

Electromyography and Clinical Neurophysiology

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Nerve conduction velocity in patients under long term treatment with antiepileptic drugs

K.-H. Krause and P. Berlit¹

Abstract

Motor and sensory nerve conduction velocity (NCV) of median nerve and motor NCV of peroneal nerve were measured in 548 epileptic patients, aged 20 to 40 years, under long term treatment with antiepileptic drugs. Compared with a control collective of 70 healthy persons in the same age the epileptics showed a reduction of all NCV's. 19 percent of the epileptic collective had at least 1 diminished NCV. Negative correlations with total amount and average daily dose of antiepileptic drugs were found in both sexes for sensory NCV of median nerve and motor NCV of peroneal nerve, only in males also for motor NCV of median nerve.

Only in females, duration of therapy correlated negative with sensory NCV of median and motor NCV of peroneal nerve. In patients under monotherapy the group with carbamazepine treatment showed the lowest NCV values, the difference being significant for motor NCV of median nerve in comparison to phenytoin and valproate sodium. It is concluded, that NCV generally is lowered under long term treatment with antiepileptic drugs and that phenytoin has no specific influence compared with the other drugs.

Introduction

A negative influence of phenytoin on the function of peripheral nerves has been discussed already in 1942 (10). Meanwhile a reversible lowering of nerve conduction velocity (NCV) has been proven in cases with acute phenytoin intoxication (1, 13, 18, 21). Concerning the possible implication of long term treatment the discussion is controversé: studies showed different results, the frequency of electrophysiological abnormalities ranging from 0% to 89% (2, 5-9, 11, 16, 22, 24-26). Ramirez et al. described a reversible neuropathy after 30 years of phenytoin administration, characterized by axonal shrinkage and secondary demyelination in sural nerve biopsy (19). The aim of our study was: 1. to compare NCV's of a big number of patients, treated for a long time with

several antiepileptic drugs, with those of a control collective, 2. to evaluate possible relations of NCV's with total amount and daily dose of anticonvulsants as well as with the duration of treatment, and 3. to compare the patients under monotherapy with respect to possible differences in NCV's.

Materials and methods

548 epileptics (313 males, 235 females) of the Heidelberg outpatient clinic for convulsive disorders were investigated, who took antiepileptic drugs since 1 year at least. To keep out influences of age, well-known for NCV's, only patients with an age of 20 to 40 years were considered. A further criterion for exclusion was the presence of a possible other cause of neuropathy like diabetes mellitus, uremia or alcohol abuse. For each anticonvulsant drug taken by the patient, duration of therapy and

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average daily dose were fixed. The whole dose and the average daily dose of all anticonvulsants were expressed as equivalent units, 1 unit equalling 50 mg phenytoin, 30 mg phenobarbital, 125 mg primidone, 50 mg CHP-phenobarbital, 200 mg carbamazepine, 50 mg mephenytoin, 250 mg ethosuximide, 300 mg valproate sodium, 2 mg clonazepam, 300 mg mesuximide, 100 mg sultiam, or 250 mg trimethadione. NCV's were compared by Wilcoxon test with the values of 70 healthy controls of the same age group. Motor conduction velocity (MCV) of median and peroneal nerve was measured with a DISA 1500 EMG system using surface stimulating and recording electrodes, sensory conduction velocity (SCV) of median nerve on the forearm in antidromic technique using ring electrodes on the second digit. Skin temperature was constant at 34°C by use of an automatic DISA skin temperature system.

Results

The epileptics had lower mean values of NCV's compared with the controls (table 1). 69 epileptics showed a slowing of MCV of peroneal nerve <45 m/s, 21 a reduction of MCV of median nerve <50 m/s and 44 a reduction of SCV of median nerve <55 m/s (see Figures 1-3). Considering overlapping in some patients, 102 epileptics remained with at least 1 pathological value of NCV. Correlation with drug parameters were calculated separately for males and females, because both sexes differed in dose of medication (Krause, 1987). In both sexes, MCV of peroneal nerve and SCV of median nerve correlated negative with drug parameters, whereas only in males MCV of median nerve showed such a correlation (Table 2). The results of the patients under monotherapy with phenytoin, carbamazepine, primidone, or valproate sodium since 1 year at least are compiled in Table 3. For all NCV's the group under carbamazepine showed the lowest values, this difference being significant for MCV of median nerve in Kruskal-Wallis test ($p < 0.05$) and after Duncan (for carbamazepine compared to phenytoin and valproate sodium).

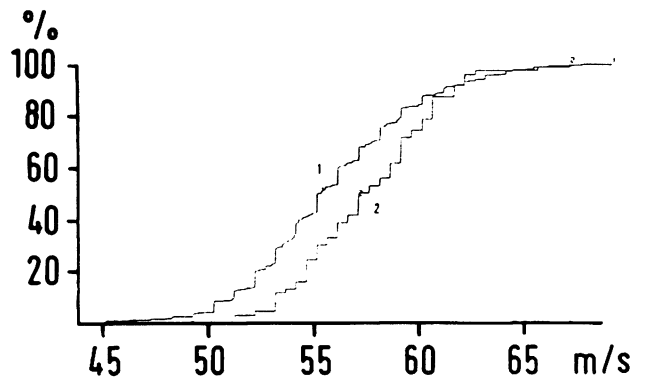


Fig. 1. — Frequency distribution of motor conduction velocity of median nerve in the forearm in epileptics (1) and controls (2).

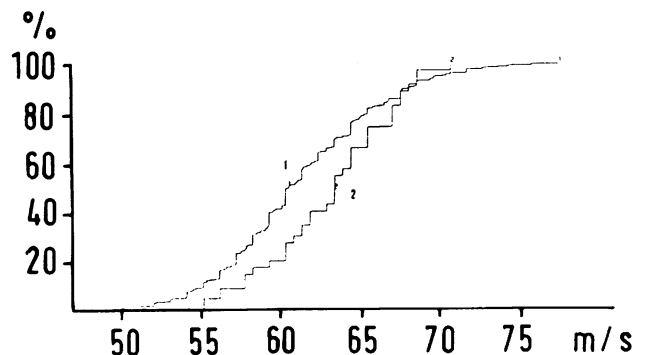


Fig. 2. — Frequency distribution of sensory conduction velocity of median nerve in the forearm in epileptics (1) and controls (2).

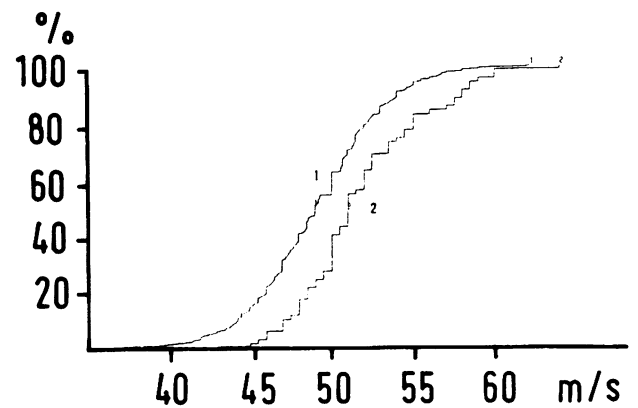


Fig. 3. — Frequency distribution of motor conduction velocity of peroneal nerve in epileptics (1) and controls (2).

Table 1. — Mean value and standard deviation of motor and sensory conduction velocity of median nerve and of motor conduction velocity of peroneal nerve in epileptics and controls with *p* value of Wilcoxon test

	Epileptics			Controls			<i>p</i>
	n	x	SD	n	x	SD	
Median nerve, MCV	548	55.8	4.0	70	57.5	3.3	0.0002
Median nerve, SCV	548	61.1	4.8	70	62.7	3.9	0.0014
Peroneal nerve, MCV	548	49.3	3.9	70	51.9	4.0	0.0001

Table 2. — Correlations between drug parameters (total amount of all anticonvulsants as equivalent units, average daily dose as equivalent units per day and duration of antiepileptic treatment) and motor and sensory conduction velocity of median as well as motor conduction velocity of peroneal nerve (*R*=Spearman's rank correlation coefficient and *p* value) in epileptic males (*m*, *n*=313) and females (*f*, *n*=235)

	Sex	Equivalent units total	Equivalent units/d	Duration
Median nerve, MCV	<i>m</i>	<i>R</i> = -0.13254 <i>p</i> = 0.0190	<i>R</i> = -0.13747 <i>p</i> = 0.0149	<i>n.s.</i>
	<i>f</i>	<i>n.s.</i>	<i>n.s.</i>	<i>n.s.</i>
Median nerve, SCV	<i>m</i>	<i>R</i> = -0.14491 <i>p</i> = 0.0103	<i>R</i> = -0.19165 <i>p</i> = 0.0007	<i>n.s.</i>
	<i>f</i>	<i>R</i> = -0.17483 <i>p</i> = 0.0072	<i>R</i> = -0.13712 <i>p</i> = 0.0357	<i>R</i> = -0.15571 <i>p</i> = 0.0169
Peroneal nerve, MCV	<i>m</i>	<i>R</i> = -0.13731 <i>p</i> = 0.0151	<i>R</i> = -0.14558 <i>p</i> = 0.0099	<i>n.s.</i>
	<i>f</i>	<i>R</i> = -0.19850 <i>p</i> = 0.0022	<i>R</i> = -0.14275 <i>p</i> = 0.0287	<i>R</i> = -0.19896 <i>p</i> = 0.0022

Table 3. — Mean value and standard deviation of motor and sensory conduction velocity of median nerve and motor conduction velocity of peroneal nerve in epileptics under monotherapy with phenytoin (PHT), primidone (PRM), carbamazepine (CBZ) or valproate sodium (VPA)

	PHT n=53	PRM n=81	CBZ n=53	VPA n=32
Median nerve, MCV	57.5 ± 4.2	56.1 ± 4.2	54.9 ± 4.3	56.7 ± 3.9
Median nerve, SCV	62.7 ± 4.5	62.2 ± 4.4	60.7 ± 6.0	61.9 ± 4.7
Peroneal nerve, MCV	50.0 ± 3.8	50.0 ± 3.9	49.3 ± 4.5	49.5 ± 3.5

Discussion

The results of our study indicate, that long term therapy with antiepileptic drugs causes a lowering of NCV's. The influence of medication is confirmed by the negative correlations found between NCV's and mean daily dose as well as total amount of anticonvulsants taken by the epileptics. In accordance with the results of Geraldini et al. (12) and Taylor et al. (23) the males showed no correlations with duration of therapy, whereas the females of our study did so. The correlations between medication and MCV of peroneal nerve were more pronounced than those with NCV's of median nerve; so, the MCV of peroneal nerve seems to be the most sensitive in our study. The frequency of 19% of patients with at least one diminished NCV is in accordance with the incidence described by Lovelace and Horwitz (16) and Swift et al. (22).

Swift et al. also found lowering of MCV of peroneal nerve as the most frequent sign of nerve involvement (22). As own investigations have shown, most cases with slowing of NCV's are only subclinical neuropathies with neither subjective complaints nor pathological reflex status (15). Concerning type of drugs, in former studies especially phenytoin has been thought to cause lowering of NCV's ("hydantoin neuropathy"); but patients with chronic intake of antiepileptic drugs mostly have a combination therapy with at least 2 drugs (20). The influence of the other anticonvulsants on NCV is not clear, regarding the literature: Results concerning the possible role of carbamazepine are controversial (3, 4, 17, 21, 22, 24, 25); in patients under monotherapy with barbiturates an influence has been suggested, but only 6 patients were examined (21). Comparing the patients under monotherapy we found no significant differences in the MCV of peroneal nerve and SCV of median nerve; but MCV of median nerve was lower in the patients under carbamazepine compared to those under phenytoin and valproate sodium. It is of interest, that Danner et al. in their prospective study also found lower NCV of median nerve under treatment with carbamazepine compared to phenytoin (3). All in all, the results of our study

suggest, that 1. under long term antiepileptic treatment mean NCV is lowered and that 2. phenytoin in long term therapy has no specific influence on NCV compared to other antiepileptic drugs. This is also confirmed by the results of Geraldini et al. (12). Therefore we propose to change the term "hydantoin neuropathy" into "anticonvulsant neuropathy" or "neuropathy induced by antiepileptic drugs".

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