation points. Figure 3B shows that the action potential duration T decreases with the rising conduction velocity and it seems that each peak of conduction velocities in Figure 3A, representing a certain group of nerve fibres, has its own linear correlation between conduction velocity and action potential duration.

Figure 3C shows the relation between action potential amplitude and conduction velocity. The correlation is poor. In parts of another sweep the correlation between amplitude and conduction velocity was better, whereas the correlation between duration and velocity was not so good. The correlation between the action potential amplitude and duration in Figure 3D is also poor. This poor correlation only reflects in another way the scatter of data between conduction velocity and amplitude. This high scatter will partly be due to pressure or damage, not visible in the action potential form, reducing the amplitude and increasing the duration of the action potentials (81, 88) (for an obvious deformation see Figure 2), since the root was lying

for more than 1/2 an hour on the recording electrodes and no paraffin oil was used (see also discussion).

Ventral root afferents

During the recording of efferent activity from the ventral roots of the 3 HT's and the patient, single unit potentials were observed, which travelled in the opposite direction like afferent ones (67). The first explanation of these fast-conducted action potentials was that they were reflected efferent potentials. But these afferent-like single unit potentials occurred quite often and the distribution of their conduction velocities had no similarity with the 3 peak distribution of the α-motoneurons; Buchthal and Rosenfalk (10) measured quite similar conduction velocities in humans for orthodromic and antidromic transmission in sensory fibres of the arm. Artificial potentials caused by trigger points in the process of root drying could mainly be excluded since their activity increases quite quickly, but this was not the case. The identification that most of these afferent-like single unit potentials were potentials from afferent fibres in ventral roots was done by activity increase measurements.

Figure 4a shows an afferent single unit potential alongside two efferent ones (HT3). The

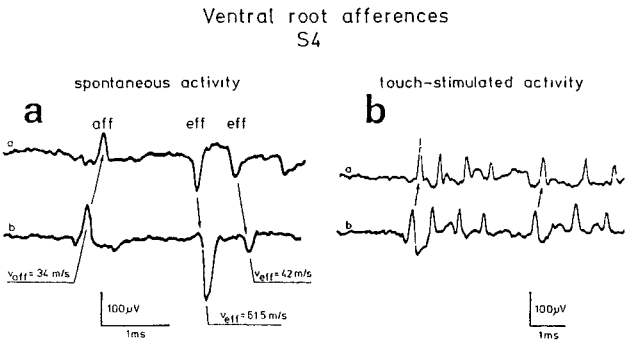

Fig. 4. — Ventral root afferents of the S4 ventral root from the HT3.

a. Registration of afferent and efferent action potentials, calculated conduction velocities are indicated. aff = afferent, eff = efferent.

b. Recording of touch-stimulated afferent action potentials. Left (no potentials), before activity increase. Moment of the touching of the skin is not indicated. Arrows mark two corresponding action potentials.

afferent single unit potential is conducted more slowly than the 2 efferent potentials. Figure 4b shows the activity increase of afferent fibres in a human ventral root due to touching the sacral dermatoms. The burst of afferent single unit potentials lasted for about 80 msec and the range of conduction velocities was between 20 and 41 m/sec at about 34 °C (see Fig. 5 and Table 1). The number of fibres responding to touch were 16 ("each conduction velocity value being taken only once"). The decreased efferent activity during the afferent activity in Figure 4b could have been evoked by a preceding touch of the skin, which resulted in a reflex pause of efferent activity (96), but other reasons are possible. Figure 5B shows the conduction velocity histograms of the ventral root afferents. Not all afferents are stimulated by the touching of the skin. The fastest recorded afferent fibre was not stimulated by touch and had about the same conduction velocity as the fastest efferent fibre (Fig. 5A). In another sweep, not shown here,

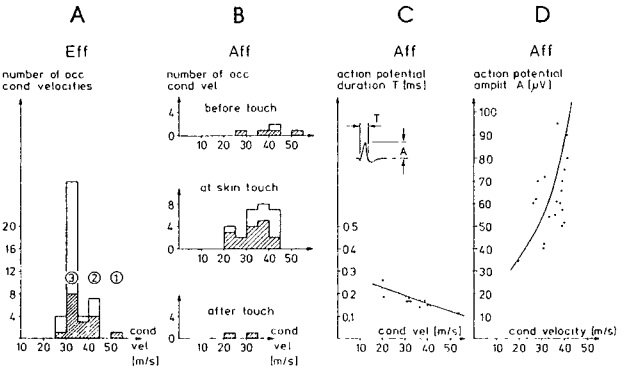

Fig. 5. — Analysis of two 800 ms recordings of afferent and efferent action potentials of the sacral ventral root from the HT3.

A. Conduction velocity histogram of spontaneously active efferents. Hatched part, each conduction velocity value taken only once; open plus hatched part, all values are taken. Numbers mark peaks.

B. Conduction velocity histograms of ventral root afferents before touch (spontaneous activity), at or shortly after touch and after touching the skin, when the stimulated activity was over. Hatched and open histogram parts as in A.

C. Duration of afferent action potentials in relation to their conduction velocities. Correlation line drawn by eye.

D. Amplitude of afferent action potentials in relation to their conduction velocities. Correlation line drawn by eye.

two afferents could be found with conduction velocities between 50 and 55 m/sec. The conduction velocity distribution of the touch stimulated afferents shows 2 peaks (Fig. 5B). These peak values are different to the ones of the efferent fibres of the same root (Fig. 5A). Peak conduction velocity values of all measured cases are summarized in Table 1 (section correlation between electrophysiology and morphology). The action potential duration of the touch-stimulated afferents and the ones not stimulated by touch (propably group I afferents) decrease with the conduction velocity as is shown in Figure 5C. Even though the scatter is large, the action potential amplitudes show an increase with the conduction velocity, as is shown in Figure 5D.

A mechanical stimulation of the bladder in the same HT (HT3) showed no afferent activity increase of measurable amplitude; only the efferent activity increased.

Morphology and Morphometry

After the electrophysiological measurements root pieces of the 3 HT's were fixated and embedded for light and electron microscopy.

Nerve fibre diameter spectra

To correlate nerve fibre diameters with conduction velocities, the nerve fibre diameters and the myelin sheath thicknesses were measured by hand from the light microscope cross-sections partly shown in Figure 8b and Figure 9b and similar to the electron microscope cross-sections of Figure 6 and Figure 7. In the histograms of the root from the HT1, 4 peaks of most often occurring myelinated nerve fibre diameters were mainly observed. 3 were seen in the myelin sheath thickness range between 1.8 and 2.3 μm and one large peak was observed in the range $d = 0.25 - 0.75\,\mu$m (Fig. 8a). In the histograms

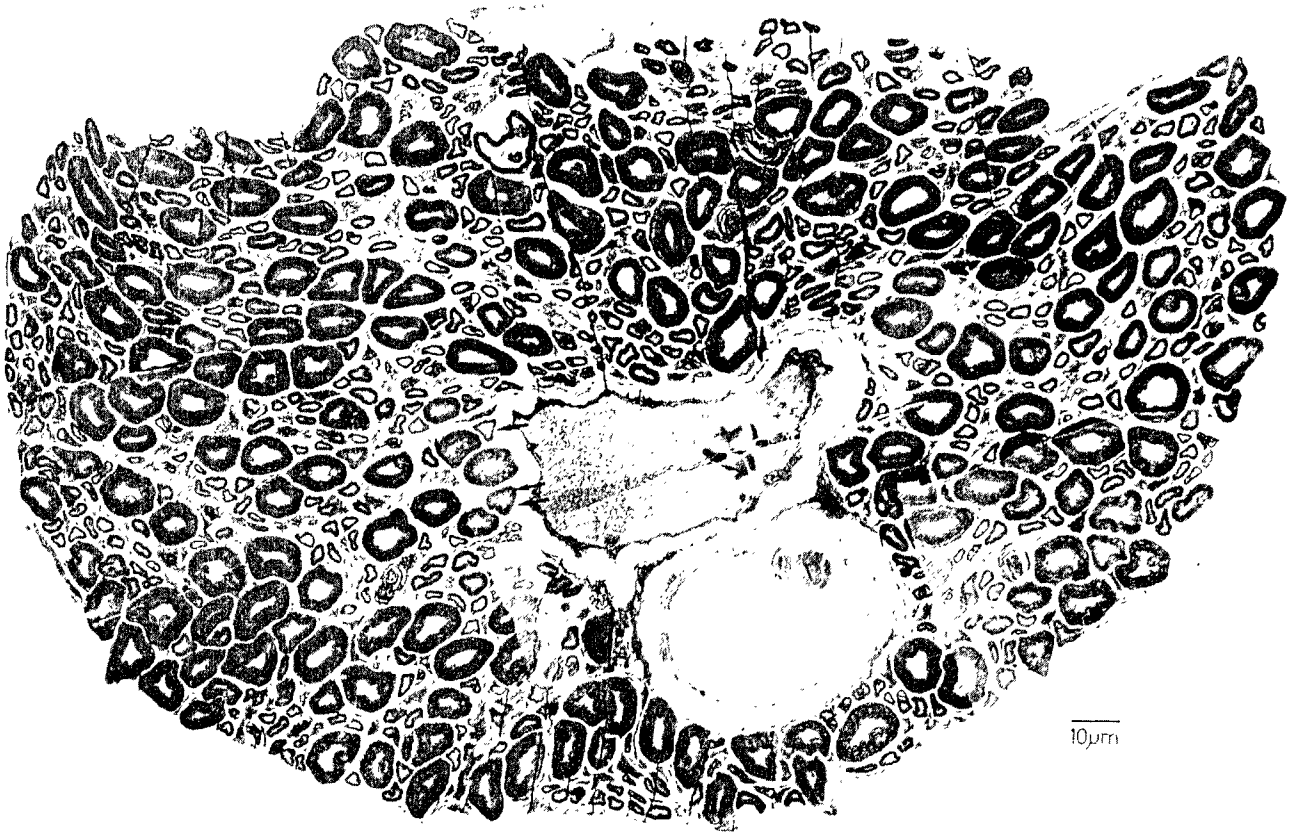

Fig. 6. — Montage of several electron microscope photographs of the S4 ventral nerve root from the HT1. Single arrow marks a nerve fibre with a comparably thin myelin sheath; double arrow marks a nerve fibre of same size with a comparable thick myelin sheath. Scale not corrected for shrinkage.

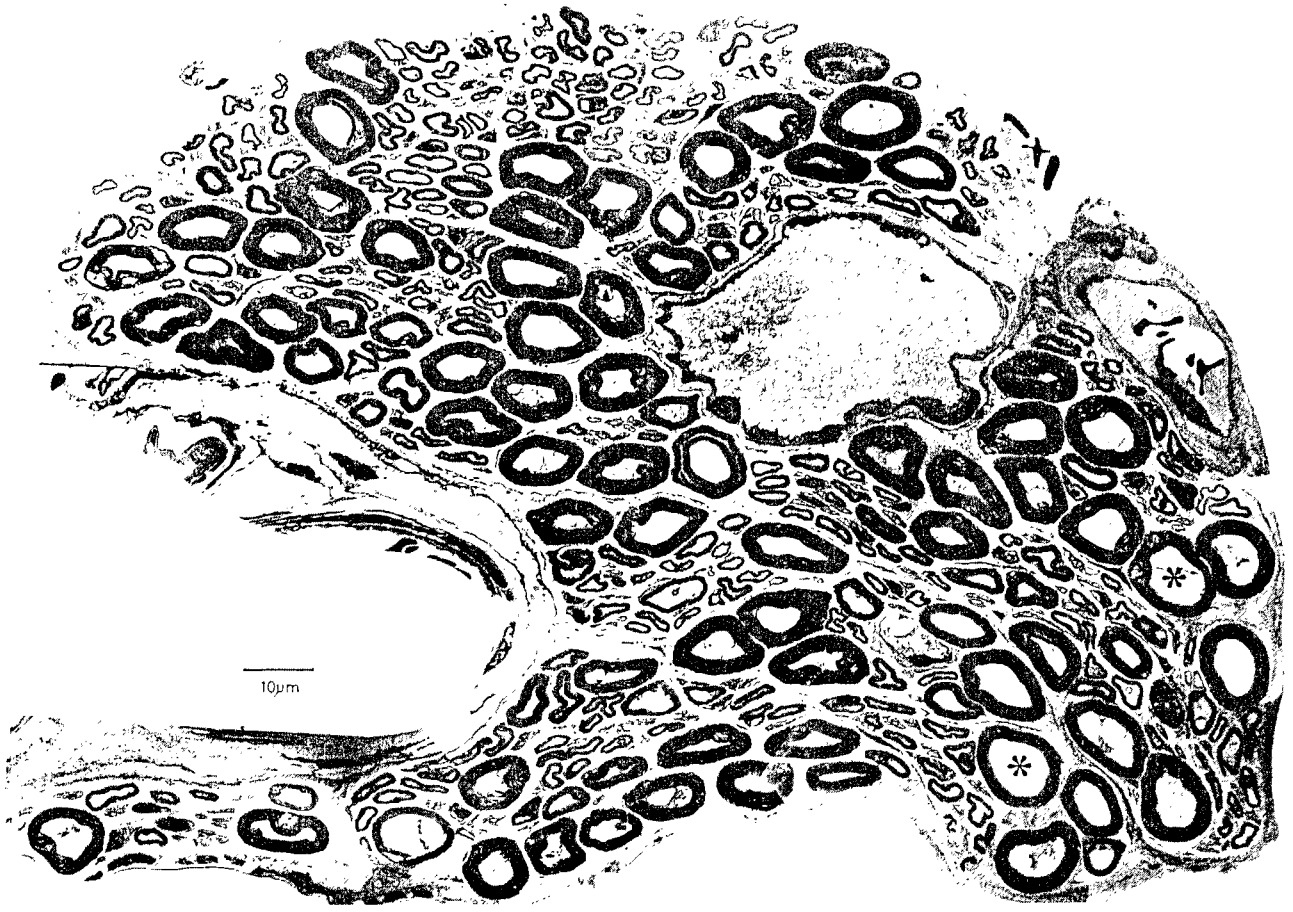

Fig. 7. — Montage of several electron microscope photographs of the ventral root (S4 or S5) from the HT3. Note, 2 of the thickest nerve fibres marked with an asterisk have a comparably thin myelin sheath. Scale not corrected for shrinkage. The largest blood vessel of the root is not fully shown.

from the roots of the HT2 and HT3, 2 additional peaks were recognized in the range $d = 1.8-2.3\,\mu m$ (Table 1).

To gain some understanding of these peaks, the nerve fibre diameter histograms of Figure 8 (HT1) have been compared with the histograms of an L4 ventral root of another human cadaver (Fig. 9), since at that level there is no sympathetic or parasympathetic nervous outflow through the root filaments, and, according to first electrophysiologic experience, there may be only a few thick afferents in the ventral roots. The large peak No. 4 of the $d = 0.25-0.75\,\mu m$ myelin sheath thickness range of the S4 ventral root (Fig. 8) is almost completely missing in the L4 histograms (Fig. 9) and therefore the peak No. 4 of Figure 8 will mainly represent the parasympathetic nervous outflow through the S4 ventral root. By comparing the peak No. 1 of Figure 8 with that of Figure 9, one can see that the peak No. 1 of the S4 ventral root contains, with 1% (about 5 fibres), only few fibres, whereas in the L4 ventral root 57% of all myelinated fibres are in that peak. The nerve fibres of peak No. 1 most likely represent the largest α-motoneurons and their percentage is large in the L4 ventral root (supplying skeletal muscle with mainly "phasic" functions) and is small in the S4 ventral root (supplying muscles with mainly postural functions (95)).

Teased fibre dissection (HT1)

With normal effort, 130 (Fig. 10B) of the 570 myelinated fibres (Fig. 8) were teased and, additionally, the diameters of 95 of these were mea-

sured (Fig. 10D). As the similar shape of the diameter histogram of the teased fibre dissection (Fig. 10D) in comparison to that of Figure 8 indicates, a somewhat representive selection was teased. In order that several internodal lengths could be determined, as much of a length of a fibre as possible was teased.

Figure 10A shows a montage of 3 photographed teased single fibres of different thicknesses. Several internodal lengths are measured and marked. It can be seen that the internodal lengths are not always similar. It seemed that every 3rd to 5th was markedly different to the other ones. On 3 thick fibres it was found that the longer internode corresponded with a thicker nerve fibre as is shown in Figure 10A, fibre 1, so that the ratio of internodal length to nerve fibre diameter (Fig. 10C) was not changed. Figure 10B shows the frequency distribution histogram of the internodal length. This distribution

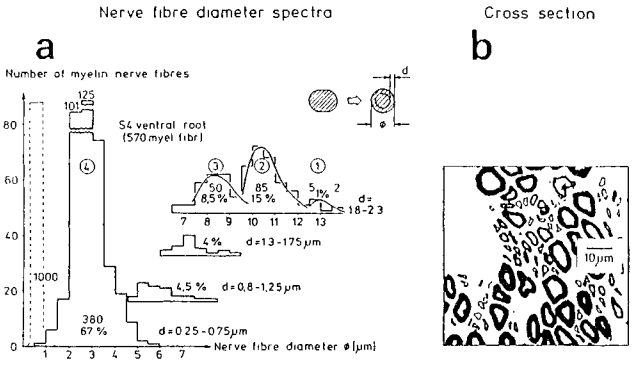

Fig. 8.

a. Nerve fibre diameter frequency distribution histograms from the S4 ventral root of the HT1, measured from the light microscope cross section partly shown in b. The 4 histograms are marked with the myelin sheath thickness range of the nerve fibres which are contained in it. All 570 myelinaed fibres were measured. Dashed line peak gives the nerve fibre diameter spectrum of about 1000 unmyelinated fibres (from a part of Fig. 6 in a higher magnification). The approximate drawn distribution curves (not gaussian) indicate more clearly the peaks in the diameter spectrum for $d = 1.8-2.3\,\mu m$. The approximate number of fibres contained in a peak is written into the peaks and also their percentage of all myelinated fibres.

b. Characteristic cross sectional area of the root, contrasted with the copying-machine Minolta 450 zoom.

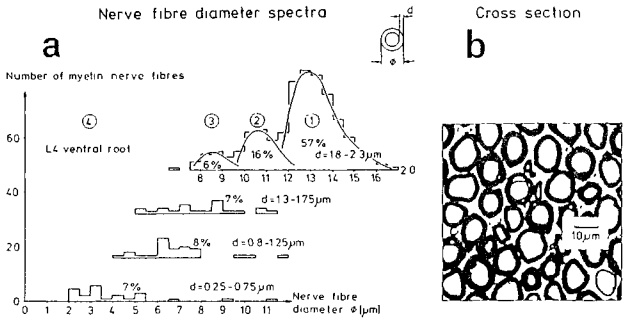

Fig. 9.

a. Nerve fibre diameter frequency distribution histograms of an L4 ventral root from a 47-year-old female human cadaver, removed 2-5 hours after death. The myelin sheath thickness ranges are the same as in Figure 8. 317 fibres were measured.

b. Part of the light microscopical root cross section from which the nerve fibres had been measured, contrasted with the copying-machine Minolta 450 zoom.

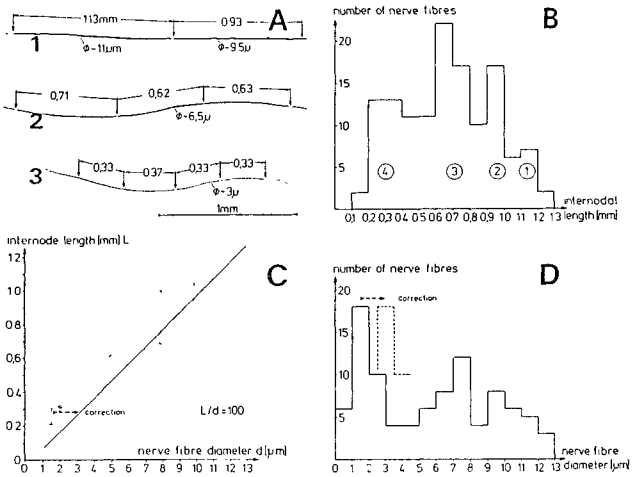

Fig. 10. — Quantified results of the teased fibre dissection of the whole S4 ventral root from the HT1. No shrinkage correction.

A. 3 original nerve fibre dissections, several internodes are measured and marked. Approximate fibre diameter (∅) are indicated.

B. Frequency distribution histogram of internodal lenghts. Numbers indicate peaks. Class borders are $<$ and $\leq$. If more than one internodal length of a nerve fibre was measured (30% of the cases) then the mean value was used.

C. Internodal length in relation to its nerve fibre diameter. A correction for small nerve fibres was performed (see D).

D. Very approximate nerve fibre diameter frequency distribution. Since the diameter of the nerve fibres were measured under the stereo-microscope with a $\times 100$ magnification, the small nerve fibres were estimated more than measured. By comparing this diameter frequency distribution with that of Figure 8 a correction for small nerve fibre diameters was performed and indicated in the Figure.

also shows 4 characteristic peaks, as the frequency distribution of the nerve fibre diameters does (Fig. 8). This similarity can be understood since the nerve fibre selection of the teased fibre dissection of Figure 10D is somewhat representative and the relation between internode length L and fibre diameter d is proportional (Fig. 10C). By dividing the internodal length in Figure 10B by $L/d = 100$ one gets a somewhat similar histogram as in Figure 8 (if the 4 histograms are added).

Figure 10C shows the relation between internodal length and nerve fibre diameter. The relation factor L/d is very near to 100. Figure 10D shows the nerve fibre diameter frequency distribution histogram of the teased fibres. The histogram is somewhat similar to that of Figure 8; peak No. 1 cannot clearly be seen.

Correlation between electrophysiology and morphology

Summarized mean conduction velocities and mean nerve fibre diameters

Table 1 summarizes the mean values of conduction velocity peaks of conduction velocity distribution histograms and the mean values of nerve fiber diameter peaks of the corresponding nerve fibre diameter distribution histograms from all measured cases. The way of correlating the 3 peaks of the efferent conduction velocity frequency distribution histograms with the peaks 1, 2 and 3 of the motor nerve fibre diameter spectra in Table 1 is most likely justified (85, 95, 97, 98, 99), since the action potential amplitude increases with the conduction velocity and the nerve fibre diameter in a first approximation (Fig. 3C and Fig. 5D) (23, 58, 68). Also, peak 1 of the motor nerve fibre diameter spectra represents the thickest fibres and the peak with the highest mean conduction velocity of Figure 3A and Figure 5A most likely represents the peak of fastest conducting fibres, since these values agree with the values of the literature (4, 16, 38, 82).

The mean conduction velocity values of the 3 peaks of efferent nerve fibres are designated with α_1, α_2 and α_3. From Table 1, it can be seen that the mean conduction velocity values of these different α-motoneuron populations are measured consistently. Their actual values seemed to depend strongly on the temperature during the measurement and on the age of the human. The exact temperature of the root, from what has been recorded, is not known. Only the central temperature and the temperature of the central fluid, mixed with 0.9% NaCl washing solution and blood at the measuring place, was measured. In one case (HT3) the operational field was heated with "warm light". The conduction velocity measurements of the HT3 (V_{α_1} between 54 m/sec and 61.5 m/sec) may be nearest to the conduction velocity values of healthy young humans since the α_1-motoneuron value is most near to the values obtained with the evoked potential method (4, 16) (see discussion). The values of the afferent conduction velocities in Table 1 are from touch stimulated afferents (the fastest ones numbered with "1") and spontaneously active afferents (53 m/sec for the HT3).

The values of the mean nerve fibre diameters of the 3 α-motoneuron classes from the measured cases in Table 1 are quite similar. The intermingled ventral root afferents occurring possibly in different cases with different percentages in the ventral roots, may have shifted the mean values of the peaks a little. The afferent nerve fibre diameter peaks of Table 1 (gr. I and touch 1) are probably from afferent fibres, since these peaks were not consistently occurring, were missing in the L4 histogram, where maybe only a few ventral root afferences are present and had values (touch 1) which one would expect for afferent skin fibres (69).

Conversion factors from the conduction velocities to the nerve fibre diameters

From the mean conduction velocities and the mean nerve fibre diameters conversion factors can be calculated for the 3 α-motoneuron classes. The conversion factors for the HT3 are: measurement a: 3.7 m/sec/μm (α_3), 4.0 (α_2) and 4.5 (α_1) (temperature of the nerve root around 34 °C); measurement b: 4.4 m/sec/μm (α_3), 4.85 (α_2) and 5.1 (α_1) (temperature more almost

Table 1. — *Mean conduction velocity values of peaks of most regularly occurring conduction velocities taken from conduction velocity histograms like that of Figure 3A and Figures 5A, B, and mean nerve fibre diameter values of peaks of most regularly occurring diameters taken from nerve fibre diameter histograms like those of Figure 8, and Figure 9 of 1 cadaver (Cad), 3 brain dead human cadavers (HT's) and one patient (Pat). The values in the brackets give approximate numbers of occurring conduction velocities (each value taken only once) contained in the peaks and approximate numbers of nerve fibres contained in the peaks. Values from efferent fibres are in the upper part of the table; a_1, a_2 and a_3 designates different α-motoneuron classes. For explanation of γ-motoneurons see the text. Values from afferent fibres are in the lower part of the table; gr. I designates group I afferences and touch 1, 2 and 3 designate probably different classes of touch stimulated afferences. 8.1^x and 9.1^x are mean diameter values taken from a skin nerve branch of Ref. 69. f = female, m = male. a, b, two measurements with different temperatures for the HT3. centr. = central, d = dorsal, v = ventral, S = sacral, L = lumbal. The diameters of the roots are only approximate. 8% shrinkage correction for the nerve fibre diameters. All nerve fibres in the diameter peaks had myelin sheet thicknesses between 1.8 and 2.3 μm.*

		Sex	age	Mean conduction velocity [m/sec]					Temperature [°C] in centr. spinal fluid		Mean diameter [μm]				Ventral root	Root diameter [mm]
				γ	a_3	a_2	a_1				a_3	a_2	a_1			
Cad	efferent	f	47		—	—	—		—	—	8.5	10.5	12.7		L4	part of root
HT1	efferent	f	32	—	23 (14)	32 (7)	50 (2)		32°	—	8.0 (50)	10.3 (85)	12.7 (5)		S4	0.3
HT2	efferent	m	55	—	20 (10)	27 (6)	39 (2)		32°	32.5°	8.3 (40)	10.3 (120)	12.5 (70)		S4 or S4+S5	0.25
HT3	efferent	f	21	—	31.5 (11)	40 (5)	54 (1)	a	33.5°	34° (heated)	8.5 (12)	10.1 (34)	12.1 (6)		S5 or S4	0.15
				—	37.5 (7)	49 (6)	61.5 (1)	b	33.5°	> 34° (heated)						
Pat	efferent	m	43	16 (2)	24 (6)	30 (5)	—		37°	34°	—	—	—		S3 or S4 d or v+d	0.3
HT1	afferent	f	32						32°	—				13.7 (2)	S4	0.3
HT2	afferent	m	55		20.5 (1)	29 (1)	38 (2)		32°	32.5°			11.4 (30)	14.5 (2)	S4 or S4+S5	0.25
HT3	afferent	f	21		21 (4)	36 (12)	53 (1)		33.5°	34°		9.1^x	11.2 (12)	13 (2)	S5 or S4	0.15
Pat	afferent	m	43	17 (4)	24 (9)	33.5 (20)	47.5 (2)		37°	34°	8.1^x		—	—	S3 or S4 d or v+d	0.3
				3 touch	2 stimulated		1 gr. I				3	2	1 touch	gr. I		

37 °C). The exact values of the conversion factors depend quite strongly on the measuring temperature of the conduction velocity, the age of the human and the shrinkage of the nerve fibres with the fixation procedure and is therefore not known. But safe is the conclusion for the α-motoneurons, that the different α-motoneurons have different conversion factors, and the thinner α-motoneurons with slower conduction velocities have on average a smaller value for the conversion from the conduction velocity to the nerve fibre diameter.

A safe correlation between conduction velocities and nerve fibre diameters for the ventral root afferents is not possible, since not all ventral root afferents are known and it is not possible to get diameter peak values of very small afferent fibre populations on top of the motoneuron distributions. But maybe one can correlate the fastest afferent conduction velocities (maybe from the central branch of group I afferents) with the small peak of thickest nerve fibres. Then the group I afferences would have a slightly larger nerve fibre diameter and a slightly smaller conduction velocity than the α_1-motoneurons. Their conversion factor would be, at 4.1 m/sec/μm smaller than the corresponding value of 4.5 of the α_1-motoneurons (34 °C). The smaller conversion factor originates in the smaller conduction velocity and the smaller conduction velocity could originate in a thinner myelin sheath. If one looks into Figure 7 one can indeed find about 2 very large fibres with a comparable thin myelin sheath. But this is only a suggestion, since, firstly, it has not been proven that the fastest afferents are really group I afferents, secondly, the normal variation in myelin sheath thicknesses may be so large that such conclusions are not possible and, thirdly, it has been reported that very large motoneurons in the cat also have a comparable thin myelin sheath (101), even though in that report there is doubt about the completeness of de-afferentation and de-efferentation by a dorsal or ventral rhizotomy. There is also indication that one can relate the prominent peak of touch afferences (36 m/sec for the HT3) with the other afferent diameter peak of Table 1 (11.2 μm for the HT3 (see discussion ventral root afferents)). These most often occurring touch afferents will then have a conversion factor of 3.2 m/sec/μm at about 34 °C. Using a temperature correction factor of 3.3 %/ °C (10, 38, 57) for the conduction velocities, very approximate conversion factors for the group I afferents and the fastest measured touch afferents can be obtained at about 37 °C (4.5 m/sec/μm (group I) and 3.5 m/sec/μm (touch 1)).

Number of nerve fibres from which has been recorded (HT1)

As can be seen from the nerve fibre diameter spectra of Figure 8, this human S4 ventral root (Fig. 6) consisted of about 1000 unmyelinated fibres (a representative 1/5th of the cross-section had 214) and 570 myelinated fibres. The number of nerve fibres in peaks 1, 2 and 3 was 140. The number of determined conduction velocities lay according to Figure 3A between 23 and 62. Therefore, from the 1570 nerve fibres of the human S4 ventral root, electrical activity was recorded from only about 40 fibres of peaks 1, 2 and 3 (approximate value between 23 and 62), which had been spontaneously active during the recording time of 800 msec. Due to the correlation of the electrophysiologically identified peaks of the frequency distribution histogram of conduction velocities with the morphologically identified peaks of the frequency distribution histograms of nerve fibre diameters, these 40 fibres belonged to the approximately 140 nerve fibres of the peaks 1, 2 and 3.

With an electrode distance of 4 mm in each pair and mean internodal lengths of the peaks of 1.2 mm, 0.95 mm and 0.7 mm (Fig. 10B), about 3, 4 and 6 internodes of mean nerve fibres of each group lay between the recording electrodes.

Different activation of α-motoneurons

If one compares the number of α_1-, α_2- and α_3-motoneurons (diameter column, numbers in brackets) with the number of occurring action potentials from the α_1-, α_2- and α_3-motoneurons (conduction velocity column, numbers in brackets) of Table 1, then one recognizes, that the

class of α_3-motoneurons has the highest number of single unit potentials ("each value taken only once" and also "all values taken"), but only the second highest amount of nerve fibres. This can also be seen if one compares the size of the peaks of Nos. 2 and 3 of Figure 3A and Figure 5A with those of Figure 8a. This means that the α_3-motoneurons have the highest spontaneous activation. Since they supply the slow, fatigue-resistant (S) muscle fibres, these muscle fibres seem to get the highest activation at rest or with a little stimulation.

The recruitment of motoneurons and the muscle fibres they innervate, therefore, has also to be seen with respect to the different functions they have and not only with respect to the motoneuron size as described by the "size principle" (95).

Discussion

Electrophysiology

Extracellular action potential form

A statistical evaluation of the single unit potential recordings from the ventral nerve roots was possible, since the electrical activities were low enough, that the single unit potentials only sometimes merged. Those action potentials, which seem to have original, not changed, wave forms were used for the statistical evaluation. Since damage and pressure blocks conduction (62, 91), reduces the amplitude, increases the duration and changes single peaked action potentials into double or triple peaked action potentials (59, 62, 63, 80, 81, 88, 89), multiple peaked action potentials with obviously deformed wave forms were not taken into account. Small alterations from the original wave form will have escaped detection and will have given rise to a larger scatter of the correlation points in Figure 3B,C,D and Figure 5C,D.

Apart from changes due to mechanical alterations during the measurements, some changes of the original action potential wave forms will have occurred, since not all nerve fibres in the nerve roots were lying close to the recording electrodes. Action potentials generated further away from the recording electrodes will have decreased in amplitude, increased in duration and changed in wave form (more volume conductance (15, 38, 85)).

But even in the ideal recording situation, where all nerve fibres would be lying close to the recording electrodes, the action potential wave form may vary a bit along the nerve fibre, according to the different situation of the node of Ranvier in respect to the recording electrodes (35, 60, 76). Only 3 internodes lie between the electrodes of a pair of electrodes for the nerve fibres contained in peak 1 of Figure 8 (mean internode length = 1.2 mm (Fig. 10B); electrode distance = 4 mm). Furthermore, the measurement of several internode lengths from one fibre indicated that every 3rd to 5th internode length was quite different to the other ones (Fig. 10A) and also the nerve fibre diameter was then different to the other ones. Therefore, one cannot exclude some changes in the action potential form from differences of the internodes, even though the nerve fibre could have partly compensated for such differences by a different internodal cleft or a different channel density. But before discussing changes of the action potential wave form due to the saltatoric conduction in myelinated nerve fibres, the mechanical alterations have to be excluded because they seem to be the main cause of changes in the action potential wave form.

In spite of these sources of varying the wave forms of extracellular single unit potentials, a statistical evaluation of the single unit potential wave form seems useful as Figure 3B and Figure 5C,D show.

Conduction velocities

The conduction velocity frequency distribution histograms of Figure 3A and Figure 5A show 3 peaks in the hatched histograms where 2 peaks seem to overlap. If the fibres in these peaks represent classes of nerve fibres (3 classes of α-motoneurons (see below)), then these classes, characterized by a mean conduction velocity and a range of conduction velocities, overlap.

A direct comparison of the mean values of

conduction velocities of peak 1, 2 and 3 with clinical values, ranging from 40 to 74 m/sec is not possible. The conduction velocity determination with compound action potentials promotes substantial numbers of fibres and the fastest fibres. Measurements on single fibres may not be done on a representative selection. Conduction velocities of fastest conducting fibres of the more recent literature are 56.5 m/sec (maximum sensory velocity) (4) and 59.2 ± 3.3 m/sec in 20 young adults (16) for temperatures between 34 °C and 36 °C. The measurement of the HT3 with 54 m/sec (measurement a) and 61,5 m/sec (measurement b) for α_1-motoneurons (Table 1) with root temperatures between 34 °C and 37 °C are quite compatible with those values.

By stimulating the posterior tibial nerve at the ankle, Desmedt & Cheron (16) stimulated α-motoneurons antidromically and group I (from muscle spindles and tendons) and skin afferents orthodromically as fastest fibres and measured a mean conduction velocity of 59.2 m/sec. The skin afferents can most likely be excluded as the fastest conducting fibres because the touch stimulated skin afferents of the HT3 in Figure 5 had highest conduction velocities of 41 m/sec (mean of the faster peak 36 m/sec). In the patient, highest conduction velocities of 39 m/sec (mean of the fast peak 33.5 m/sec) of touch stimulated afferents were measured (notice lower measuring temperature). A direct comparison of the α_1-motoneuron conduction velocities of the HT3 with the 59.2 m/sec value is still not possible because it is not known which nerve fibres conduct faster in the nerve roots the α_1-motoneurons or the group I afferents. Behse and Buchthal (4) measured that the sensory velocity was 3 to 6 m/sec faster than the motor in accordance with cat data (100). But they measured in the peripheral nerves. In the spinal canal it could be that the motor fibres conduct sligthly faster because of being thicker and the afferents conduct slightly slower, since the central branch of the myelinated afferents may be a bit different to the peripheral branch, even though they are reported to be of same diameter in the cat (31). Also the electrical properties of the surrounding tissue will not be exactly the same. In the cat, there are also afferents from muscle spindles in ventral roots (see discussion ventral root afferents). It may be, that they also exist in humans. In the measurement of the HT2, the HT3 and the patient, the fastest conducting afferents (38 m/sec in the HT2, 53 in the HT3 and 47.5 in the patient) were not activated by the touching of the skin (see Fig. 5B). It is therefore likely that these measured conduction velocities belonged to the afferents from the group I.

The actual conduction velocity values were, at 38 m/sec (HT2) and 53 m/sec (HT3), slightly smaller than the velocity values of the α_1-motoneurons namely 39 m/sec and 54 m/sec respectively. Because small technical errors may have entered this comparison and only a few α_1-motoneurons and fast conducting afferents have been measured it is safer to conclude that these group I afferents had about the same conduction velocity as the average α_1-motoneurons.

For a discussion of the γ-motoneurons and the afferent conduction velocities see section γ-efferents and section ventral root afferents respectively.

Systematic errors of the conduction velocity values may arise from the measurements of conduction time and distance and from the problem of measuring the precise temperature of the root. To calibrate the conduction velocity spectra obtained with the single unit potential recording method, with the more accurate values obtained by the evoked potential method, one would need exact temperature measurements for the clinical conduction velocity values, if the temperature differs from the central temperature. Corrections of the conduction velocity because of differences in the measuring temperature, can be done with a correction factor of 2.1 m/sec/ °C (10) (~3.7 %/ °C), 3 %/ °C (38) or 3.5 %/ °C (57).

Action potential amplitude and duration in dependence on the conduction velocity

The action potential duration of α-motoneurons decreases with rising conduction velocity (Fig. 3B). Paintal (58) fitted a round curve through such correlation points, giving rise to the suggestion that the action potential duration

decreases continuously with rising conduction velocity. A round curve could have been fitted here too. But straight lines with different steepnesses were fitted here, since, firstly, the correlation points seem to fit the straight lines better and, secondly, the conduction velocity range of each straight line of Figure 3B seems to match the conduction velocity ranges of the peaks of Figure 3A.

The fitted lines in Figure 3B seem not only to touch each other, but seem to cross each other, indicated for 2 of them by a continuously dashed line. Such a crossing would mean that there are classes of nerve fibres characterized by fixed values "conduction velocity/action potential duration", which overlap. Electrophysiologically, a nerve fibre may be defined more precisely than by its conduction velocity alone. Additional parameters such as duration or amplitude of the action potential or steepness of potential increase may be necessary for a more complete definition.

The action potential duration of the mainly touching afferents of Figure 5C also decreases with rising conduction velocity. Here, a straight line could be fitted through the correlation points.

The action potential amplitude increases with rising conduction velocity for the efferent nerve fibres in Figure 3C and for the afferent fibres in Figure 5D. But the scatter of the correlation points is large. What is unclear, is how much of this scatter originates in the mechanical alteration of nerve fibres, since the nerve fibres in the nerve roots in the spinal canal have nearly no protective tissue (see Fig. 6). For the time being, it seems that the action potential duration of a single nerve fiber shows a better correlation with its conduction velocity than the action potential amplitude. Only differences in the amplitude are more easily recognized for a quick diagnosis during an operation.

Morphology

The nerve fibre diameter frequency distribution histograms of Figure 8 of the S4 ventral root of the HT1 (see Table 1 for all cases) show 3 peaks in the range of myelin sheath thicknesses between 1.8 and 2.3 μm and 1 large peak in the range of myelin sheath thicknesses between 0.25 and 0.75 μm. It seems that there are more than 4 peaks since peak 3 seems to consist of 2 fused peaks. A part of this additional peak may lie in the myelin sheath thickness range between 1.3 and 1.75 μm. There may be another small peak, around 5 to 6 μm, at the foot of peak 4 of parasympathetic nerve fibres, which partly lies in the range of myelin sheath thicknesses between 0.8 and 1.25 μm. For the possible meaning of these peaks, see the discussion about the γ-motoneurons.

Since the analysed roots were ventral ones and the 3 peak diameter distribution is also observed in a muscle nerve branch (69), the peaks 1, 2 and 3 probably represent 3 different classes of α-motoneurons innervating different kinds of extrafusal muscle fibres (85, 95, 97, 98, 99). The α-motoneurons of peak 1 (α_1-motoneurons) could supply the fast, easily-fatigued muscle fibres (FF), whereas peak 2 fibres (α_2-motoneurons) supply the fast, fatigue-resistant muscle fibres (FR) and peak 3 fibres (α_3-motoneurons) the slow, fatigue-resistant (S) muscle fibres (85, 95, 97, 98, 99), similar to the case in the frog (11, 53, 72, 73), where the thick α-motoneurons supply the twitch muscle fibres and the thin α-motoneurons the slow muscle fibres.

The peaks of the motoneurons in the nerve fibre diameter frequency distribution histograms of Figure 8 and Figure 9 overlap, as is indicated by overlapping distribution curves. Such overlapping could mean that each peak represents a class of nerve fibres and different classes of nerve fibres overlap. To histologically differentiate 2 classes of nerve fibres from each other there must be additional parameters for the classification, for example the thickness of the myelin sheath. This view is supported by the observation that the nerve fibre of a certain diameter can have quite a different myelin sheath thickness, as is indicated in Figure 6. Regeneration with transient thin myelin sheaths is not so likely in a normal nerve root. A fixed relationship between the nerve fibre diameter or axon diameter and the myelin sheath thickness may hold only within a certain class of nerve fibres (101).

The relationship value L/d of the internodal length L to the nerve fibre diameter d equals 100 and confirms the values of the literature (21, 34, 47, 58, 84, 101). As is indicated in Figure 10 this relationship L/d = 100 seems to hold even for variations of the internodal length in the same nerve fibre.

Correlation between electrophysiology and morphology

Correlation between electrophysiological and morphological parameters of α-motoneurons

By correlating the mean conduction velocity of a certain peak of conduction velocities with the mean diameter of the most likely corresponding peak of nerve fibre diameters, correlation factors between mean conduction velocities and mean nerve fibre diameters of these groups of nerve fibres could be calculated. Even though a direct comparison to conversion factors of the literature (6, 48, 52, 58, 64, 84, 100) is not possible, since the α-motoneurons are not split up into different classes. somewhat comparable values, possibly representing the fastest α-motoneurons (α_1), are 5.6 m/sec/μm (6, 100) and 5.2 m/sec/μm (52). The conversion factors of the α_1-motoneurons, calculated from the HT3 of measurement a and b are 4.5 and 5.1 m/sec/μm taking 8% shrinkage (19,34) of the nerve fibres into account. It seems, that the conversion factor from the conduction velocity to the nerve fibre diameter for humans is lower than that for mammals originating in a lower conduction velocity (100, 101). It is unclear whether a correlation factor from the conduction velocity to the nerve fibre diameter is the same for all fibres in a group of nerve fibres or is only a mean of different conversion factors within this group. The former case would be more interesting, since such correlation factors would then be one characteristic measure for a class of nerve fibres.

A correlation between the morphologically identified parasympathetic fibres (peak 4 of Fig. 8) with electrophysiologically identified fibres was not possible, since slower conducting fibres could not be reliably identified electrically. The signal to noise and artefact ratio has to be improved further to see the slower conducting fibres with longer action potential duration as well (Ref. 68, Fig. 2A). For other possible reasons see the discussion about γ-efferents.

If further measurements support such a characterisation of nerve fibres by classes, then the correlation should be tried between a set of electrophysiological values (conduction velocity, action potential duration, ...) and a set of morphological values (nerve fibre diameter (axon diameter), myelin sheath thickness, ...).

γ-efferents

In the ventral roots of the 3 HT's no slowly conducted efferent action potentials were recorded (Table 1). They could have escaped detection because of low amplitude and low activity (1). More likely is the explanation that the γ-motoneurons were not active any more because of low blood pressure, since small nerve cells die earlier in spinal asphyxia (24, 25, 27) and there is a big problem, as in kidney removals, to keep the blood pressure to somewhat normal level in HT's. This view is supported by the measurement from the patient (Pat) where 2 more slowly conducting efferents could be identified (16 m/sec). The quality of the recording conditions did not allow the measurement of efferents with even lower action potential amplitudes and slower conduction velocities in the patient. Furthermore, slow efferents (and afferents) have been detected in a dorsal root recording, not documented here. Therefore γ-efferents are most likely to have been detected in the recording from the patient and should be detectable with the single unit potential recording method (67) under good recording conditions.

In the cat at least 2 kinds of γ-efferents have been identified (6). One γ-fibre population was thinly myelinated, had a mean nerve fibre diameter of about 3.5 μm and conducted slowly. The other γ-fibre population was thickly myelinated, had a mean nerve fibre diameter of about 6.5 μm and conducted much faster. The compound action potentials of the γ-efferents showed even more splitting (6).

Two or more kinds of intrafusal motoneurons can be expected in the nerve fibre diameter spectra of Figure 8. Fibres are possibly intermingled in the α_3-motoneuron peak ($d = 1.8-2.3\,\mu$m), in the myelin sheet thickness ranges between 1.3 and 1.75 μm and between 0.8 and 1.25 μm, and at the foot of the parasympathetic peak of nerve fibres. But before analysing the nerve fibre diameter distribution spectra in more detail, consistent electrophysiologic measurements of γ-efferents and possibly β-efferents (7) are needed.

Ventral root afferents

Touch stimulated afferents have been identified in sacral ventral roots (Fig. 5, Table 1). In the HT3 they conducted at velocities between 20 and 41 m/sec (to be compared with measurement a of α-motoneurons) at temperatures between 34 and 37 °C (Fig. 5B). In the cat, afferent nerve fibres in ventral sacral roots, responding to light pressure, have been found to conduct between 15 and 50 m/sec at about 37 °C (65). The measured conduction velocities are therefore compatible with those measured in the cat.

In the HT3, where skin afferents have been best identified, a mechanical stimulation of the urinary bladder was not answered by an activity increase. Only the efferent activity increased. This can be understood by the non-existance of afferents from mechanoreceptors of the bladder wall in this ventral root. But since the mechanical stimulation may have stimulated the avoidance reactions (104) or viscerosomatic reflexes (14) the efferent activity increased.

There are indications that some of the ventral root afferent nerve fibres can also be seen in the nerve fibre diameter distribution histograms and therefore also in Table 1. In the spectra of the HT2 and HT3 for myelin sheath thicknesses between 1.8 and 2.3 μm a strong peak was observed at 11.2 μm between the α_1-motoneurons and α_2-motoneurons (Table 1). In both cases the ventral roots responded, with an activity increase of afferent single unit potentials, to the touching of sacral dermatoms. In the HT1 no stimulation has been done and in the HT2 only the skin stimulation has been performed. A correlation between this fibre diameter peak and the large conduction velocity peak of the touch stimulated afferent fibres (36 m/sec, HT3) is not possible with the data measured here since there may have been other afferents in these ventral roots which have not been stimulated. But in the nerve fibre diameter spectra of a skin nerve branch (69) a peak, containing the majority of thickly myelinated fibres ($d = 1.8-2.5\,\mu$m), was observed with also a mean diameter value of 11.2 μm. It does seem therefore that the diameter peak at 11.2 μm consists mainly of the most offen occurring touching afferents and belongs to the touch-stimulated conduction velocity peak of 36 m/sec (HT3). In the skin nerve branch of Ref. 69 further 2 small diameter peaks of thickly myelinated fibres were observed at 9.1 and 8.2 μm. If one correlates the other 2 touch-stimulated conduction velocities with these 2 nerve fibre diameters as in Table 1 then one obtains the following approximate conversion factors at 34 °C: 4.1 (gr.I), 3.2 (touch 1), 2.3 (touch 2) and 2.1 m/sec/μm (touch 3) (corrected values for 37 °C: 4.5; 3.5; 2.5; 2.3). These conversion factors are lower than in the cat (37 °C: 5.7 (gr.I); 5.6 (skin gr.I) (100)). The main reasons for that difference is, as in the case of the efferents, the lower conduction velocities in humans (53 m/sec at 34 °C in Table 1 and Fig. 5; max. sensory vel. = 56.5 m/sec at 34 till 36 °C (4)) compared to the ones in the cat (max. sensory vel. in a muscle nerve = 94 m/sec and in a skin nerve = 64.5 m/sec at 37 °C (100)). In the ventral roots of the 2 HT's touch 1 and touch 2 afferents were observed and in the patient where it was possibly recorded from a mixed root, the 3 groups were observed. It is unclear to what mechanoreceptors they belong to (Pacinian corpuscle, Meißners corpuscle, Merkel's disks, Ruffini's cylinders (36)). With further measurements on dorsal and ventral roots, including discharge characteristics, this should be possible to clarify. The remaining possible ventral root afferents are expected to be thinner (joint afferences and pain fibres (22, 33, 83, 90)) or thicker (group I afferents) since in the cat sensory fibres in ventral roots have been observed to come

from muscle spindles, skin, joints and bladder and rectal walls (13, 40, 65).

It has been shown quite consistently that 3 classes of α-motoneurons seem to exist (Table 1). The nerve fibre diameter peaks of these motoneurons may be more or less altered by the ventral root afferents depending on how many myelinated ventral root afferents of large diameter are in the ventral roots. Animal experiments suggest that there are not so many (40, 65). The measurements done so far on humans suggest that there may be only a few myelinated afferents in the lumbal and in the thoracical regions, whereas in the sacral range there may be more afferents in the ventral roots. During an operation on patients it can be diagnosed with certainty whether a root is a ventral or a dorsal one (67) in the lumbal range. But in the sacral range the intermingled afferents among the efferents can sometimes make it difficult to decide on the basis of the electrophysiologic recording alone whether a root is a dorsal, a ventral or a mixed one.

Clinical implications

To improve operational techniques in "Restorative Neurology" (rehabilitation) like nerve anastomoses, root stimulation and pain treatments, reliable human data are needed. Extrapolation from one species to another is hazardous, and more so, as the phylogenetic separation increases (17, 41, 42, 55, 69, 70, 77). Assumptions made from commonly used laboratory animals have general applicability, but ultimately must be tested in man, if possible (42).

Pain treatment by deafferentation

Pain fibres in ventral roots have been found in animals (13) and it has been suggested that they also exist in humans (22, 33, 83, 90) and need not necessarily lead through the lateral spinothalamic tract (42). It should be tried to record A_δ pain fibres in ventral roots to see whether rhizotomies (71, 90), including partial ventral rhizotomies, with damage of only the small fibres, give as good results as tractotomies with less risk.

Improvement of bladder function by nerve root stimulation

Urinary bladder function in paraplegia and other disorders can be improved by ventral root stimulation (8, 9). This can, in principle, be achieved by fatiguing the external sphincter and the detrusor at different times (78, 79), or by a specific stimulation of the motor neurons innervating the bladder (8, 9, 11, 45). It has been reported that the striated bladder sphincter is composed of all 3 muscle fible types in the dog (79), of the intermediate type in the cat (50) and in humans of red muscle fibres (S) in Ref. 100 and of fast-twitch muscle fibres (FF) in Ref. 51 (unclear situation). The electrophysiologic and morphologic human data, presented here, suggest that a selective stimulation of α-motoneurons or parasympathetic fibres should be possible. Unwanted reflex activity by also stimulating afferent fibres in ventral roots has to be expected in some patients, since ventral root afferents exist in human.

Nerve anastomoses in paraplegia

The human spinal cord does not regenerate after traumatic injury because the nerve fibres in the CNS grow only for short distances and they establish connections inappropriately (39, 86, 87). Nerve anastomoses (12, 93) from the intercostal nerves to the sacral roots of the cauda equina (PNS) have partly proven to substitute function from proximal the lesion to the urinary bladder in humans (12). An essential improvement of nerve anastomoses seems to be possible if the number of donor and acceptor nerve fibres (92), the mismatching of sensory and motor fibres between donor and acceptor nerves (efficiency), and functional aspects are taken in consideration (69, 70). To avoid dyssynergia of the urinary bladder after regeneration (12, 69, 70), the nerve fibres to the detrusor and the sphincter externus have to be identified by nerve root stimulation (efferents) and single unit potential activity increase measurements (afferents, Fig. 4) and reinnervated with muscle branches of certain functions from the intercostal nerves. The ventral root afferents allow a transposition of functions from the intercostal nerves to ventral roots with efferent and corre-

sponding afferent pathways. But a relearning of functions is only possible for the patient if the interneurons in the for plasticity (102) used spinal cord parts are still functioning (24, 28).

Intraoperative spinal cord diagnosis

Partial damage of the spinal cord may be in such a way that the small spinal cord nerve cells (mainly interneurons) are distroyed, whereas the large nerve cells (α-motoneurons) are still functioning. Such partial damage can occure in spinal asphyxia (24, 27, 28, 30, 43, 44, 54), where the small nerve cells do not get enough oxygen, but the large nerve cells, situated differently, do get enough (43, 44) or possibly in the condition after spinal cord lesions, when nerve cells atrophy in the spinal cord because of lack of activity and trophic influence from other spinal neurons, mainly interneurons (56). An important question in the dysfunction of reflexes in paraplegia is whether the dysfunction of reflexes (3, 18) occurs mainly because many small nerve cells are not functioning any more or because the activity of the γ-motoneurons is too high because of loss of central inhibition. By recording the activity from the small γ-motoneurons information about their functional stage and about small nerve cells can be obtained.

The recordings from the 3 HT's showed probably no γ-efferent activity. It is possible that the γ-motoneurons will not have functioned any more. Some effects from a spinal shock cannot be excluded because a quick brain death is accompanied by a spinal shock, whereas in a slowly-manifestating brain death phylogenetic lower vegetative functions will occure such as coordinated reflexes (104). In the recording from the patient, where there was of course good blood pressure, no spinal shock and no damaged spinal cord, normal γ-efferent activity was probably recorded.

Recording the activity of spinal cord motoneurons is a first step of detailed intraoperative spinal cord diagnosis. A further step would be to record physiologically-stimulated afferent activity (36, 67, 82) (Fig. 4B) and the resulting reflex activity of the spinal cord from ventral and dorsal roots to get further information about the circuitry of the spinal cord (24) and the kinds of motoneurons used to exert the reflexes.

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