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Combined Recordings of Compound Nerve Action Potentials and Spinal Cord Evoked Potentials in Differential Diagnosis of Spinal Root Lesions

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Summary. Three cases are presented to demonstrate the diagnostic value of sensory neurography in combination with somatosensory evoked potentials in the diagnosis of proximally located neuropathy and its differentiation to centrally located demyelinating processes. Simultaneous recording of cortical and spinal evoked potentials, as well as peripheral nerve action potentials, revealed in two cases (herpes zoster, Guillain-Barré syndrome) a site of lesion at the spinal roots suggesting radiculitis and in one case (tick bite) a site of lesion central to the source of lumbar evoked potentials suggesting myelitis. In all cases almost complete recovery of sensory conduction velocities suggests a complete repair myelination not previously described.

Key words: Somatosensory evoked potential – Sensory nerve – Action potential – Demyelination – Radiculitis – Myelitis

Introduction

Satisfactory information concerning the site and extent of a localized peripheral nerve lesion can in most cases be drawn from fractionated measurements of sensory and motor conduction velocities. In contrast lesions of the spinal roots have hitherto, not been directly accessible to electrophysiological measurements, with reflex measurements (H reflex, F wave) only allowing indirect assessment of the conduction properties of the proximal segments (Kimura and Butzer 1975; King and Ashby 1976; Lachman et al. 1980). The latter procedures, however, do not allow for a differentiation between lesions of the spinal roots, the plexus, and proximal parts of peripheral nerves. A more refined method, which reveals lesions of the spinal roots is the simultaneous recording of sensory action potentials from single nerves and spinal evoked potentials following afferent nerve stimulation. For illustration three cases will be reported that demonstrate the

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power of the method to localize a lesion within the spinal roots and to differentiate it from a lesion within the cervical or lumbosacral plexus, or myelopathy (Stöhr et al. 1982a).

Methods

Stimulation was performed using 0.1 ms square wave pulses delivered at a rate of 5 Hz to the wrist (median nerve) or the ankle (posterior tibial nerve) and in two cases through ring electrodes around digit II and III for purely sensory stimulation of the median nerve. Stimulus strength was adjusted to just below the threshold for pain (at least three times sensory threshold) for pure sensory stimulation, and 4 mA above motor threshold for mixed nerve stimulation. Simultaneous recordings were accomplished by platinum needle electrodes (DISA 25 C 04) placed subcutaneously at the following sites: popliteal fossa, spinal processes L 5, L 1, and 3 cm caudal to Cz (C'z) with posterior tibial nerve stimulation; upper arm, Erb's point, spinal process C 7, and C 2, and 2 cm posterior to C_3 and C_4 (C_3' C_4') respectively with median nerve stimulation at the wrist. With finger nerve stimulation additional recordings were done from the wrist. The bandpass of the recording system was set to 10–20 Hz and 1 kHz; 2000–8000 periods were averaged. Latencies were measured by a system-integrated marker to the first positive peak of the evoked responses (see Fig. 1); the onset of N 11 (second negative peak at C 7; Figs. 2 and 3) was taken for the arrival of the impulse volley at the cervical cord (Desmedt and Brunko 1980).

Results

Figure 1 reveals typical evoked responses from the median nerve between wrist and Erb's point. It can be seen that the sensory nerve conduction velocity (NCV) increases from the distal to the proximal segment of the nerve (Fig. 4), a finding regularly seen in normals (Desmedt and Brunko 1980).

Case 1. Guillain-Barré syndrome in a 16-year-old male presenting with rapidly progressive weakness and development of flaccid tetraplegia which persisted for 4 months. Somaesthesia was only slightly affected with distally located hypaesthesia and hypalgesia. Examination of CSF exhibited 6/mm^3 cells, total protein 2800 mg/l, IgG 77.8 mg/l.

Median nerve stimulation (fingers II and III or wrist) evoked normal responses and revealed normal sensory NCV between the fingers and the axilla, but a considerably prolonged latency more rostrally. The most prominent findings were the severe attenuation of the neck somatosensory evoked potentials and a severe reduction in NCV between Erb's point and the cervical cord (23 m/s) suggesting a prominent demyelination largely confined to the nerve roots (Fig. 2a and b). Figure 4 illustrated the conduction velocities calculated from Fig. 2a and b and clearly demonstrates that impulse conduction was predominantly affected between Erb's point and the cervical cord. The delay of the first scalp-recorded negativity ("*N* 20") was due to demyelination of the primary sensory neuron, since the central conduction time was normal (actually 6.5 m/s, upper limit of *N* 13a-*N* 20 = 7.2 m/s, Stöhr et al. 1982b). A control examination 8 months later with almost complete remission of clinical symptoms revealed normal latencies and amplitudes of the SEP (Figs. 3 and 4).

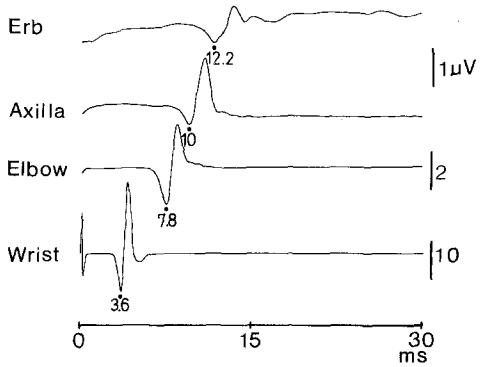

Fig. 1. Normal 31-year-old female. Stimulation at fingers II and III with ring electrodes. Simultaneous recording from wrist, elbow, axilla, Erb's point, and the spinous process C 7. NCV from distal to proximal, 51, 63, 76, 77, 79 m/s

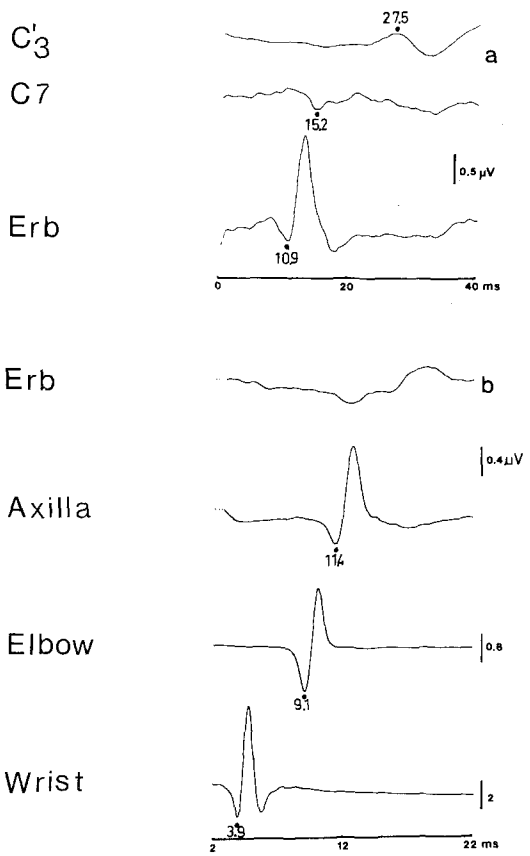

Fig. 2a, b. 16-year-old male with Guillain-Barré syndrome. **(a)** *(top)* Stimulation of the median nerve at the wrist and simultaneous recordings from Erb's point, the spinal process C 7 and the scalp at C' 3. Erb's point potential slightly attenuated and delayed. The onset of the cervical response (*N* 11) and the peak of the second negative component of the neck-SEP (*N* 13), as well as the scalp recorded potential (*N* 20) are severely attenuated and delayed. Central conduction time (*N* 13–*N* 20) is normal (6.5 m/s). **(b)** *(bottom)* Stimulation at fingers II and III: normal potentials above the median nerve at the wrist, elbow, and axilla; distorted and slightly attenuated potential at Erb's point

Case 2. Herpes zoster radiculitis C 5–Th 2 on the left in a 71-year-old female presenting with radicular pain and characteristic dermatological signs in C 5–Th 2, followed within a few days by Horner's syndrome, flaccid paralysis of the left arm, and severe disturbances of somaesthesia on the left. Somatosensory evoked potentials following median nerve stimulation (Fig. 5a) exhibited normal latencies and amplitudes on the right. On the left side the amplitude of Erb's

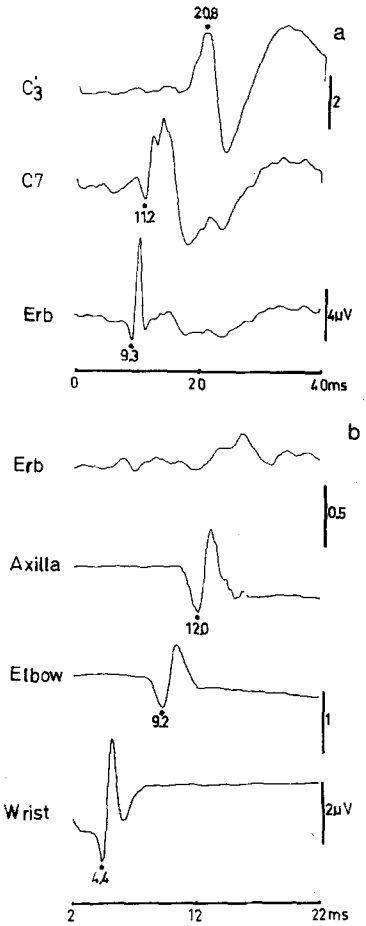

Fig. 3a, b. Same patient as in Fig. 2, 8 months later. **(a)** *(top)* Following median nerve stimulation at the wrist, somatosensory evoked potentials at Erb's point, C 7 and scalp (C_3') exhibit normal latencies and amplitudes. **(b)** *(bottom)* Normal NAPs following stimulation of the fingers II and III

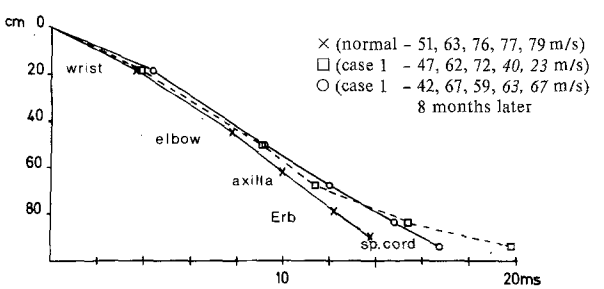

Fig. 4. Diagrammatic illustration of nerve conduction velocities in different segments between fingers II and III at zero *(ordinate)* and spinal cord at 94 cm. Latencies were measured at the wrist, elbow, axilla, Erb's point, and the spinal process C 7, respectively. In normals (×——×) NCV increases e.g. from 51 m/s distally to 79 m/s proximally. In case 1 with Guillain-Barré syndrome there is a reduction of NCV between axilla and Erb's point and most prominently between Erb's point and spinous process C 7 in the acute stage of the illness (□- - -□). This reduction of NCV recovered completely at the time of the second investigation (○- - -○)

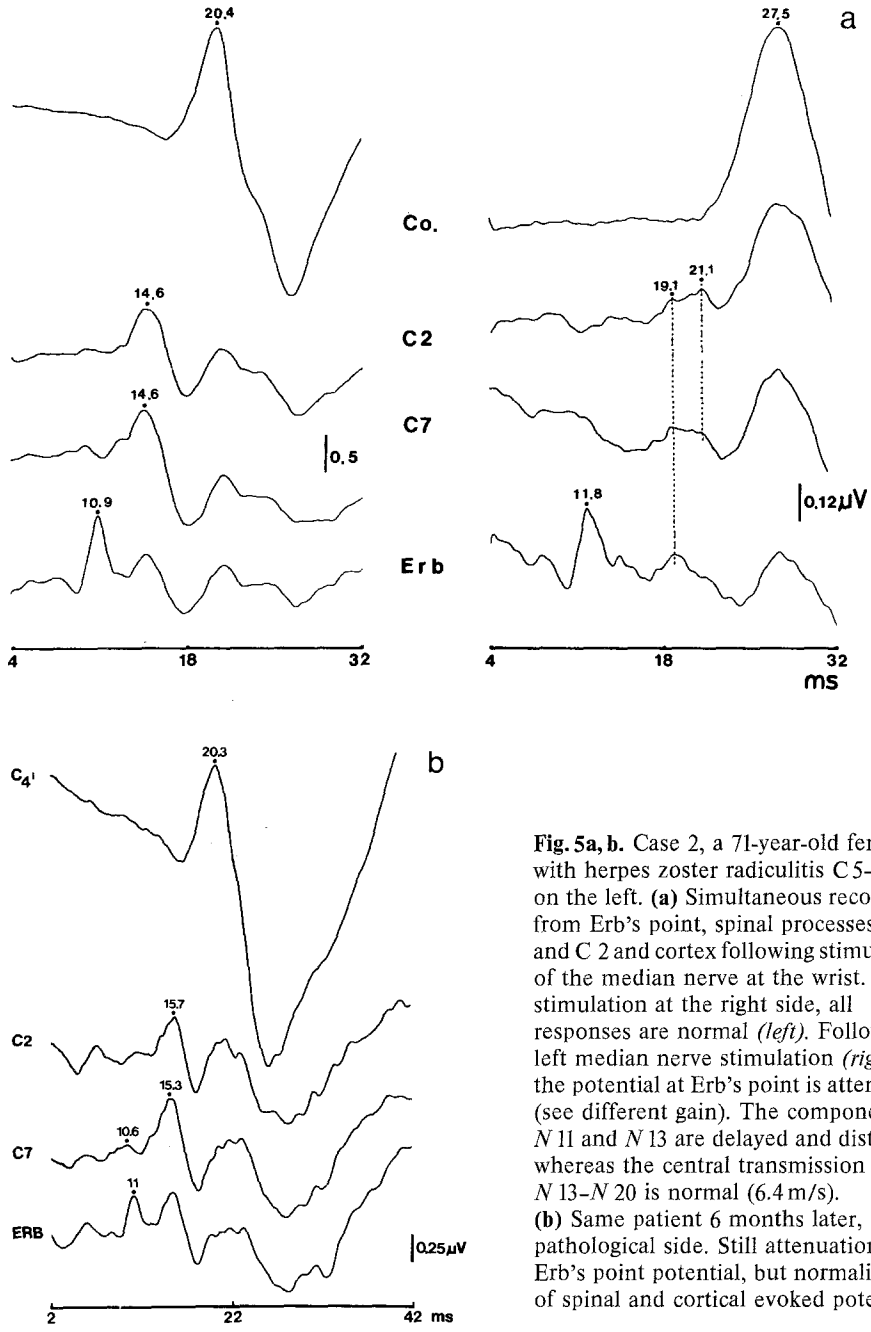

Fig. 5a, b. Case 2, a 71-year-old female with herpes zoster radiculitis C 5–Th 2 on the left. **(a)** Simultaneous recordings from Erb's point, spinal processes C 7 and C 2 and cortex following stimulation of the median nerve at the wrist. After stimulation at the right side, all responses are normal *(left)*. Following left median nerve stimulation *(right)*, the potential at Erb's point is attenuated (see different gain). The components *N* 11 and *N* 13 are delayed and distorted, whereas the central transmission time *N* 13–*N* 20 is normal (6.4 m/s). **(b)** Same patient 6 months later, pathological side. Still attenuation of Erb's point potential, but normalization of spinal and cortical evoked potentials

point potential was reduced to 25% as compared to the normal side indicating a moderate ganglionic (or infraganglionic) lesion (Jones 1979; Stöhr et al. 1981). Additionally, there was a severe delay of the cervical evoked response (*N*13) with an interpeak latency *N*9–*N*13 of 9.2 m/s, as compared to 3.7 m/s on the normal

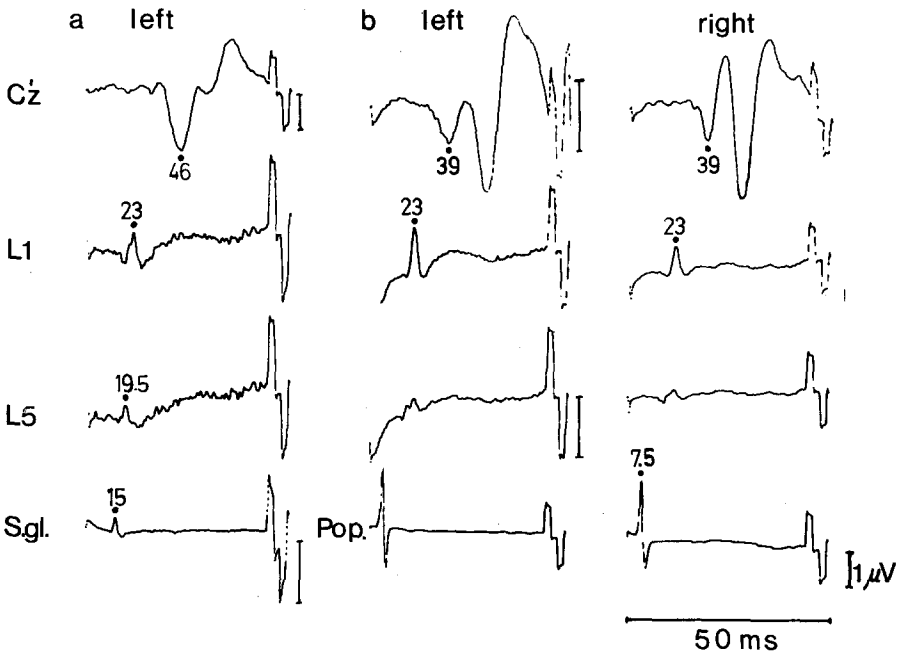

Fig. 6a, b. Case 3, a 25-year-old female with tick myelitis. Evoked potentials following tibial nerve stimulation at the ankle and simultaneous recording from the scalp (C_z'), above the lumbosacral cord (L 1), the cauda equina (L 5) and the sciatic nerve at the sulcus glutaeus (S. gl.) and at the poplitea (pop.) respectively. **(a)** Following stimulation at the involved left side the NAP (S. gl.), as well as the cauda and cord peak (L 5, L 1) are normal. The primary cortical response (P 40) is moderately delayed (46 ms, as compared to 39 ms at the normal side), but not attenuated. The interpeak latency between *N*20 (L 1) and P 40 exceeds the upper limit of normality (20.8 ms, Riffel and Stöhr 1982) by 2.2 ms. **(b)** Three weeks later complete recovery of the absolute latency of P 40 and of the central transmission time (*N*20–P 40 = 16 ms) occurred, whereas the amplitude of P 40 is about half of the normal side

side suggesting demyelination between the brachial plexus and the dorsal horn, assuming the dorsal horn as the generator site of *N*13, recorded above the spinous process C 7 (Desmedt and Chéron 1981; Stöhr et al. 1982b). The prolongation of the primary cortical potential (*N*20) to 27.5 m/s can be attributed to this peripherally located lesion, since the central conduction time is entirely normal (interpeak latency *N*13–*N*20 = 6.4 ms). A control examination after 6 months showed almost complete recovery of latencies, with a still moderate attenuation of the evoked potential amplitude (Fig. 5b).

Case 3. Tick myelitis in a 25-year-old female presenting with the diagnosis "polyradiculitis", and complaining of paraesthesia and hyperalgesia on the lower left limb. She had been bitten by a tick on the left leg 10 days previously with subsequent erythema. The results of neurological examination were normal. Somatosensory evoked potentials following stimulation of the left posterior tibial nerve (Fig. 6a) revealed a delayed cortical response (P 40 = 46 ms compared to 39 ms on the normal side), whereas latencies and amplitudes of the simultaneously recorded potentials over the popliteal fossa, the spinous processes L 5

and L 1 were normal. These findings point to a myelitis as the cause of the complaints instead of a radiculitis as tentatively diagnosed by the first examiner. A control somatosensory evoked potential 3 weeks later when the patient did not have any further complaints showed a complete normalization of P 40, without any side difference of latencies (Fig. 6b).

Discussion

Simultaneous recordings of spinal evoked potentials and of nerve action potentials (NAP) following distal stimulation of sensory or mixed nerves permit unequivocal localization of lesions to infraganglionic or supraganglionic segments of the afferent pathway. The diagnostic significance of this method has recently been discussed in relation to traumatic lesions of the brachial plexus (Jones 1979; Stöhr et al. 1981). In the majority of cases the lesion could be localized in the plexus, radices or a combined lesion at both sites could be proven. In supraganglionic lesions *N*11 and *N*13 as well as the scalp-recorded potentials are absent or attenuated and sometimes slightly delayed with a normal potential at Erb's point, whereas in infraganglionic lesions the potential at Erb's point is attenuated or absent, frequently with a relatively well preserved neck somatosensory evoked potential (Jones 1979; Stöhr et al. 1981). The same criteria hold in non-traumatic lesions of nerve plexus and nerve roots, respectively. In demyelinating processes additionally prolonged interpeak latencies indicate the site of a lesion between the presumed generator sites of the potentials. Additionally this method allows for a differentiation of peripherally and centrally located lesions. Normal spinal evoked potentials and NAPs and a prolonged central transmission time unequivocally prove localization of the lesion centrally from the spinal roots and rule out for example the tentative diagnosis of polyradiculitis as in our case 3.

The cases reported here showed evidence of demyelination in the proximal parts of the peripheral nerves, the spinal roots, or the dorsal columns respectively with a considerable reduction of impulse conduction velocity around the spinal roots (infraganglionic and especially supraganglionic, cases 1 and 2) or a delay of the central conduction time due to myelitis (case 3). To our knowledge neither in herpes zoster (case 2) nor in tick myelitis (case 3) has demyelination been reported. For herpes zoster a ganglionitis with oedema, haemorrhage and thrombotic plugs, and subsequent demyelination could account for the slow conduction velocity within the dorsal roots. In Guillain-Barré syndrome focal demyelination has been reported. In the case presented here (case 1), demyelination seemed to be restricted to the most proximal segments of the peripheral nervous system, especially to the nerve roots. Perhaps this localization accounts for the favourable outcome with complete recovery within 4 months following a 4-month period of tetraplegia. This favourable outcome is reflected in the complete normalization of neurophysiological measurements (Figs. 3 and 4). In cases 2 and 3, too, latencies of spinal cord and cortical evoked potentials became normal within 6 months and 1 month, respectively, pointing to an almost complete recovery of the myelin sheath. This finding suggests another type of repair

myelination in proximal segments of peripheral nerves and in central axons, as compared to distal parts of peripheral nerves, where even several years after an acute demyelinating disease (e.g. Guillain-Barré syndrome) low conduction velocities are reported in some of the patients (McLeod 1981; Eisen and Humphries 1974; McLeod et al. 1976).

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