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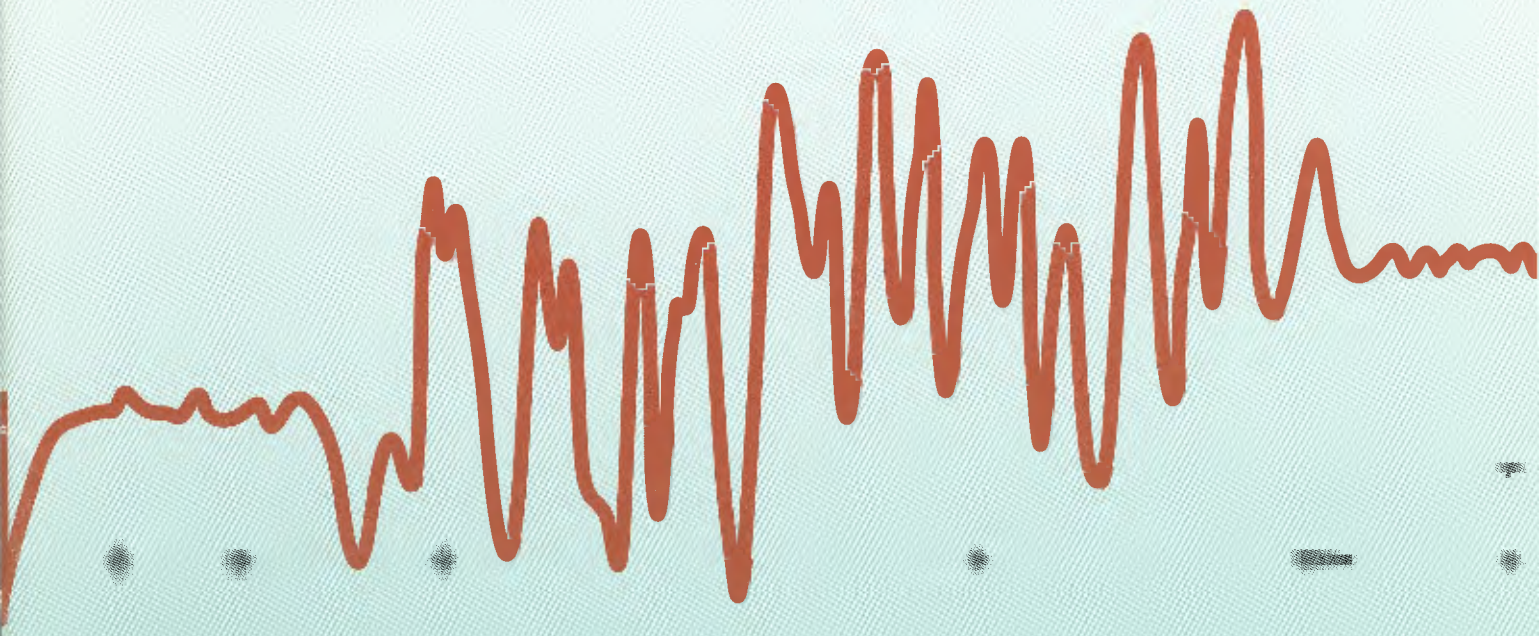

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Sensory Nerve Conduction
Near-Nerve Recording

by

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Abstract

Near-nerve recording of sensory action potentials evoked by electrical and tactile stimuli is a valuable tool in the assessment of nerve pathophysiology. In most instances where percutaneous recording fails to discriminate sensory potentials from noise they can be picked up by needle recording. To localize the site of focal injuries by determination of conduction across the presumed site of lesion, the use of needle electrodes for stimulation and recording is superior to surface electrode technique. In regenerating nerve fibres, sensory potentials can be recorded with needles several months before they can be obtained with surface electrodes. Correlations between histological findings in sural nerve biopsies with sensory potentials from the same nerve have resulted in valuable information as to the contribution of different nerve fibre diameters to conduction velocity, shape and amplitude of sensory potentials in different types of neuropathies.

Key words: *Near-nerve electrode technique, sensory nerve conduction, sensory nerve action potential, focal neuropathies, polyneuropathies, tactile stimulation.*

During the last three decades, investigation of conduction in sensory nerves has been an important integral of the electrodiagnostic armamentarium. The invention of the technique dates back to 1949 when Dawson and Scott published a paper on the recording of nerve action potentials through the skin in man. As Gilliatt (1978) later pointed out, two technical achievements consisting of high-gain, low-noise amplifiers and photographic superimposition developed for investigation of the central nervous system turned out to be very important for future peripheral nerve conduction studies.

Sensory nerve function is usually evaluated in two different ways, either by recording sensory nerve action potentials (SNAP) conducted orthodromically or antidromically, depending on the technique used and the nerve studied. For instance, it is most common to determine orthodromic conduction in the distal segments of median and ulnar nerves using surface electrodes for stimulation and recording (Dawson and Scott 1949, Fullerton 1963, Gilliatt et al. 1965, Oh 1984, Andersen 1985a), but antidromic in radial, dorsal ulnar cutaneous branch, lateral cutaneous femoral, sural, peroneal and tibial nerves (DiBenedetto 1970, Cape 1971, La Fratta and Zalis 1973, Burke and Skuse 1974, Burke et al. 1974, Butler et al. 1974, Guiloff and Sherratt 1977, Schuchmann 1977, Truong et al. 1979, Sarala et al. 1979, Jabre 1980, Critchlow et al. 1980 and Oh 1984). When near-nerve electrode technique is applied, conduction is always studied orthodromically in upper as well as lower extremities (Buchthal and Rosenfalck 1966, Ertekin 1969, Payan 1969, Trojaborg and Sindrup 1969, Behse and Buchthal 1971, Trojaborg 1976, Inouye and Buchthal 1977, Inouye 1978, Stohr et al. 1978, Falck et al. 1984).

The advantages of the different techniques in use have to be balanced against the disadvantages, and the choice of method depends on whether it is used for daily routine work or with the aim of increasing the diagnostic yield.

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The advantages of the near-nerve recording technique over the surface electrode method according to Buchthal and Rosenfalck (1966), Wagner and Buchthal (1972), Buchthal et al. (1975) and Rosenfalck (1978) can be summarized as follows:

1) the amplitude of the sensory nerve action potential is 2-4 times larger with needle than with surface recording (Fig. 1);

2) the latency of the initial positive peak of the SNAP is determined with greater accuracy, the positive peak is shorter and better defined with a needle than with a surface electrode because the leading-off area of the former is smaller than the latter, the needle electrode is closer to the nerve and the low-pass skin filter effect is absent;

3) irregularities in the shape of the SNAP can be visualized indicating affection of fibres of certain diameters;

4) when the SNAP is within the noise level with surface electrodes, it can usually still be recorded with needle electrodes (Fig. 2);

5) it enables the recording of SNAPs at longer distances from the stimulating electrodes for determination of sensory conduction velocities (SCV) in distal, intermediate and proximal nerve segments (Fig. 3);

6) SCV of the fastest and slowest conducting fibres can be estimated in normal nerves, covering the spectrum of fibre diameters from 14 to 4 μm as well as in demyelinated, remyelinated and regenerated nerve fibres;

7) the noise from the electrode-tissue surface is 2-3 times lower than with percutaneous electrodes, resulting in a signal-to-noise ratio that is 5 times higher than that obtained with surface electrodes, thus giving a better resolution when averaging technique is needed to record SNAPs;

8) when needle electrodes are used for stimulation, the distribution of current in relation to the nerve is better defined, the maximum field strength being near the bared tip of the electrode;

9) the current required to obtain a maximal response is 10 times less than with surface electrodes;

10) the smaller the stimulating current the lesser the spread along the nerve, giving a better coincidence between the site of the stimulating cathode and the site of the initiation of the nerve impulse;

11) the lower the stimulus strength the lesser the chance to stimulate neighbouring nerves;

12) the induced stimulus artefact interferes less with the SNAP take-off, making latency measurements more precise.

The disadvantages of the near-nerve technique have been claimed to be distress caused by needle insertions, the possibility of penetration into or through the nerve trunk and that the application of the technique may take appreciably more time than the percutaneous method (Brown and Bolton 1984). However, needle electrodes have been used routinely for stimulation of and recording from nerves during more than 25 years in the Laboratory of Clinical Neurophysiology, "Rigshospitalet", University Hospital of Copenhagen without encountering any difficulties or side-effects. The increased accuracy and information obtained with needle electrodes far outweigh the inconvenience of using them.

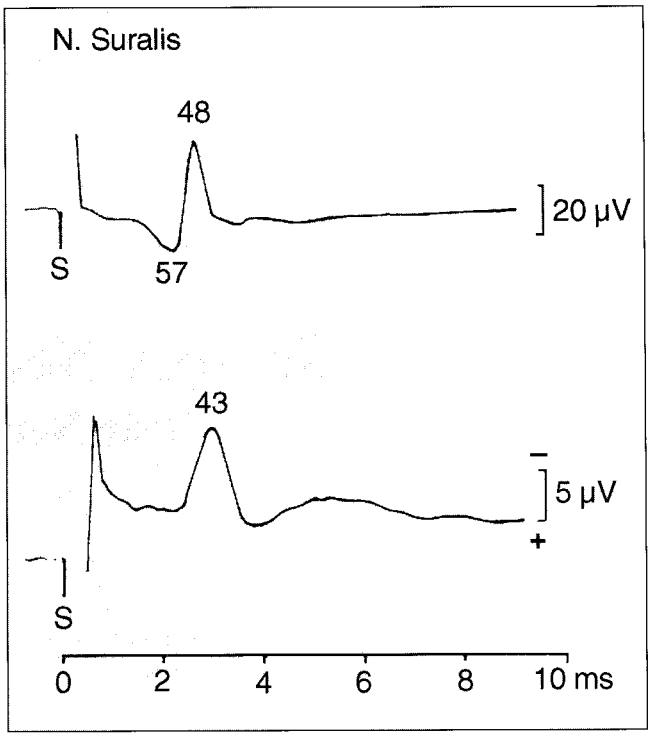

Fig. 1. Sensory nerve action potentials (SNAP) from the sural nerve recorded at mid-calf after stimulating the nerve behind the lateral malleolus. Upper trace, near-nerve monopolar recording, lower trace, bipolar surface electrode recording, with the active electrode placed at the same site as the stigmatic needle electrode. Note the difference in peak-to-peak amplitude of the SNAPs, the upper being 45 μV, the lower 10 μV; note also the absence of a well defined positive peak and the slower rise time of the negative peak of the percutaneously recorded potential. The figures above and below the traces denote the conduction velocities calculated from the latencies of the positive and negative peaks, respectively. S = stimulus.

The variability in amplitude of the SNAP is the same whether recorded with needle or surface electrodes (Buchthal and Rosenfalck 1966). However, in repeated studies the SNAP amplitude in the same subject may vary 20-40% (Trojaborg 1970, Rosenfalck 1978) due to variation in the distance between electrode and nerve at different examinations, whereas the SNAP picked up with surface electrodes will remain essentially constant. Moreover, it has been postulated, in a short report, that the amplitude of SNAPs recorded with surface electrodes is more frequently abnormal than that

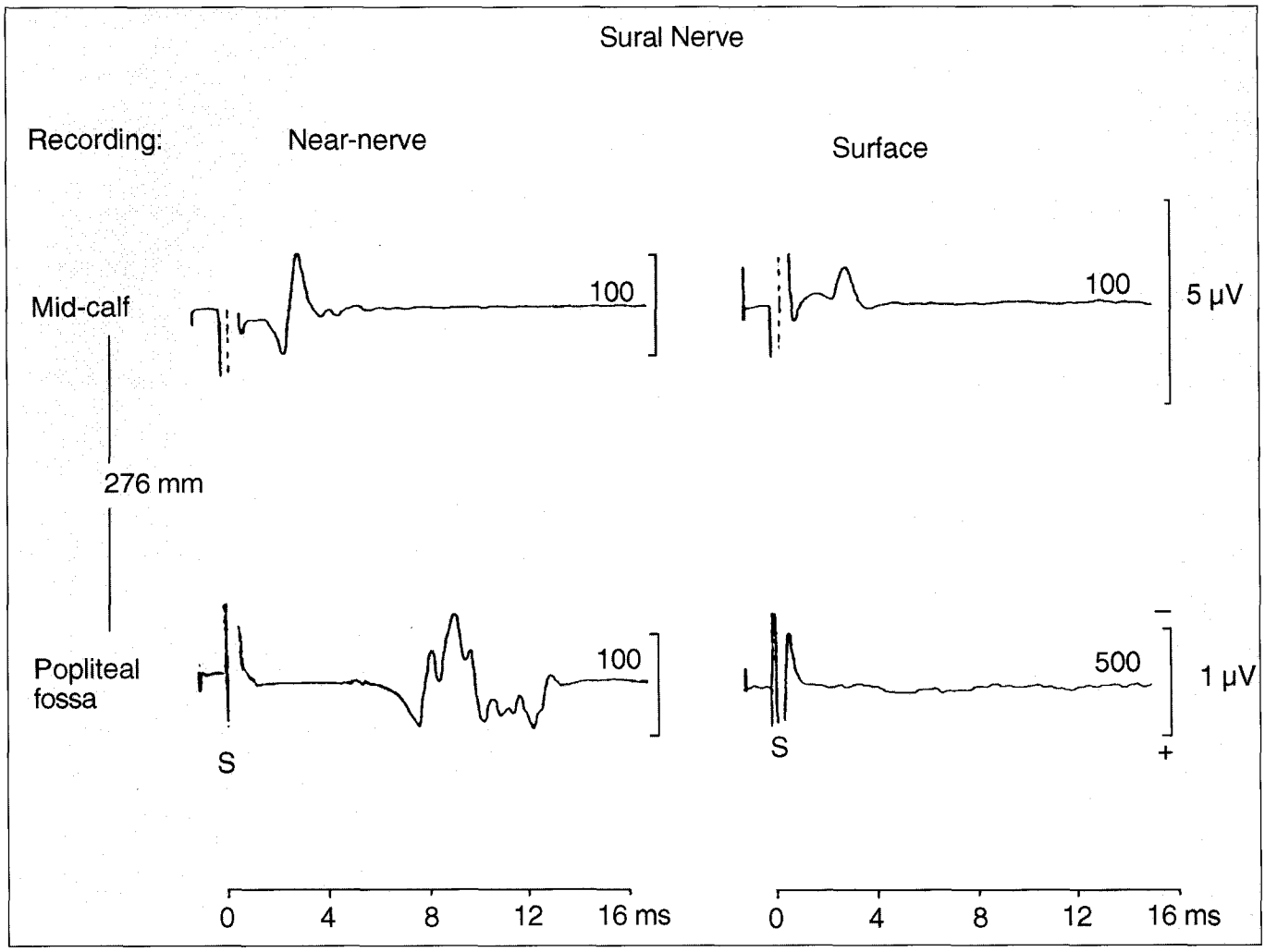

Fig. 2. Sensory nerve action potentials recorded from the sural nerve by near-nerve and surface electrodes (left and right columns, respectively). A stigmatic electrode was placed close to the sural nerve at mid-calf and in the popliteal fossa with reference to electrodes placed at a transverse distance of 30 mm at the same level as the near-nerve electrodes. A supramaximal stimulus (S) was applied to the sural nerve behind the lateral malleolus. Surface electrodes were then placed with the active electrode at the same site as the near-nerve electrodes with reference to electrodes 25 mm proximal to them. Responses of 100 stimuli were averaged to record SNAPs at mid-calf (upper two traces) and 100 and 500 stimuli, respectively, were averaged for recording in the poplitea fossa (lower two traces). Note the absence of a response at the proximal site (right lower trace).

recorded with needle electrodes in patients with peripheral nerve pathology (Andersen 1985b), a statement, however, that has to be reconsidered.

Whether near-nerve or surface electrode technique is used, monopolar is superior to bipolar recording (Buchthal and Rosenfalck 1966, Winkler et al. 1991). Although SNAP amplitude might be higher when the recording electrodes are placed longitudinally over the nerve, this depends on the interelectrode distance, as do the shape and duration of the SNAP. The compound nerve action potential represents the difference betweeen the time-displaced responses recorded at the active and reference electrodes, respectively. The SNAP recorded monopolarly is much less affected by the "distant" reference electrode placed at a transverse distance from the stigmatic electrode of 30-40 mm.

The following review will deal only with near-nerve recording technique.

Methods of Near-Nerve Electrode Recording

Stimulation and Recording Procedure

Sensory nerves are stimulated via surface electrodes wrapped around the digits or by needle electrodes placed near the nerves.

Using near-nerve stimulation, the electrical stimulus is applied through insulated stainless needle electrodes 0.7 mm in diameter (Buchthal and Rosenfalck 1966). The near-nerve electrode with a bared tip of 3 mm is adjusted close to the nerve; the remote electrode with a bared tip of 5 mm is placed at the same level as the stigmatic electrode at a transverse distance of 30-40 mm. To activate all nerve fibres, a stimulus of 0.2 msec duration and at least five times above the sensory threshold is applied, whether using surface or needle electrodes. It is delivered from a constant current stimulator isolated from ground.

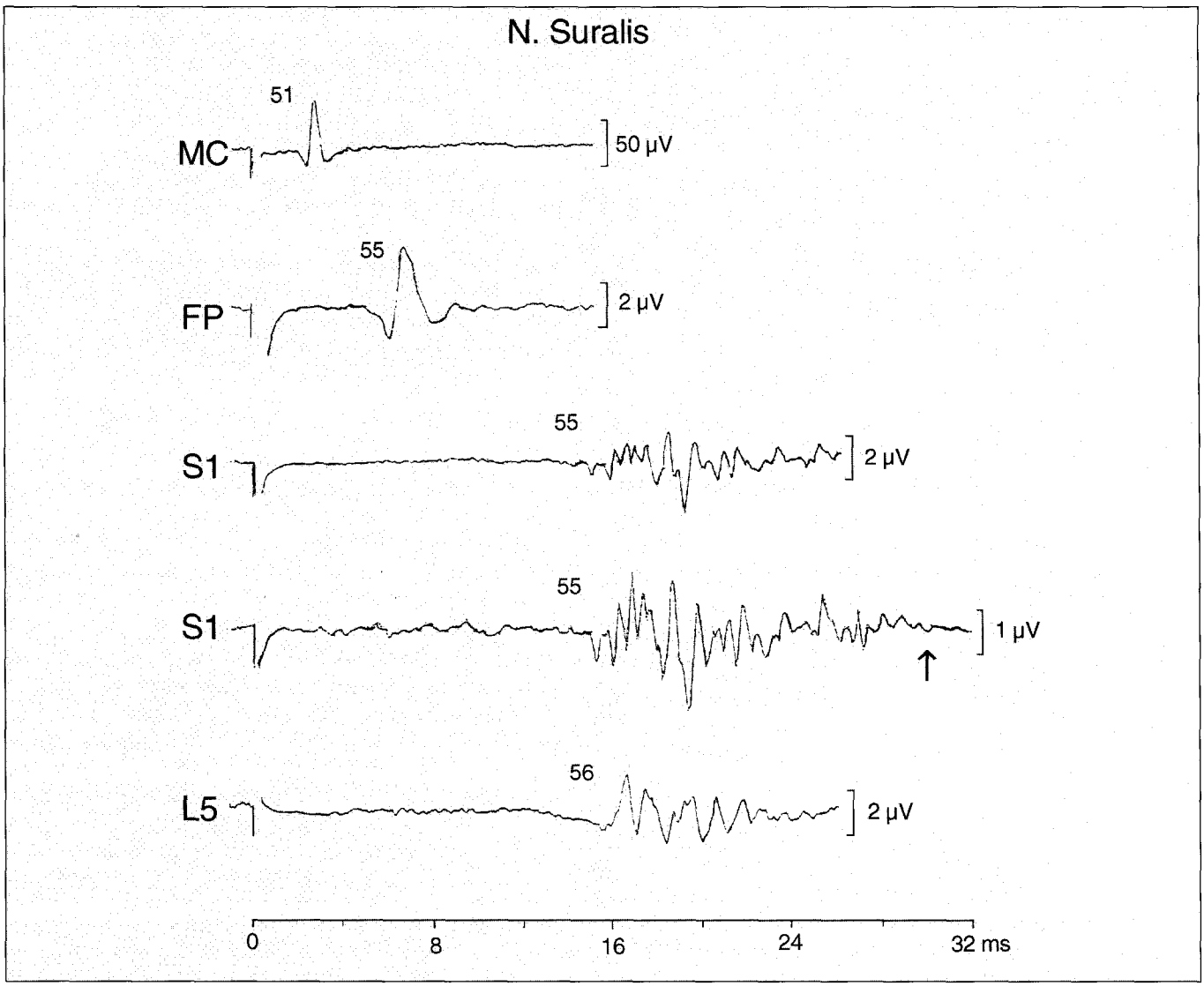

Fig. 3. Sensory nerve action potentials (SNAP) recorded over the sural nerve at mid-calf (MC) 127 mm proximal to the site of stimulation at the lateral malleolus, at the popliteal fossa (FP), S1 and L5 spinal roots. The stimulus was 2.5 mA i.e. 12.5 times the lowest stimulus strength needed to elicit a small nerve action potential at MC. The SNAP at S1 was recorded at two different sampling times (30 and 50 msec, respectively) and at two different amplifications (note the difference in calibration signals for the two S1 traces) to determine the end of the potential (arrow). The figures above each trace denote the conduction velocity in m/sec for successive nerve segments tested. The SNAPs at MC and FP were responses to single stimuli, those recorded at S1 and L5 averaged responses of 250 stimuli. The temperature along the whole limb was 35 °C.

It is important to keep the impedance of the recording electrodes as low as possible as this will minimize the electrode noise, cause less distorsion of the nerve action potentials and improve the resolution when averaging is necessary to obtain SNAPs. By electrolytic treatment the electrode impedance can be reduced from 50 kΩ to about 1 kΩ at low as well as at high frequencies, and the noise from 1 µV to 0.35 µV rms (10-5000 Hz, Buchthal and Rosenfalck 1966).

The electrolytic treatment can be performed via the EMG equipment's stimulator output. The electrode pair is placed in a cup filled with physiological saline and the electrode cable is plugged into the socket of the stimulator output. A 50 mA stimulus of 1 msec duration is applied at a frequency of 20 Hz for 30 sec with the stimulus polarity set at negativity.

Following this, an 80 mA stimulus of the same duration and frequency is applied for another 30 sec with a positive stimulus polarity.

Guidelines for Electrode Placements

This applies to the active or stigmatic near-nerve electrode only. The inactive or remote electrode is always inserted at the same level as the near-nerve electrode at a transverse distance of 30-40 mm unless otherwise stated. The optimal electrode position is achieved using a muscle innervated by the nerve under investigation as a guide whenever possible.

By minor adjustments of the needle, a threshold for evoking a muscle action potential of about 0.5 mA can be obtained. For pure sensory nerves such as the superficial branch of the radial nerve, the sensory branch of the musculocutaneous nerve and the sural nerve, the position of the stigmatic electrode is changed until a nerve action potential can be evoked at the recording electrode at a stimulus strength of 0.3-0.7 mA. To ensure an optimal position of both recording and stimulating electrodes, the procedure can be applied for both an orthodromic and an antidromic propagated impulse.

Upper Limb Nerves

1) Median Nerve

Stimulation of sensory fibres:
a) through surface electrodes wrapped around digit I and III (eventually DIV). The anode is placed at the distal phalanx as far distal as possible, the cathode about 20 mm proximal to it to avoid or minimize simultaneous activation of digital radial nerve fibres.

b) at palm via needle electrodes placed as described below.

Recording from sensory fibres:
a) at palm: 15-20 mm distal to the distal edge of the flexor retinaculum along a line pointing to the midline of digit III about 90 mm from the stimulating cathode on digit III.

b) at wrist: 20-30 mm above the distal crease between the tendons of the flexor carpi radialis longus and the flexor digitorum superficialis.

c) at elbow: just medial to the brachial artery at the elbow bend.

d) at axilla: lateral to the brachial artery between this and the medial border of the short head of the brachial biceps.

e) at supraclavicular fossa: about 20 mm proximal to the clavicle lateral to the sternocleidomastoid muscle between the middle and the medial 1/3 of the clavicle.

2) Ulnar Nerve

Stimulation of sensory fibres:
via surface ring electrodes placed around the distal phalanx of DV or DIV. The dorsal cutaneous branch can be stimulated just proximal to the base of the 5th metacarpal bone on the dorsum of the hand.

Recording from sensory fibres:
a) at wrist: 20 mm proximal to the pisiform bone just lateral to the tendon of the flexor carpi ulnaris.

b) at wrist (ulnar dorsal cutaneous nerve): about 40-60 mm proximal to the styloid process of the ulnar bone.

c) at elbow: 50 mm distal to the acromion of the ulnar bone in the ulnar groove.

d) at elbow: 50 mm proximal to the acromion in the ulnar groove.

e) at axilla: in the medial groove between the short head of the brachial biceps and the brachial triceps muscles dorsal to the brachial artery.

f) at supraclavicular fossa: see median nerve e).

3) Radial Nerve

Stimulation of sensory fibres:
a) via surface electrodes placed around the proximal phalanx of the thumb.

b) by needle electrodes placed at the wrist as indicated below.

Recording from sensory fibres:
a) at wrist: along the lateral border of the radial bone about 20 mm proximal to the styloid process where it can be easily palpated.

b) at elbow: in the groove between the brachioradial muscle and the brachial biceps tendon.

c) at the spiral groove on the dorsal aspect of the shaft of the humerus approximately corresponding to its middle.

d) at axilla: in the groove between the coracobrachial and the medial edge of the brachial triceps muscles.

4) Musculocutaneous Nerve

Stimulation of sensory fibres:
a) by surface or needle electrodes placed at the wrist 40-50 mm proximal to the distal crease midways between the tendon of the flexor carpi radialis and the radial bone.

b) via needle electrodes placed at the elbow as indicated below.

Recording from sensory fibres:
a) at elbow: at the crease lateral to the tendon of the brachial biceps and medial to the brachioradialis muscles where it can be easily palpated.

b) at axilla: between the axillary artery and the median nerve medially, and the coracobrachial muscle laterally, just above the level of the tendon of the latissimus dorsi muscle.

c) at the anterior cervical triangle just behind the sternocleidomastoid muscle, approximately 60 mm above the clavicle.

5) Axillary Nerve

Stimulation of sensory fibres:
via platinum needle electrodes inserted subcutaneously in the sensory area over the deltoid muscle.

Recording from sensory fibres:
a) at the anterior cervical triangle as described for the musculocutaneous nerve (see section c).

6) Suprascapular Nerve (Articular Branch)

Stimulation of sensory fibres:
via needle electrodes placed in the sensory area of the articular branch just posterior to the acromioclavicular joint at the junction of the medial surface of the acromion and the posterior surface of the clavicula.

Recording from sensory fibres:
near the suprascapular nerve in the supraclavicular fossa using the supraspinatus muscle as an indicator for optimal electrode placement.

Lower Limb Nerves

1) Saphenous Nerve

Stimulation of sensory nerve fibres:
a) through surface or needle electrodes placed at the medial site of the leg just above the medial malleolus.

b) via needle electrodes placed at the medial site of the knee just below the lower edge of the patella and below the medial epicondyle.

c) via needle electrodes at the thigh where the nerve descends deep to the sartorius muscle going through the adductor canal (Hunter's canal) from which it exits about 150 mm above the medial epicondyle.

Recording from sensory nerve fibres:
a) at the inguinal ligament just lateral to the femoral artery.

b) at the thigh as described above (see c).

2) Lateral Cutaneous Femoral Nerve

Stimulation of sensory fibres:
120-140 mm distal to the inguinal ligament on a vertical line going through the spina iliaca.

Recording from sensory fibres:
At the groin just medial to the anterior spina iliaca superior.

3) Tibial Nerve

Stimulation of sensory fibres:
a) via surface ring electrodes placed around the big toe.

b) the interdigital plantar nerves can be stimulated via platinum electrodes placed on each side of adjoining toes.

c) at the site where the two branches join, the cathode being placed 20 mm proximal to the anode.

Recording from sensory fibres:
a) at ankle: equidistant from the medial border of the insertion of the Achilles tendon and the medial malleolus.

b) at popliteal fossa: in the middle between the tendons of the semimembranosus and the biceps femoris muscles.

c) at the gluteal fold: equidistant from the greater trochanter of the femur and the ischial tuberositas.

4) Peroneal Nerve

Stimulation of sensory fibres:
a) the superficial branch is stimulated 50-70 mm proximal to the superior retinaculum between the tendons of the long peroneal and the long extensor digitorum muscles.

b) the deep branch (dorsal digital cutaneous nerve), which supplies adjacent sides of the first and second toe, is stimulated over the dorsum of the foot corresponding to the first interstice.

Recording from sensory fibres:
a) at ankle: 40-60 mm proximal to the tip of the lateral malleolus just lateral to the tendon of the anterior tibial muscle.

b) below capitulum fibulae: about 20 mm distal to the fibular head close to the superficial peroneal nerve.

c) about 70-90 mm proximal to the fibular head in the popliteal fossa medial to the tendon of the biceps femoris muscle.

5) Sural Nerve

Stimulation of sensory fibres:
a) at dorsum pedis: at the lateral edge of the foot approximately midway between the little toe and the lateral malleolus.

b) at the lateral malleolus: about 30 mm proximal to the tip of the lateral malleolus equidistant from the fibula and the Achilles tendon.

Recording from sensory fibres:
a) at the lateral malleolus: see above.

b) at mid-leg: 120-140 mm proximal to the lateral malleolus.

c) at the popliteal fossa and gluteal fold: see under tibial nerve.

d) at spinal root S1: a line between the posterior iliac spines levels with the second sacral spine. The first sacral foramen is about 30 mm above the midpoint between the two spines.

Maximum and Minimum Conduction Velocity (CV)

The CV of the fastest conducting fibres is calculated from the latency of the first positive peak of the SNAP, which is determined by myelinated fibres 13-14 μm in diameter. The CV of the slowest conducting fibres is evaluated from the latest component of the SNAP which can be distinguished from noise. This component is identified by averaging the responses to 500 and 1000 stimuli as it remains similar in shape but increases in amplitude proportionally to the calibration signal summated together with the sensory action potential (Fig. 4). The minimum CV in the sural nerve is 15 m/s, and the lower 95% confidence limit is 11 m/s (Behse and Buchthal 1971, Shefner et al. 1991a,b). According to Gasser and Erlanger (1927), the conduction velocity varies proportionally with the diameter of the nerve fibres. With this in mind, the conversion factor for sural nerve fibres was determined to be 4.4, based on the relation between the fastest component of the SNAP and the diameter of the largest myelinated nerve fibres determined in the same nerve (Buchthal et al. 1975, Behse and Buchthal 1977a, Buchthal and Behse 1977, 1987). Moreover, the conversion factor was the same for the fast and the slower components of the SNAP (Buchthal 1974). In a similar study, Tachmann et al. (1976) calculated the conversion factor as 4.8 for sensory fibres of the radial nerve and 4.6 for sural nerve fibres compared to 4.7 in a computer-based modelling of SNAPs (Stegeman and DeWeerd 1982a).

At least 10 normally conducting nerve fibres are required to obtain an averaged sensory potential of about 0.05 μV after 500 to 1000 stimuli (Buchthal 1974, Behse et al. 1975). To distinguish a sensory nerve action potential from noise using surface electrodes would probably require the presence of at least 400 fibres conducting at a normal rate, considering the amplitude relation of SNAPs for near-nerve and surface recordings. However, the exact relation has not been evaluated by comparing actual recordings with sural nerve histology.

The amplitude of the SNAP measured from the largest positive to the largest negative deflection increases about proportionally with the logarithm of the number of nerve fibres, more steeply for fibres above than for those below 10 μm.

In the sural nerve the amplitude of the main component of the SNAP originates from about 1600 myelinated fibres (normal range 1000-2500) with a diameter of 9-14 μm. Later components originate from fibres of 4-7 μm in diameter (Behse and Buchthal 1973, Buchthal 1974).

Factors Influencing Nerve Conduction Velocity

These are generally stated to be age, temperature, gender and height, although there is no overall agreement as to their degree of effect on nerve conduction parameters.

a) Age

A linear decrease in CV with increasing age of 0.8-1.8 m/s per 10 years has been described (Wagman and Lesse 1952, Norris et al. 1953, Downie and Newell 1961, La Fratta and Canestrari 1966, Buchthal and Rosenfalck 1966, Behse and Buchthal 1971, Casey and Le Quesne 1972, La Fratta and Zalis 1973, Nielsen 1973a, Burke et al. 1974, Singh et al. 1974, Trojaborg 1976, Vandendriessche et al. 1981, Horowitz and Krarup 1992) but does not apply to all nerves in the same individual (Burke et al. 1974, Lang et al. 1985, Rivner et al. 1990). There are also reports that deny any relation within ages from 6-84 years (La Fratta and Smith 1964, Levy and Pool 1966, Burke et al. 1974, Dioszeghy 1986). In a recent study (Trojaborg et al. 1992) of 92 normal subjects aged 15-44 years, sural nerve CV was found to decline 0.09 m/s per year. By applying quadratic regression analysis to study the relation between age and CV, Taylor (1984) obtained a similar value (0.07 m/s) between the ages of 15-44 years. In both studies the change with age was of no clinical significance.

b) Sex

Some authors have claimed that CV may be up to 6 m/s faster in women than in men (La Fratta and Smith 1964, Kemble 1967a,b, Lang et al. 1985, Gadia et al. 1987), findings not supported by others (Nielsen 1973a, Campbell et al. 1981, Horowitz and Krarup 1987, 1992, Trojaborg et al. 1992).

The average amplitude of the sensory nerve action potential is higher in women than in men according to several studies (Casey and Le Quesne 1972, DiBenedetto 1972, Shirali and Sandler 1972, Felsenthal 1978, Bolton and Carter 1980, Moon et al. 1985, Horowitz and Krarup 1987, 1992, Trojaborg et al. 1992), but so far a plausible explanation for the difference has not been suggested.

c) Temperature

This is the most important factor influencing CV. The estimated decrease in CV per degree decline in temperature varies between 1.1-2.4 m/s, depending on the nerve studied and the technique used (Henriksen 1956, Gassel and Trojaborg 1964, Trojaborg 1964, Buchthal and Rosenfalck 1966, Casey and Le Quesne 1972, De Jesus et al. 1973, Lowitzsch et al. 1977, Ludin and Beyler 1977, Halar et al. 1980, Bolton et al. 1981, Cummings and Dorfman 1981, Halar et al. 1981, Bolton et al. 1982, Stegeman and DeWeerd 1982b, Halar et al. 1983, Geerlings and Mechelse 1985, Todnem et al. 1989, Trojaborg et al. 1992). Only few studies neglect the effect of temperature on CV (Soudmand et al. 1982, Rivner et al. 1990).

It cannot be recommended to correct CV values determined in cold limbs under pathological circumstances, as fibres of different calibres react differently to temperature changes (Douglas and Malcolm 1955).

An inverse relation between SNAP amplitude and temperature has been described. When the temperature decreased 6 °C the amplitude increased by 6-10 μV (DiBenedetto 1976, Bolton et al. 1982). These findings, however, had not been reproduced by others (Buchthal and Rosenfalck 1966, Casey and Le Quesne 1972, Stegeman and DeWeerd 1982b, Todnem et al. 1989, Trojaborg et al. 1992), and Lang and Puusa (1981) found a dual influence of temperature on the SNAP amplitude, which increased during focal cooling from 30-20 °C as well as during focal heating from 20-35 °C

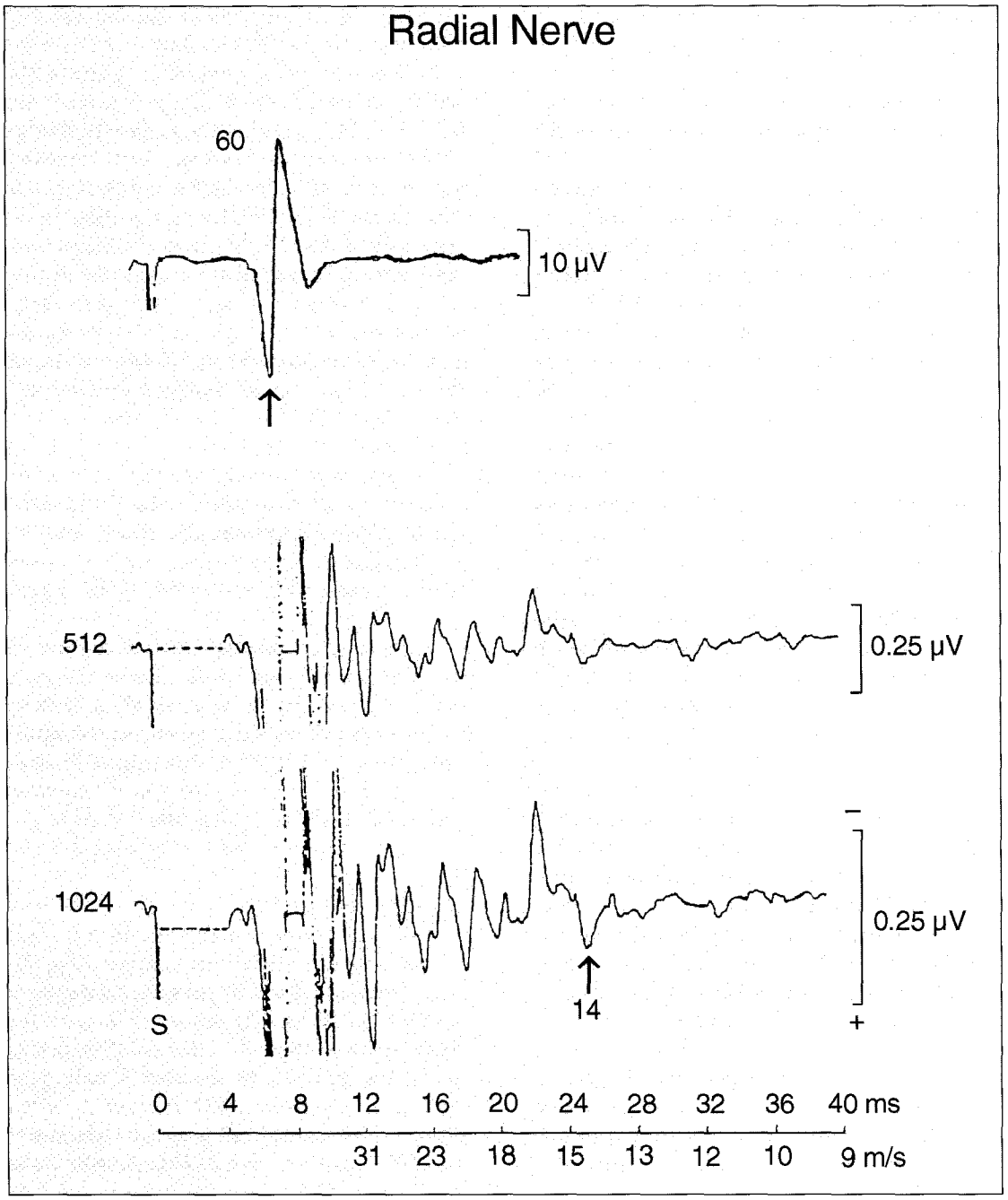

Fig. 4. Sensory nerve action potentials recorded over the radial nerve at elbow after stimulating the sensory branch at wrist. Upper trace, response to a single stimulus (S), lower 2 traces, average responses after 512 and 1024 stimuli, respectively, recorded at high gain for identification of slow components. The figure above the upper trace indicates the maximum conduction velocity (CV) in m/sec, that below the lower trace the minimum CV. The stippled lines in the two lower traces represent a 4 msec delay before sampling.

d) Height

A possible inverse relation between height and CV was first introduced by Lang et al. and Bjorkqvist et al. in 1977, and later supported by others (Campbell et al. 1981, Soudmand et al. 1982, Gadia et al. 1987, Rivner et al. 1990). An increase in height of 100 mm was found to be related to an average decrease of CV of 3 m/s in men, but less than 2 m/s in women (Lang et al. 1977). Similarly, Rivner et al. (1990) found a decrease in CV of 3.2 m/s per 100 mm increase in height, i.e. a difference of 22 m/s between their shortest and tallest subjects. It is likely that this wide scatter of velocities in part reflects the negligence of temperature influence. No attempts were made to obtain an even temperature along the nerve segments examined; the temperature measured at the sole of the foot varied between 25-37 °C (Rivner et al. 1990). In more recent studies of the sural nerve in normal subjects, no significant difference in CV was found between men and women or between subjects of different heights (Horowitz and Krarup 1987, 1992, Trojaborg et al. 1992).

Clinical Application of the Near-Nerve Electrode Technique

Focal neuropathies:

In most cases of nerve injuries caused by trauma or entrapment, the site of the lesion can be established with greater accuracy when investigating the sensory than the motor conduction. Localization of a focal lesion implies recording of evoked SNAPs below and above the presumed site of nerve affection over short distances as well as evoking compound muscle action potentials at proximal and distal sites. Spread of the stimulating current may invalidate the accurate assessment of motor CV but does not influence SCV calculation.

The carpal tunnel syndrome (CTS)

Using surface electrodes for recording, the technique may fail to obtain a sensory potential at wrist in 20-70% of CTS patients (Kaeser 1966, Thomas et al. 1967, Kopell and Goodgold 1968, Hongell and Mattsson 1971, Sedal et al. 1973).

Under such circumstances, the absence of a SNAP does not support the diagnosis of CTS if the distal latency to the abductor pollicis muscle is normal. In contrast to surface recording, a SNAP was present at wrist and elbow in all 111 patients with signs and symptoms of CTS when near-nerve technique was applied (Buchthal et al. 1974). Moreover, in 25% of the patients in whom motor conduction and EMG were normal, the lesion was located from abnormalities in sensory conduction. In most patients conduction and amplitude of SNAPs following stimulation of DI were as abnormal as when DIII was stimulated. However, abnormalities would have been missed in 10% of the patients if only one of the digits had been stimulated. That is, in 10% of the CTS patients the SCV was slowed in the segment DI-wrist but normal in the segment DIII-wrist or vice versa. Finally, the SCV from the digits to wrist was normal or borderline-slow in one-quarter of the patients. In these, excluding the segment from digit III to palm with normal or near normal CV, slowing was then significant from palm to wrist (Buchthal and Rosenfalck 1977a).

When stimulating the thumb, the electrode placed near the median nerve invariably picked up a SNAP from the radial nerve as well (Buchthal and Rosenfalck 1966, Trojaborg and Sindrup 1969). This does not obscure the onset of the potential from the median nerve unless electronic averaging is needed to record the SNAP. In about half of the CTS patients, the amplitude of the SNAP following stimulation of DI was 2 μV or less and the CV was slowed. In these cases, a SNAP from the radial nerve preceded that from the median nerve. A similar "double peak" potential can be seen when stimulating the ring finger (Lauritzen et al. 1991) and was first described by Simpson (1978, 1990), who named the procedure the „Camel test".

By analogy with the TV world it could perhaps be called the "Twin Peak Test". Figs. 5 and 6 exemplify D-palm-wrist recordings and DIV stimulation, respectively, in patients with CTS.

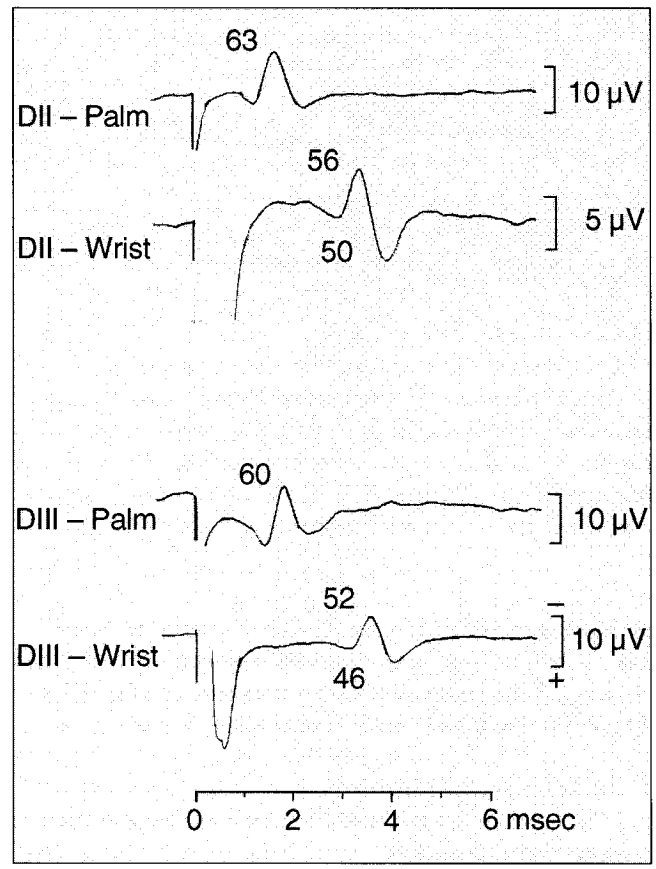

Fig. 5. Sensory nerve action potentials (SNAP) recorded simultaneously over the median nerve at palm and wrist after stimulation of the index (DII) and middle finger (DIII). The figures above traces denote the conduction velocity (CV) in m/sec between the site of stimulation and recording, those below the CV from palm to wrist. Note normal CV from digits to wrist and digits to palm, but slowed CV in the segment palm-wrist, 17% and 23% for fibres of DII and DIII, respectively. From a 45-year-old woman with paraesthesiae localized to DII and DIII.

Ulnar nerve lesions

a) At the thoracic outlet

Compression of the lower cervical roots or inferior trunk of the brachial plexus, the so-called thoracic outlet syndrome, is a rare condition (Gilliatt et al. 1970). Electrical studies are important to determine the degree of involvement and if possible to localize the lesion. This, however, can only be done by exclusion. Contrary to other focal nerve lesions, conduction studies across the site of compression are difficult to accomplish. Changes in F-wave latency are of little help (Wulff and Gilliatt 1979) and the findings by Inouye and Buchthal (1977) in one of three patients with a cervical rib of a SNAP with diminished amplitude at the eighth spinal nerve and conducted at a slowed rate have not been confirmed by others. Studies of motor conduction across the thoracic outlet are not reliable tools, nor do somatosensory evoked potentials contribute to the diagnosis (for reference see Smith and Trojaborg 1987). They state that the combined findings of chronic partial denervation of the ulnar and median innervated

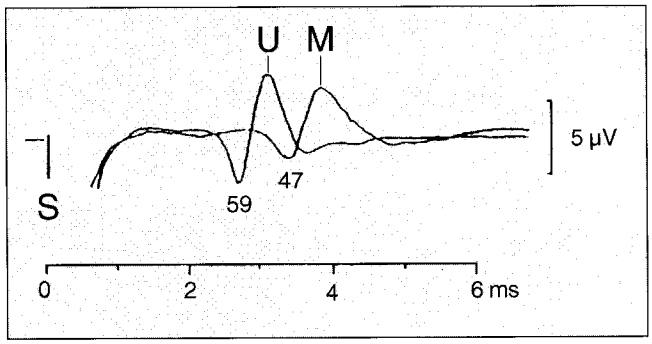

Fig. 6. Sensory nerve action potentials recorded simultaneously over the ulnar (U) and median (M) nerves at wrist after stimulation of the ring finger (DIV). The figures below traces denote the conduction velocities in m/sec from DIV to U and M, respectively. From a 40-year-old woman with signs and symptoms of carpal tunnel syndrome. S = stimulus.

small hand muscles decreased SNAP amplitude from digit V and sometimes also from digit III, and normal motor and sensory CV are compatible with a compression of the C8 and T1 roots or the lower trunk of the brachial plexus.

b) At the cubital sulcus

The most common cause of ulnar nerve dysfunction is compression at the elbow region during anaesthesia or sleep, repeated trauma or mechanical abnormalities of the elbow joint. Localization of the site of lesion is essential, because the ulnar nerve is liable to injury at other sites. The symptomatology may present more or less similarly, independent of the site of involvement.

The most comprehensive, careful and detailed electrophysiological study of ulnar nerve lesions with respect to localizing the site of affection was performed by Payan (1969). He demonstrated by near-nerve electrode technique the diagnostic value of recording sensory nerve action potentials at three sites along the course of the ulnar nerve. He was able to record SNAPs at wrist, and below and above the elbow in 90% of his cases. For comparison, Gilliatt and Thomas (1960) were unable to obtain a SNAP at wrist in any of their patients with clinical established ulnar nerve lesions at the elbow. Nor could they record a mixed nerve action potential proximal to the elbow with surface electrodes. Similarly, Kaeser (1963) found that the SNAP was absent or too small to be used for measurement in 29 cases. A later study (Tackmann et al. 1984) using near-nerve technique confirmed the findings of Payan (1969) and expressed the same view as Payan, that sensory parameters had a somewhat greater sensitivity than motor parameters with respect to diagnosing ulnar nerve dysfunction.

Many reports have dealt with the problem of determining the exact value for conduction through the transsulcus region and how to position the arm during the electrophysiological investigation (Harding and Halar 1983, Kincaid et al. 1986, Kincaid 1988). This applies mainly to MCV, which is said to vary between 34-52 m/s (mean values) and most likely reflects the inaccuracy in distance measurement across the ulnar nerve groove. Whether the elbow is extended or flexed, the nerve volley has to travel along the same length of nerve. However, measuring the distance between sites above and below the elbow joint with the arm at 45-degree flexion more likely reflects the actual nerve stretch (Harding and Halar 1983). On the other hand they found, contrary to Kincaid et al. (1986), that 135-degree elbow flexion causes erroneous MCV and SCV (antidromically determined) estimates over the transsulcus area. This might be due to dislocation of the nerve during elbow flexion, which occurred in 26% of the patients studied by Payan (1969).

Combining all parameters, sensory as well as motor, including CV determinations in fibres to both abductor digiti minimi (ADM) and adductor pollicis brevis (AP) (Ebeling et al. 1960) and latency to flexor carpi ulnaris, raises the diagnostic yield. By doing so, 96% of 50 ulnar nerve lesions were localized by electrophysiologiocal means, compared with 26 on clinical grounds (Payan 1969). These findings were later confirmed by Tackmann et al. (1984) using a similar technique.

In experienced hands, a complete ulnar study may take about an hour to perform if averaging is necessary to obtain SNAPs. When interpreting findings, it is worth noting that both MCV and SCV can be slowed in the forearm segment as lesions of the ulnar nerve are likely to comprise both demyelination and wallerian degeneration (Neary and Eames 1975). Therefore, to have localizing value, the transcubital CV has to be disproportionately slower than in the forearm. Also, if only MCV is investigated, it is necessary to study conduction in the upper arm as well. Slowing proximal to the lesion may occur due to retrograde changes in compressed fibres similar to findings in the carpal tunnel syndrome (Thomas 1960) and is associated with reduction in fibre diameter and density as shown in animal experiments (Fullerton and Gilliatt 1967).

Finally, it should be noted that a normal SNAP amplitude at wrist does not preclude a more proximal lesion as it can be found in 13% of the cases with transcubital injury (Payan 1969).

c) At the wrist

To differentiate between the sites of involvement of the distal branches of the ulnar nerve, i.e. proximal, in or distal to Guyon's canal, it is necessary to determine distal latencies to ADM and AP and to study the conduction in the superficial terminal sensory branch as well as in the dorsal cutaneous branch (Jabre 1980, Kim et al. 1981).

Fig. 7 is an example of an ulnar nerve affection involving the superficial terminal branch but not the dorsal cutaneous branch. This alone, however, does not preclude a more proximal ulnar nerve lesion, as compression in the cubital sulcus may preferentially involve some sensory fibres more than others, just as fibres to ADM may be spared but fibres to AP entrapped or vice versa (Ebeling et al. 1960, Stewart 1987).

In the present case sensory CV across the elbow was normal.

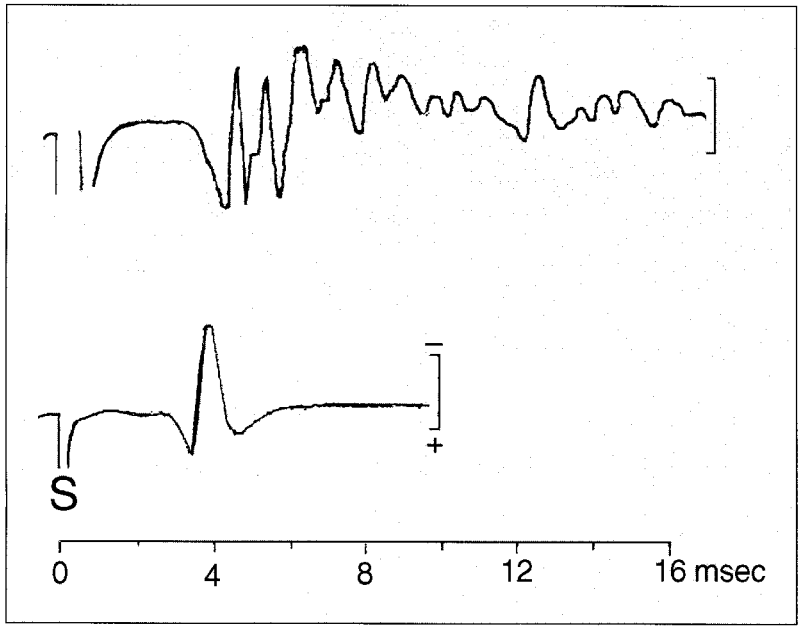

Fig. 7. Sensory nerve action potentials (SNAP) from a 44-year-old waitress who complained of progressive weakness of the small hand muscles and paraesthesiae localized to digits IV and V. The symptoms began insidiously about half a year before the present recording. On examination there were weakness and wasting of the small hand muscles innervated by the ulnar nerve and hypaesthesia in the ulnar nerve distribution sparing its dorsal aspect. Upper trace: SNAP recorded over the ulnar nerve at wrist after 500 stimuli to digit V. The maximum velocity (CV) was 44 m/sec, the minimum 11 m/sec, the amplitude of the main component 0.5 μV. Lower trace: SNAP recorded over the dorsal cutaneous branch of the ulnar nerve, CV 48 m/sec, amplitude 4 μV. These findings are consistent with an affection of the ulnar nerve at the entrance or within Guyon's canal. Calibration signal: upper trace 0.25 μV, lower trace 2.5 μV.

Radial Nerve

Damage to the radial nerve may occur as a complication to fracture of the shaft of the humerus as a direct consequence of the blow that fractured the bone. The nerve can also be caught between the ends of the broken bone or lacerated by a bony spur, traumatized due to separation of bony fragments or during reposition of the fracture. During healing the nerve can be compressed or trapped by callus.

The so-called "Saturday night palsy" is a classic example of neuropraxia due to compression of the radial nerve at the lateral border of the humerus, where it pierces the lateral intermuscular septum, or just below it; here the nerve is placed superficially and closely related to the humerus.

To delineate possible differences between these disorders, to determine the degree of involvement, and to predict the prognosis, it is essential to stimulate and record above and below the presumed site of nerve damage. In a study of 58 patients with different types of radial nerve injury a classification of the type of damage was attempted (Trojaborg 1970). In patients with palsy secondary to fracture, outgrowth in motor and sensory fibres was equal and estimated to be about 1 mm per day. In patients with Saturday night palsy, there was considerable slowing in both motor and sensory fibres across the presumed site of the lesion with return to normality within 6-8 weeks consistent with local demyelination as the cause of nerve palsy. Changes in sensory conduction were present even when there was no sensory deficit clinically and there was no difference in susceptibility of motor and sensory fibres to compression.

In a study of 31 patients with electrophysiological evidence of conduction block, the delay of the sensory action potential recorded above the presumed site of nerve compression and the attenuation of its amplitude was thought to be due to a block of the largest myelinated fibres. This assumption was based on the fact that the amplitudes of the fastest and slower conducted components in the patients differed little from corresponding components in the normal nerve (Trojaborg 1978).

Brachial plexus lesions

In cervical and lumbosacral compression syndromes with clinical evidence of sensory involvement, the presence of normal SNAPs conducted at normal rates is consistent with involvement of the sensory nerve roots proximal to the dorsal root ganglion. Similarly, in brachial plexus injuries with muscle paralysis and anaesthesia corresponding to one or more roots, the presence of SNAPs favours the diagnosis of

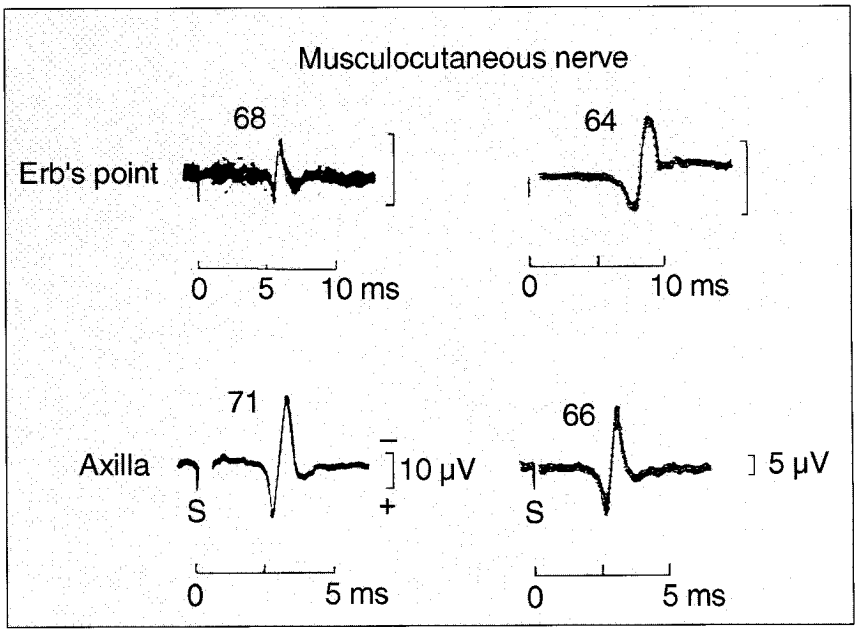

Fig. 8. Sensory nerve action potentials evoked by supramaximal stimulation of the lateral cutaneous nerve of the forearm at the elbow (S) and recorded at axilla and Erb's point in a 20-year-old normal subject (left) and in a 17-year-old man with avulsion of the fifth cervical root following a motor-bike accident (right). Upper left trace: photographic superimposition of 15 traces. The figures above the traces give the maximal conduction velocity (m/s) in the segment of the nerve distal to the point of recording.

root avulsion (Bonney and Gilliatt 1958, Trojaborg 1976, 1979, 1991). An example is shown in Fig. 8.

Paralysis of the brachial plexus as a complication of generalized anaesthesia or as a consequence of carrying heavy weight on the shoulders (rucksack palsy) has been described in several cases (for ref. see Trojaborg 1977b). These palsies may be due to a conduction block caused by local demyelination alone or combined with axonal loss. The characteristic electrophysio-logical findings were severe attenuation of amplitude of the compound motor and sensory action potentials evoked or recorded above the site of nerve injury compared to those evoked or recorded below. In addition, there was slowing of motor and sensory conduction across the damaged area (Trojaborg 1977b). Similar findings have been described in a case of idiopathic brachial lesion with conduction block of the ulnar nerve at the inferior part of the brachial plexus (Krarup and Sethi 1989).

Peroneal nerve lesions

Symptoms and signs of common peroneal neuropathies can vary considerably and make it difficult to localize the site of involvement on clinical grounds alone. The varied selectability in motor and sensory involvement has been related to differing degrees of damage to individual fascicles within the nerve (Sourkes and Stewart 1991). In this respect, EMG and nerve conduction studies can be helpful.

In 38 of 47 patients (81%) with a history suggestive of compression of the common peroneal nerve in the region of the fibular neck , electrical studies of sensory function localized the site of lesion using three criteria: 1) slowing of CV across the region of the capitulum fibulae, but normal distal to it (64%), 2) more than 10 m/s slower CV across than below, but within the normal range (9%) and 3) slow CV throughout the nerve, but more so across than below, the difference being > 10 m/s (9%, Singh et al. 1974). In 3 patients the lesion was localized on account of an abnormal shape of the SNAP recorded above the head of the fibula. The amplitude as such was of poor localizing value as it was reduced below the 95% limit of normal about equally often distally and proximally. For comparison, the lesion was correctively localized by MCV determinations in one-third of the 47 patients. The use of surface electrodes would have failed to detect a sensory response in about one-third of patients with localized compression palsies of the peroneal nerve (Lovelace et al. 1973). In a study of 116 common peroneal mononeuropathies, the antidromic recorded SNAP in the superficial peroneal nerve was considered of no diagnostic value as it was normal in 20% and absent or of low value in 80% (Katirji and Wilbourn 1988). They did not, however, attempt to record SNAPs above the site of lesion.

In a follow-up study of 14 patients with peroneal compression lesions it was shown that less than half made a complete recovery clinically. Abnormal electrophysiological findings persisted in some up to three years after the onset of symptoms even in the presence of clinical recovery (Smith and Trojaborg 1986). An example is shown in Fig. 9. These findings suggest that wallerian degeneration is a common feature of peroneal mononeuropathies in agreement with the findings of others (Singh et al. 1974, Kitirji and Wilbourn 1988) and are thus

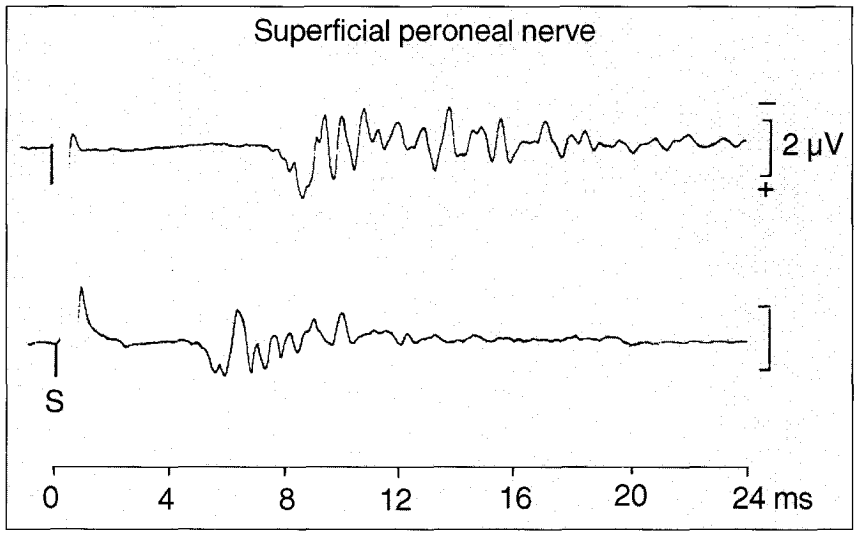

Fig. 9. Sensory nerve action potentials recorded over the superficial peroneal nerve below (lower trace) and above the fibular head (upper trace) after stimulation of the nerve proximal to the superior extensor retinaculum. Each of the responses represents the average of 500 stimuli. The conduction velocity in the segment from the site of stimulation (S) to below the fibular head was 50 m/sec, in between the two recording sites 36 m/sec. From a 36-year-old man with a cross-leg peroneal compression palsy of 4 months' duration.

compatible with findings in ulnar nerve pressure palsies at the elbow (Payan 1969, Nielsen et al. 1980).

Polyneuropathies:

Different types of polyneuropathy have been intensively explored using near-nerve electrode technique for stimulation and recording of sensory potentials (Lamontagne and Buchthal 1970, Buchthal and Rosenfalck 1971b, Buchthal 1973, Behse et al. 1972, 1977, Nielsen 1973b, 1974, Behse and Buchthal 1977a, 1978, Buchthal and Behse 1977, 1978, Tackmann and Winkenberg 1977, Boysen et al. 1979, Trojaborg 1981, Daugaard et al. 1987, Hansen et al. 1989, Shefner et al. 1991a).

As to polyneuropathies in general, it is more common to find abnormalities of conduction in sensory than in motor fibres (Bannister and Sears 1962, Lamontagne and Buchthal 1970, Buchthal and Rosenfalck 1971b). Moreover, sensory fibres are more apt to be affected earlier than motor fibres; increased temporal dispersion causing changes in the shape of the SNAP is easier to detect than minimal changes in evoked compound motor action potentials (Gilliatt and Willison 1962, Buchthal and Rosenfalck 1966).

Compared to conventional techniques, near-nerve electrode recording is far superior. For instance, SNAPs were absent in 40% of patients with neuropathy using surface recording (Gilliatt and Sears 1958, Downie and Newell 1961, Liberson 1963, Mayer 1963, Kaeser 1966, Fullerton and O'Sullivan 1968, Chopra and Hurwitz 1969) compared with 1% when using near-nerve technique (Buchthal and Rosenfalck 1971).

Axonal neuropathies (alcoholic, diabetic and uraemic):

Mild slowing of SCV correlates well with loss of the largest fibres and is associated with a reduced SNAP amplitude. In some diabetics, however, SCV might be slower than predicted from biopsy findings (Behse et al. 1977). The disproportional slowing was not due to myelin damage and in experimental diabetes no explanation was found for the reduced MCV in morphological terms (Sharma and Thomas 1974, Sharma et al. 1976). In uraemic polyneuropathy, besides axonal damage there might be additional impairment of nerve conduction caused by a toxic component, considering the fast recovery after successful kidney transplantation (Nielsen 1974). Similarly, in diabetes, recovery of the neuropathy, clinically and electrophysiologically, has been described after pancreas and kidney transplantation and is probably not related to nerve regeneration, taking the time relation into account (Kennedy et al. 1990). If kidney transplantation only is performed, the effect on the neuropathy is less successful, as illustrated in Fig. 10.

Hereditary neuropathies:

The electrophysiological hallmark of the hypertrophic type of Charcot Marie Tooth's disease (HMSNI) is the markedly reduced maximum CV, which is usually below 30 m/s and accounted for by uniform demyelination of all nerve fibres combined with axonal degeneration (Dyck and Lambert 1968, Buchthal and Behse 1977). SNAPs are usually absent with surface electrode recordings (Dyck and Lambert 1968) but were present in all the cases studied by Buchthal and Behse (1977) using near-nerve electrode technique.

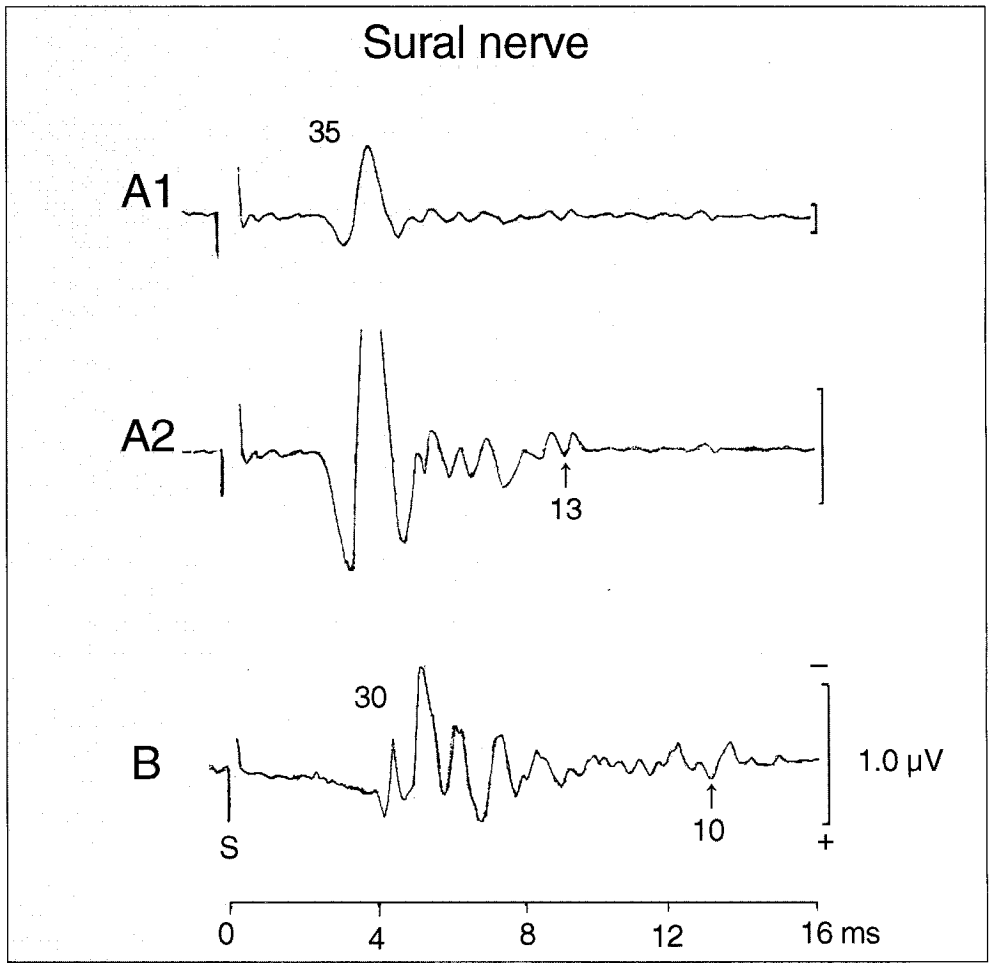

Fig. 10. Sensory nerve action potentials (SNAP) from a 50-year-old man with sensori-motor polyneuropathy. Since the age of 11 he had suffered from insulin-dependent diabetes mellitus. The sural nerve was stimulated at the lateral malleolus (S) and the SNAP was recorded from the mid-calf 120 mm proximal to the site of stimulation. The first recording (A1) at age 46 revealed a reduced maximum conduction velocity (CV), 32% reduced compared with the normal mean for age; the SNAP amplitude was 2.8 μV, 72% reduced. Minimum CV was determined by averaging the responses to 500 stimuli (A2) at an amplification four times higher than A1. The figures above and below the traces indicate the CV in m/sec in the fastest and slowest conducting fibres, respectively. The bottom trace (B) shows the SNAP recorded four years later after a successful kidney transplant had been performed. Nevertheless, there was deterioration of the neuropathy clinically, more pronounced CV slowing and further reduction of SNAP amplitude. Minimum CV still within the normal range.

In the axonal type (HMSNII), the recorded SCV in the sural nerve is equal to the velocity predicted from the histogram of fibre diameters from biopsy of the same nerve, i.e. slowing can be explained by the presence of axonal degeneration. It is noteworthy that a decrease in sural nerve CV from the normal 53 m/s to 30 m/s can be accounted for solely by loss of the largest diameter fibres (Behse and Buchthal 1977b).

Only when SCV is slowed to less than 60% of normal is it justified to assume demyelination as the cause of slowing (Behse and Buchthal 1978). Using surface recording, SNAPs were absent in 50% of the patients with HMSNII (Dyck and Lambert 1968), whereas a potential was present in all the cases studied by Buchthal and Behse (1977).

Sensory neuropathy:

The syndrome of acute sensory neuropathy has been reviewed recently (Windebank et al. 1990). Electrophysiological testing typically showed an absence of sensory potentials. Sural nerve biopsies revealed loss of large myelinated fibres and axonal atrophy.

One such case has been studied using near-nerve technique (Buchthal and Rosenfalck 1971b). The amplitude of the desynchronized SNAP recorded over the median nerve was only 1% of normal and was the same at wrist and elbow, indicating that the separate components arose from single nerve fibres. Another case studied recently is illustrated in Fig. 11.

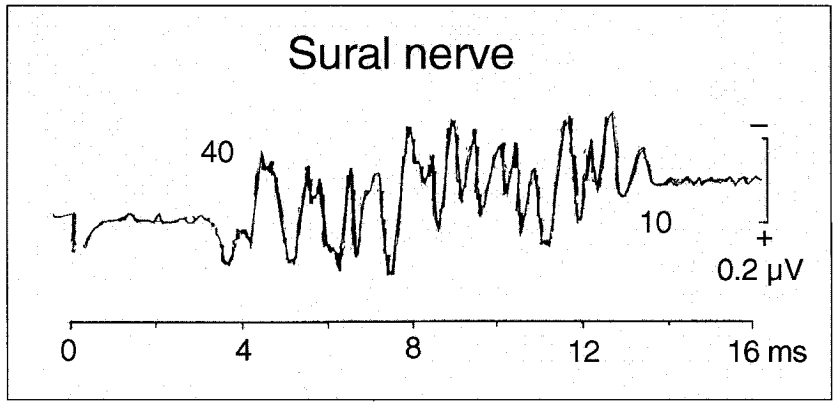

Fig. 11. Sensory nerve action potential from a 32-year-old woman with pure sensory neuropathy localized to the lower limbs of acute onset 2 months prior to the present recording. The sural nerve was stimulated at the lateral malleolus via needle electrodes (S), the evoked response was recorded at mid-calf through needle electrodes. The response represents the average of 500 stimuli. The 15 mA stimulus applied at the ankle was not perceived. The figures above and below the trace denote the maximum and minimum conduction velocity, respectively.

Minimum conduction velocity in neuropathies:

In axonal neuropathies with no or only minimal reduction of the maximum SCV, a reduction in minimum CV may be found, eventually as the first manifestation of abnormality (Behse et al. 1977, Tackmann and Minkenberg 1977). In a study of patients with peripheral nerve disorders of various aetiology small components of the SNAP in the median nerve were conducted significantly slower than in control nerves (normal mean 20.5 m/s, SD 3.1 m/s, Tackmann and Minkenberg 1977). In 13 out of 60 median nerves (22%) they found that the SCV and the SNAP amplitude of the main component were within the normal range. Similarly, they found slower than normal minimum CV in 48 sural nerves among which 14 (29%) had normal maximum CV and normal SNAP amplitude of the main component.

In a recent study of 102 patients with polyneuropathy, 32 (31%) had a normal maximum SCV but a reduced minimum CV. In 28 patients (27%) both maximum and minimum CV were slowed (Shefner et al. 1991a). Moreover, patients with symptoms of neuropathies but lacking signs clinically were the most likely to have isolated abnormalities in minimum CV. Among 45 patients with focal neuropathies one-fourth had a normal main component of the SNAP and normal maximum CV but a reduced minimum CV. Slowed minimum CV can be expected to be present in neuropathies in which regeneration of nerve fibres occurs. According to sural nerve biopsy findings, presence of regeneration is common in diabetic neuropathy and can account for the finding of late SNAP components. On the other hand, in alcoholic neuropathy histological signs of regeneration are rare and so are the very slow SNAP components (Behse and Buchthal 1977a). In accordance with this, Shefner et al. (1991a) found a normal minimum CV in 4 of their 5 patients with alcoholic neuropathy. They suggested that slowed minimum SCV reflecting regeneration could perhaps explain dysaesthetic nerve pain, which is a common complaint in diabetic neuropathy.

In the context of minimum sensory conduction velocity, it is interesting that in motor neuron disease in which sensory function is thought to be normal there may be electrophysiological abnormalities. Thus, Shefner et al. (1991b) found a reduction of the minimum SCV in half of their 18 patients with ALS although maximum SCV and SNAP amplitude were normal. Moreover, repeated determinations at 3-month intervals confirmed the findings and illustrated the stability over time and the reliability of the minimum CV measurements. The findings were interpreted as indicating prominent nerve fibre regeneration, suggesting that a dying back axonopathy affecting sensory nerves is present together with the neuronopathy.

Segmental demyelinating neuropathies:

Two entities are distinguished clinically: an acute, the Guillain-Barre syndrome (GBS) and a chronic inflammatory demyelinating polyneuropathy (CIDP). Electrophysiologically they are indistinguishable (Donofrio and Albers 1990).

Moreover, electrodiagnostic differences have not been identified between the relapsing or stepwise progressive forms of CIPD (Prineas and McLeod 1976). In some patients primary demyelination is the dominating feature, in others axonal degeneration, but in most there is a combination of both (Brown and Feasby 1984a,b). Whether one or the other, it will be reflected in the electric changes. The characteristic findings in GBS are the increased temporal dispersion of evoked action potentials and the evidence of a partial conduction block, the latter being best demonstrated in motor nerve fibres (Lewis and Sumner 1982).

In GBS, motor fibres are clinically more involved than sensory fibres and evoked SNAPs may be entirely normal in patients with prominent motor abnormalities (Arnason 1975, Kennedy et al. 1978, Soffer et al. 1978). However, in about half the cases SNAPs are absent (Eisen and Humphrey 1974),

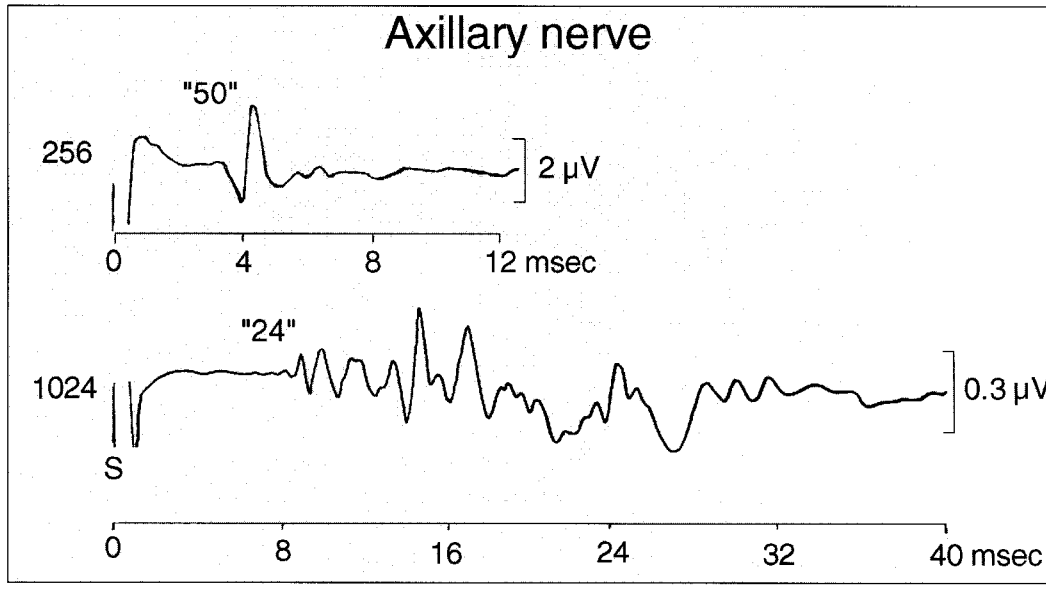

Fig. 12. Sensory nerve action potentials recorded at the supraclavicular fossa after stimulation of sensory fibres of the axillary nerve in the deltoid region. Above, from a 31-year-old normal subject; below, from a 19-year-old patient with an upper trunk lesion of the brachial plexus. The upper trace is the average of 256 responses, the lower that of 1024. Note the different time scales for upper and lower traces. The figures above the traces denote the approximate conduction velocity in m/sec between the site of stimulation and recording.

or of low amplitude and conducted at a slow rate (McLeod 1981). Unlike motor abnormalities, sensory changes are often patchy i.e. normal in one, abnormal in another nerve. Thus, abnormal sensory conduction along the median nerve but normal in the sural nerve was found in 42% of 86 patients with GBS, whereas the opposite never occurred (Albers et al. 1985).

In parallel with the findings of increased F wave latencies in the early stage of GBS pointing to a more proximal lesion in some patients (Kimura and Butzer 1975, Kimura 1979), a proximal delay between Erb's point and the spinal cord was found in 10 out of 11 patients within 2 weeks of the onset of paralysis using sensory evoked potential techniques (Brown and Feasby 1984b).

Sensory conduction in regenerating nerve fibres:

Changes in motor conduction during recovery from partial or total nerve interruption were first reported by Hodes et al. (1948). Information as to recovery in sensory fibres was obtained when averaging techniques became part of electrodiagnostic procedures (Trojaborg 1970). In man the rate of nerve growth has been estimated from the recovery time of SNAPs after complete nerve degeneration to be 1.0-2.0 mm per day (Trojaborg 1970, 1976, 1977a, Buchthal et al. 1975, Buchthal and Kuhl 1979, Trojaborg and Kuhl 1979). Over a distance of about 160 mm, a SNAP could be recorded four months after nerve suture of the median at wrist, taking advantage of the high resolution obtained by near-nerve recording and averaging technique (Buchthal and Kuhl 1979). For comparison, a SNAP could not be discriminated 6-11 months after traumatic radial nerve injury using surface recording (Downie and Scott 1964) nor was a sensory potential detected before 10 months after nerve suture at wrist (Ballantyne and Campbell 1973).

Similarly, after sural nerve grafting it took 18 months before a SNAP could be recorded with surface electrodes (Tallis et al. 1978).

Early on in the course of regeneration the SNAP is markedly dispersed, containing 20-40 components and during ongoing recovery the potential may consist of up to 60 components. An example of early recovery is shown in Fig. 12 and of a later recovering SNAP in Fig. 13.

The maximum rate of the SCV increased rapidly at first, about 3% per month, similar to the conduction in motor fibres, but then slowly at a rate of 0.3% per month. With increasing velocity of the maximum CV, the slowly conducted components also conducted faster, but additional slow components not seen before appeared (Buchthal and Kuhl 1979). Even 60 months after injury the SCV of the fastest fibres had not yet reached more than about 80% of normal, as illustrated in Fig. 14.

The properties of the fastest growing fibres are reflected in the change of the maximum SCV, i.e. a small proportion of myelinated fibres, whereas the SNAP amplitude is a measure of the number of fibres > 7 μm in diameter contributing to its main phase in normal nerve and in nerves with predominant axonal involvement (Buchthal and Behse 1977, Behse and Buchthal 1978). On the other hand, in regenerating nerves the peak-to-peak SNAP amplitude is a poor measure of the number of contributing nerve fibres, as the potential contains so many components of an almost equal amplitude years after recovery. Thus, the amplitude of the first phases of the SNAP changes vary little with the passage of time (Ballantyne and Campbell 1973, Buchthal and Kuhl 1979, Trojaborg and Kuhl 1979). Therefore, the cumulative amplitude obtained by adding the amplitude of all the components of the potential is a

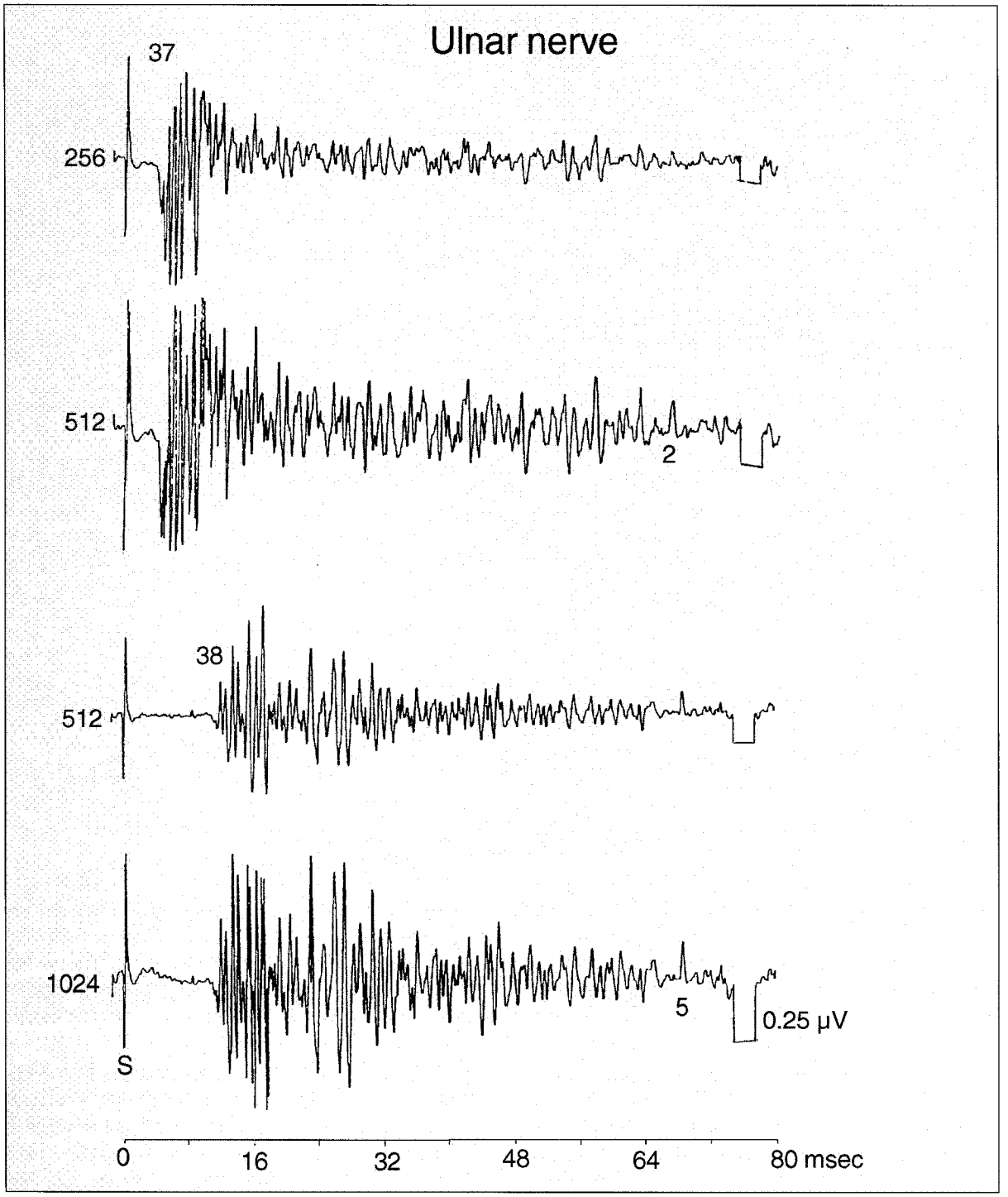

Fig. 13. Sensory nerve action potentials (SNAP) recorded over the ulnar nerve at wrist (upper 2 traces) and elbow proximal to the ulnar groove (lower 2 traces) after stimulation of digital nerve fibres of digit V (S). Average of 256 and 512 responses (upper 2 traces, respectively) and 512 and 1024 responses (lower 2 traces, respectively) for identification of slowly conducted components. Figures above traces denote the conduction velocity (CV) of the largest fibres, those below the CV of the smallest. From a 50-year-old man who 53 months previous to the present recording suffered a severe brachial plexus injury causing paralysis and anaesthesia of the whole arm.

better gauge of the number of nerve fibres activated (Buchthal and Kuhl 1979). Thus, the cumulative amplitude of the ulnar nerve sensory potential recorded at wrist, shown in Fig. 13, is 22 μV, whereas the peak-to-peak amplitude of the largest phase of the main components is 1.6 μV. At the time when the first ulnar sensory potential was recorded 30 months after nerve injury, the cumulative amplitude was 4.0 μV.

Nerve regeneration and reinnervation after arm amputation and replantation were followed electrophysiologically in a 22-year old man. He was first examined 3 1/2 years after the accident and then three times over a one-year period (Krarup et al. 1990). Near-nerve recording technique was used and SNAPs were evoked by electrical and tactile stimulation. The compound sensory nerve action potentials were of low voltage

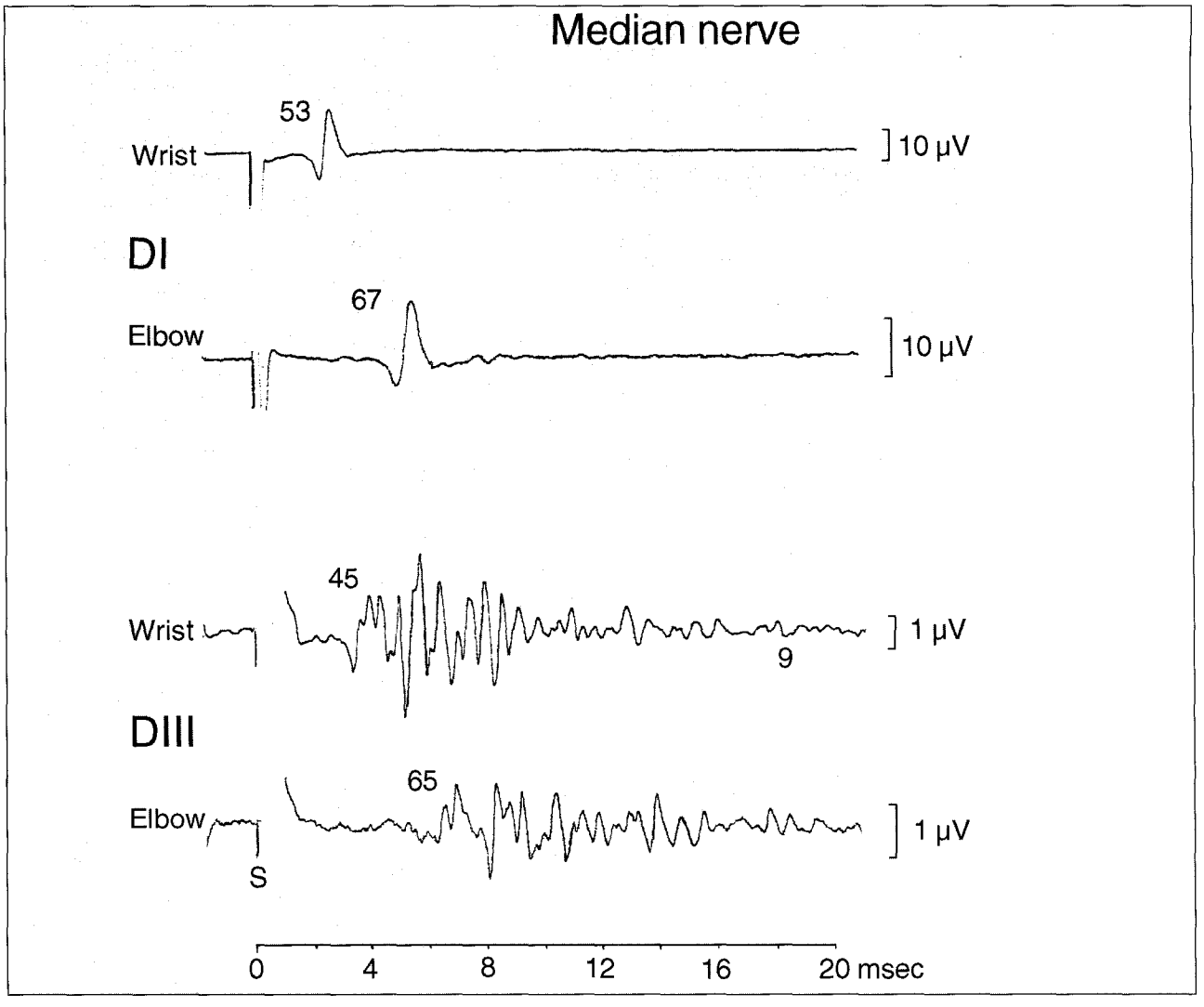

Fig. 14. Sensory nerve action potentials (SNAP) recorded over the median nerve at wrist and elbow following stimulation of sensory fibres of the thumb (DI, upper 2 traces) and middle finger (DIII, lower 2 traces). The figures above traces denote the conduction velocity in m/sec for successive nerve segments tested. From a 31-year-old woman who had had a cut over the median at wrist 5 years earlier. Note the normal SNAP from DI in contrast to the low voltage desynchronized prolonged SNAP from DIII, indicating regeneration of nerve fibres after an old lesion of the sensory branches to the middle finger.

and contained several components, the latency of the latest corresponding to a CV of 3-5 m/sec. The amplitude of motor and sensory potentials and their CV remained stable 3 1/2 – 4 1/2 years after injury indicating a steady state of regeneration and reinnervation. Amplitudes of SNAPs over the ulnar nerve, which had been repaired early by end-to-end juncture recovered by 25% compared with 1-5% for SNAPs evoked by stimulation of median nerve (DIII and DI, respectively) which was repaired seven months after injury by sural nerve grafting. Moreover, the study revealed evidence of aberrant regeneration and of abnormal connections between motor and sensory nerve fibres.

Tactile stimulation:

So far, all the information concerning SNAPs in different neurological disorders stems from electrical stimulation of peripheral nerves. However, this excludes the most distal part of the sensory neuron, the receptor. Using tactile stimulation, which is a more physiological way of studying the peripheral nervous system, information about the receptor and the most distal branches of the nerve can be obtained (Rosenfalck and Buchthal 1973). They showed that the SNAP evoked by tactile stimulation was conducted at a 10 m/s slower rate in the distal part of the median nerve than the potential evoked by an electrical stimulus. Moreover, the SNAP contained more components when evoked by tactile than by electrical stimuli. Fig. 15 shows SNAPs recorded over the median nerve at wrist and elbow after tactile stimulation and for comparison by electrical stimulation at the most distal site of the middle finger, the pulpa. The tactile stimulation was performed according to the method described by Buchthal (1980, 1982a,b); those interested in the technique are referred to the outstanding articles from 1982 published in Acta Physiologica Scandinavia.

The density of mechanoreceptors in the cutaneous area on the lateral site of the foot supplied by the sural nerve is 15-20 units per square cm, i.e. one-tenth of the density on the tip of the fingers. The SNAP evoked from the cutaneous areas of the sural or median nerves consists of 5-10 components with an

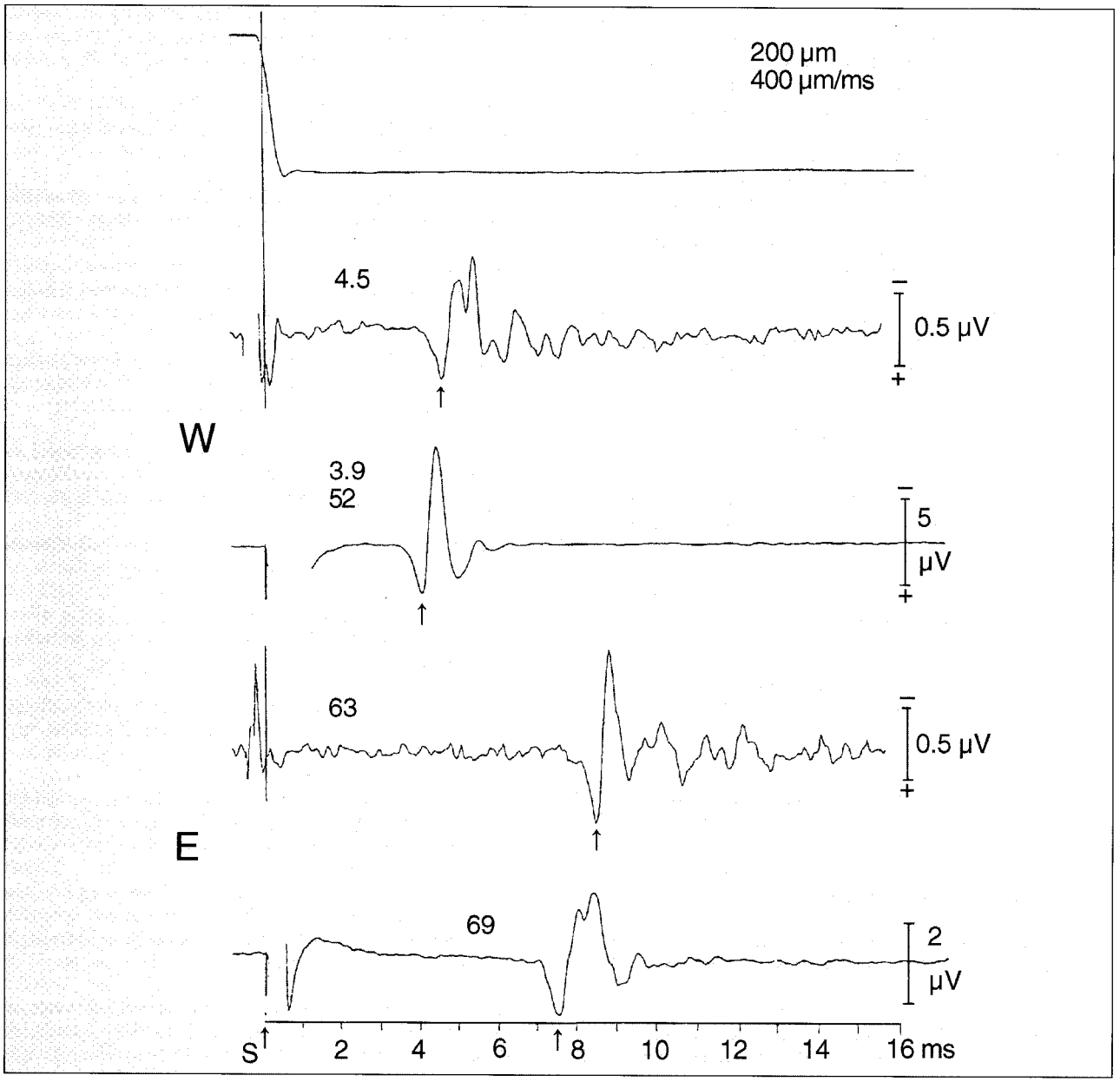

Fig. 15. Sensory nerve action potentials (SNAP) from a 25-year-old normal subject recorded over the median nerve at wrist (W) and elbow (E) after tactile and electrical stimulation (S) of the middle finger, respectively. Upper trace: depth of indentation and speed of velocity caused by the tactile probe during stimulation of the pulpa of DIII. The responses to 512 tactile stimuli are shown in traces 2 (W) and 4 (E), those to 128 electrical stimuli applied at the same site as the tactile are shown in traces 3 (W) and 5 (E). The latency to the first positive peak of the SNAP evoked by tactile stimulation was 0.6 msec longer than that evoked by electrical stimulation; the difference corresponds to the receptor delay. The figures above the bottom 2 traces indicate the conduction velocities between wrist and elbow.

amplitude of 0.3-1.0 µV in normal subjects (Buchthal 1980, Krarup and Trojaborg, to be published).

It is likely that the method could be useful in early detection of abnormalities in distal axonal lesions. Thus, in some patients with doubtful signs of diabetic neuropathy, SCV and amplitude of SNAPs evoked by tactile stimulation of the sural nerve were reduced whereas these parameters were normal using electrical stimuli (Buchthal 1980).

In a recent study of 26 male patients with cisplatin-induced neuropathy, tactile stimulation was used and SNAPs were recorded at two sites along the sural and median nerves and compared with those evoked by electrical stimulation applied at the same site as the tactile (Krarup et al. 1991). Half of the patients had electrophysiological signs of sensory neuropathy, most pronounced in those treated with high doses of cisplatin. Nevertheless, in all but one the response to tactile stimulation was within the normal range.

So far, too few studies are available to assess the value of tactile stimulation, a procedure which is tedious and time-consuming and not readily applied in routine studies. However, as a research tool it might be useful in the study of axonal neuropathies in which the routine study of sensory function fails to reveal abnormalities.

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Maximum Conduction along Sensory and Motor Nerve for Normal Subjects of Different Age[3]

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Median Nerve

Sensory Velocity					Amplitude of Sensory Potential					
	distance cm	mean m/sec	95% limits lower	upper	age years	mean µV	95% limits lower	upper		n
from:		57	47	66	15-24	43	17	106	stimulus:	25
DIGIT I	13	55	46	64	25-34	37	15	94	to DIGIT I	18
to:	SD = 1.3	54	44	63	35-44	33	13	82	recorded:	35
WRIST	9-17	52	43	61	45-54	29	12	70	at WRIST	45
		51	41	60	55-64	25	10	62		38
		49	40	58	65-74	22	9	52		16
		48	38	57	75-84	19	8	46		7
from:		70	62	79	15-24	12	5	31	stimulus:	14
WRIST	24	68	59	77	25-34	10	4	26	to DIGIT I	13
to:	SD = 2.4	66	57	74	35-44	9	3.5	22	recorded:	15
ELBOW	18-30	64	55	72	45-54	7.5	3	19	at ELBOW	22
		61	53	70	55-64	6.5	2.5	16		9
		59	51	68	65-74	5.5	2	14		5
		57	48	65	75-84	4.5	2	12		6
from:		64	55	73	15-24	16	7	35	stimulus:	34
DIGIT III	18	62	53	71	25-34	14	6.5	31	to DIGIT III	21
to:	SD = 1.5	60	51	69	35-44	13	6	27	recorded:	35
WRIST	13-21	58	49	67	45-54	11	5	25	at WRIST	46
		57	48	66	55-64	10	4.5	22		40
		55	46	64	65-74	9	4	20		16
		53	44	62	75-84	8	3.5	17		7
from:		71	62	79	15-24	7.5	3	19	stimulus.	14
WRIST	24	68	60	77	25-34	7	2.5	17	to DIGIT III	14
to:	SD = 2.3	66	57	75	35-44	6	2.5	15	recorded:	15
ELBOW	18-30	64	55	73	45-54	5	2	13	at ELBOW	22
		62	53	71	55-64	4.5	2	12		9
		60	51	68	65-74	4	1.5	11		6
		57	49	66	75-84	3.5	1.5	9		5
from:		63	56	69	25-34	25	13	49	stimulus:	3
DIGIT III	10	61	54	68	35-44	24	12	46	to DIGIT III	3
to:	SD = 1.2	59	53	66	45-54	22	11	44	recorded:	9
PALM	8-13	58	51	64	55-64	21	10	42	at PALM	5
from:		64	54	73	25-34					3
PALM	8	62	52	71	35-44					3
to:	SD = 1.3	60	51	69	45-54					9
WRIST:	6-10	58	49	67	55-64					5

[3]In: Electromyography – Sensory and Motor Conduction, Rigshospitalet, Copenhagen, 1975: 10-22. (Reproduced with permission).

Radial Nerve

Sensory Velocity						Amplitude of Sensory Potential				
	distance cm	mean m/sec	95% limits lower	upper	age years	mean µV	95% limits lower	upper		n
from:		65	57	73	15-24	35	13	94	stimulus:	22
WRIST	23	64	57	72	25-34	34	12	90	at WRIST	15
to:	SD = 2.3	63	56	71	35-44	33	12	88	recorded:	12
ELBOW	17-32	63	55	70	45-54	31	12	84	at ELBOW	21
		62	54	69	55-64	30	11	82		12
		61	54	69	65-74	29	11	80		4
from:		69	62	76	15-24				stimulus:	8
ELBOW	15	67	60	74	25-34				at WRIST	5
to:	SD = 1.9	66	59	73	35-44	10	4.5	24	recorded:	4
AXILLA	11.21	65	58	72	45-54				at AXILLA	11
		64	57	71	55-64					3

Musculocutaneous Nerve

Sensory Velocity						Amplitude of Sensory Potential				
	distance cm	mean m/sec	95% limits lower	upper	age years	mean µV	95% limits lower	upper		n
from:		68	61	75	15-24	36	17	75	stimulus:	15
ELBOW	19	66	59	73	25-34	34	16	72	at ELBOW	8
to:	SD = 2.2	64	57	71	35-44	33	16	69	recorded:	8
AXILLA	13-24	62	55	69	45-54	31	15	65	at AXILLA	13
		60	53	67	55-64	29	14	62		10
		58	51	65	65-74	28	13	59		6
from:		68	59	76	15-24	11	3.5	30	stimulus:	14
AXILLA	19	65	57	74	25-34	9	3	25	at ELBOW	6
to:	SD = 1.8	63	54	71	35-44	7	2.5	21	recorded:	7
ERB's POINT	14-24	60	52	69	45-54	6	2	18	at ERB's	10
		58	49	66	55-64	5	2	15	POINT	9
		55	47	64	65-74	4	1.5	12		4

Axillary Nerve

Sensory Velocity						Amplitude of Sensory Potential				
	distance*) cm	mean m/sec	95% limits lower	upper	age years	mean µV	95% limits lower	upper		n
from:		46	36	56	15-24	1.7	0.8	3.5	stimulus:	3
REGIO	18	44	35	54	25-34	1.4	0.7	3.0	REGIO	10
DELTOIDEI	SD = 1.9	43	33	53	35-44	1.2	0.6	2.5	DELTOIDEI	5
to: ERB's	14-24	42	32	51	45-54	1.0	0.5	2.1	(SUBCUTANEOUS)	3
POINT		40	30	50	55-64	0.9	0.4	1.8	recorded: at ERB's POINT	5

*) measured with obstetric calipers.

Ulnar Nerve

Sensory Velocity						Amplitude of Sensory Potential				
	distance cm	mean m/sec	95% limits lower	upper	age years	mean µV	95% limits lower	upper		n
from:		59	48	70	15-24	17	7	43	stimulus:	21
DIGIT V	14	58	47	69	25-34	16	6	40	to DIGIT V	15
to:	SD = 1.3	57	46	68	35-44	14	5.5	36	recorded:	23
WRIST	10-18	55	44	66	45-54	13	5	33	at WRIST	54
		56	47	65	55-64	10	4	26		56
		52	43	61	65-74	7	2.5	17		25
		49	40	58	75-84	5	2	12		8
from:		72	63	82	15-24	9	4	20	stimulus:	10
WRIST	20	70	61	80	25-34	8	3.5	19	to DIGIT V	12
to: 5 cm	SD = 2.3	69	60	78	35-44	7.5	3.5	18	recorded:	14
DISTAL to	14-27	67	58	76	45-54	7	3	17	at 5 cm DISTAL	15
SULCUS		67	57	76	55-64	4.5	2	10	to SULCUS	18
N. ULNARIS		63	54	73	65-74	3	1.5	7	N. ULNARIS	11
"across"*)	10.2	63	49	**) 72	15-24					10
SULCUS	SD = 1.4	61	49	72	25-34					12
N. ULNARIS	8-13	60	49	72	35-44					14
		58	49	72	45-54					15
		57	44	64	55-64					18
		52	44	64	65-74					11
from:		67	58	77	15-24	5.5	2	14	stimulus:	10
WRIST	30	66	57	75	25-34	5.5	2	14	to DIGIT V	12
to: 5 cm	SD = 3.0	65	56	74	35-44	5.5	2	14	recorded:	14
PROXIMAL	23-36	64	54	73	45-54	5.5	2	14	at 5 cm	15
to SULCUS		62	55	69	55-64	3.5	1.5	9	PROXIMAL	18
N. ULNARIS		58	51	65	65-74	3	1	7	to SULCUS	13
		53	46	61	75-84	2.5	1	6	N. ULNARIS	6

*) from: 5 cm distal to sulcus n. ulnaris to 5 cm proximal to sulcus n. ulnaris.
**) 95% limits determined from the cumulated distribution of velocities.

Sural Nerve

Sensory Velocity						Amplitude of Sensory Potential				
	distance cm	mean m/sec	95% limits lower	upper	age years	mean µV	95% limits lower	upper		n
from:		51	41	60	15-24	5	1.1	22	stimulus:	16
DORSUM	11	50	41	59	25-34	4.5	1.	19	at DORSUM	1
PEDIS	SD = 1.4	49	40	59	35-44	4	0.9	17	PEDIS	7
to:	7-13	49	40	58	45-54	3.5	0.8	15	recorded:	8
LATERAL		48	39	57	55-64	3	0.7	13	at LATERAL	3
MALLEOLUS		48	38	57	65-74	2.5	0.6	11	MALLEOLUS	2
from:		55	48	62	15-24	14	5.5	36	stimulus:	28
LATERAL	15	55	48	62	25-34	13	5.0	33	at LATERAL	11
MALLEOLUS	SD = 1.6	54	47	61	35-44	12	4.5	30	MALLEOLUS	14
to:	12-19	54	47	61	45-54	10	4.0	27	recorded:	22
"SURA"		53	46	60	55-64	9	3.5	24	at "SURA"	9
		53	46	60	65-74	8	3.5	22		9

Posterior Tibial Nerve

Sensory Velocity						Amplitude of Sensory Potential				
	distance cm	mean m/sec	95% limits lower	upper	age years	mean µV	95% limits lower	upper		n
from:		46	39	53	15-24	3	0.5	14	stimulus:	22
TOE I	21	45	38	52	25-34	2	0.3	10	to TOE I	3
to:	SD = 1.5	44	38	51	35-44	1.5	0.2	7	recorded:	4
MEDIAL	18-25	44	37	51	45-54	1	0.2	5	at MEDIAL	6
MALLEOLUS		43	36	50	55-64	0.7	0.1	3.5	MALLEOLUS	3
from:		56	50	63	15-24	0.9	0.2	4	stimulus:	20
MEDIAL	45	56	49	62	25-34	0.8	0.2	3.5	to TOE I	2
MALLEOLUS	SD = 2.6	55	49	62	35-44	0.6	0.1	2.5	recorded:	4
to: POPLITEAL FOSSA	39-50	55	48	61	45-54	0.5	0.1	2	at POPLITEAL FOSSA	3

Saphenous Nerve

Sensory Velocity						Amplitude of Sensory Potential				
	distance cm	mean m/sec	95% limits lower	upper	age years	mean µV	95% limits lower	upper		n
from:		60	54	67	15-24	1.5	0.4	7	stimulus:	15
MEDIAL	41	58	52	65	25-34	1.3	0.3	6	at MEDIAL	7
EPICONDYLE	SD = 3.0	57	50	63	35-44	1.1	0.3	5	EPICONDYLE	3
to:	34-47	55	48	61	45-54	0.9	0.2	4	recorded:	3
INGUINAL		53	47	60	55-64	0.8	0.2	3	at INGUINAL	5
LIGAMENT		51	45	58	65-74	0.7	0.2	3	LIGAMENT	1

Peroneal Nerve

Sensory Velocity						Amplitude of Sensory Potential				
	distance cm	mean m/sec	95% limits lower	upper	age years	mean µV	95% limits lower	upper		n
from:		57	49	64	15-24	4.5	1.2	17	stimulus:	20
RETINACULUM	29	56	48	63	25-34	4	1	15	RETINACULUM	9
SUPERIOR	SD = 3.1	55	48	62	35-44	3.5	0.9	13	SUPERIOR	16
to: 2 cm	22-35	54	47	62	45-54	3	0.8	11	recorded: 2 cm	13
DISTAL to		54	46	61	55-64	2.5	0.7	10	DISTAL to	13
CAP. FIBULAE		53	45	60	65-74	2	0.6	8	CAP. FIBULAE	6
"across"*)	11.3	55	48	63	15-24					
CAPITULUM	SD = 1.4	55	47	62	25-34					
FIBULAE	9-14	54	47	61	35-44					
		53	46	61	45-54					
		53	45	60	55-64					
*) from 2 cm distal to capitulum fibulae to 9 cm proximal to capitulum fibulae.										
from:		56	49	63	15-24				stimulus:	14
RETINACULUM	39	56	49	63	25-34				RETINACULUM	3
SUPERIOR	SD = 3.5	55	48	62	35-44	3.5	1.2	9	SUPERIOR	9
to: 9 cm	32-40	55	47	62	45-54				recorded: 9 cm	6
PROXIMAL to CAP. FIBULAE		54	47	61	55-64				PROXIMAL to CAP. FIBULAE	7

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