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A prospective 1 year follow-up study with somatosensory potentials evoked by stimulation of the posterior tibial nerve in patients with supratentorial cerebral infarction

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Summary Somatosensory potentials evoked by stimulation of the posterior tibial nerve (tibial nerve SEPs) were studied in 40 patients with supratentorial non-haemorrhagic cerebral infarction and in 25 control subjects. SEPs were recorded twice in 39 patients and thrice in 35 patients. The first examination was carried out 4–19 days after the onset of the symptoms, the second examination 56–100 days after the stroke, and the third examination 348–393 days after the stroke. Increased side-to-side differences in the P57 and N75 peak latencies and absence of the P40 peak were the most frequent abnormal findings. The latency abnormalities were associated with involvement of the subcortical white matter of the rolandic region. The absence of the P40 peak was, in contrast, closely related to the extension of the infarcted area into the cortical gray matter of the rolandic region. When all SEP abnormalities were taken into account 55% of patients showed at least one abnormality in the tibial nerve SEP during the acute stage, 51% of patients had abnormal SEPs in the second examination and 43% of patients in the third examination. A nearly significant decrease was observed in the number of latency abnormalities, but the number of amplitude abnormalities, including absent responses, did not change during the 1 year follow-up period.

Key words: Somatosensory evoked potentials; Cerebral infarction; Ischemia; Posterior tibial nerve; (Man)

The clinical diagnostic usefulness of somatosensory potentials evoked by lower limb stimulation has mainly been studied in patients with lesions in the spinal cord and with diffuse neurological diseases. This method seems to be promising for the detection of spinal cord lesions in cervical spondylosis (Perlik and Fisher 1987; Heiskari et al. 1988). Furthermore, with diffuse lesions SEPs to lower limb stimulation appear to be fairly sensitive: e.g., subclinical dysfunction of the spinal cord has been attested in the early phases of diabetes (Gupta and Dorfman 1981; Cracco et al. 1984). In permanent subcortical ischaemic lesions lower limb SEPs seem to be complementary to median nerve SEPs, increasing the number of detected abnormalities (Chu 1986a; Hassel et al. 1986). The present study was conducted to establish the significance of the tibial nerve SEPs in patients with local ischaemic lesions in the supratentorial space, and to establish the course of the changes in the tibial nerve SEPs during a prospective 1 year follow-up study.

Patients and methods

A 1 year follow-up was conducted in the Department of Clinical Neurophysiology of Oulu University Central Hospital from August 1985 to August 1988. Forty patients with non-haemorrhagic supratentorial cerebral infarction were examined. Tibial nerve SEPs were recorded 3 times and a neurological examination was performed 3 times: (1) within 4–19 (mean 9.8) days after the infarction (40 patients); (2) within 56–100 (mean 69) days (39 patients); (3) within 348–393 (mean 370) days (35 patients). Cerebral computed tomography (CT) was studied at least once during the acute stage, 1–21 (mean 5.1) days after the stroke.

The clinical neurological examination, using a form specially designed for this study, tested the following variables: level of consciousness, receptive dysphasia, expressive dysphasia, sense of touch, sense of temperature, joint position sense, sense of vibration, stereognosis, neglect, ability to move, motor performance in upper arm, muscle tone in the limbs of the affected side, enhancement of the biceps reflex, enhancement of the patellar reflex, Babinski sign. All variables were classi-

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electrode was used. Linked ears reference electrodes (A_1A_2) were used for the scalp channel, Ag/AgCl surface electrodes being placed on both earlobes and both electrodes being connected to the grid 2 of the amplifier. In the second channel (popliteal fossa) an Ag/AgCl surface electrode placed 4 cm above the "active" electrode was used as the reference. SEPs were recorded using a 4-channel Disa 1500 D electromyograph, which included a Disa 15 G 21 averager. The sampling frequency was 5 kHz. Trials with excessive artifacts were rejected automatically. The bandpass was 2 Hz–2 kHz in all channels. 1500 trials were averaged, and this procedure was performed twice on both sides. The posterior tibial nerve was stimulated at the ankle with surface electrodes. The strength of the stimulus was adjusted to slightly over the motor threshold so that

it did not produce pain; the duration was 200 μ sec, the frequency was 3 Hz. Analysis time was 200 msec. Latencies were measured on the oscilloscope screen with a cursor, and the pictures were then made with an analog x-y plotter. The amplitudes were measured on the paper. The peak latencies of the components P40, N48, P57, N75 and peak-to-peak amplitudes N32-P40, P40-N48, P57-N75 were measured (if N32 was not present, the N32-P40 amplitude was measured from the baseline to the peak of the P40 wave). On both sides means of the latency and amplitude values from both averages were calculated (averaging was done twice on both sides). Side-to-side differences of the latencies and interpeak latency P40-N75 were calculated from the mean latencies. Amplitudes were transformed to logarithms and side-to-side differences in the amplitudes

TABLE II

The intersession stability studied in 5 control subjects. The amplitude values in the table are logarithms of the measured amplitudes. Time lapse between the first and second examinations is presented as months.

	Age (years)	37	38	40	48	48
	Gender	female	female	female	female	female
	Time lapse (months)	47	33	34	37	32
P40						
Right:	first	38.0	37.5	39.0	35.5	38.7
	second	38.0	36.3	37.6	34.7	38.5
	change	0.0%	-3.2%	-3.6%	-2.3%	-0.5%
Left:	first	38.0	37.5	39.0	33.5	39.4
	second	38.4	37.3	37.8	34.4	39.1
	change	+1.1%	-0.5%	-3.1%	+2.7%	-0.8%
N75						
Right:	first	75.0	74.0	78.5	80.5	81.0
	second	77.0	77.0	78.5	82.5	81.5
	change	+2.7%	+4.1%	0.0%	+2.5%	+0.6%
Left:	first	77.5	74.5	79.0	85.0	87.0
	second	80.0	74.5	78.5	87.0	86.5
	change	+3.2%	0.0%	-0.6%	+2.4%	-0.6%
P40-N75 IPL						
Right:	first	37.0	36.5	39.5	45.0	42.3
	second	39.0	40.8	40.9	47.8	43.1
	change	+5.4%	+11.8%	+3.5%	+6.2%	+1.9%
Left:	first	39.5	37.0	40.0	51.5	47.6
	second	41.7	37.3	40.7	52.7	47.4
	change	+5.6%	+0.8%	+1.8%	+2.3%	-0.4%
P40-N48 ampl.						
Right:	first	0.564	0.457	0.423	1.106	0.966
	second	0.559	0.396	0.385	1.126	0.986
	change	-0.9%	-13.3%	-9.0%	+1.8%	+2.1%
Left:	first	0.609	0.555	0.269	1.097	0.665
	second	0.694	0.485	0.512	1.201	0.677
	change	+14.0%	-12.6%	+90.3%	+9.5%	+1.8%
P57-N75 ampl.						
Right:	first	0.643	0.548	0.574	1.075	0.981
	second	0.563	0.551	0.528	1.118	0.764
	change	-12.4%	+0.5%	-8.0%	+4.0%	-22.1%
Left:	first	0.688	0.672	0.619	1.183	0.900
	second	0.694	0.540	0.628	1.243	0.694
	change	+0.9%	-19.6%	+1.5%	+5.1%	-22.9%

TABLE III

The variables used as criteria of abnormality. 'Affected side' was drawn randomly by lot in the control group. Amplitudes were transformed to logarithms, and side-by-side differences in the amplitudes were transformed to decibels by the formula: $10 \times \log(\text{ampl}_{\text{aff}}/\text{ampl}_{\text{non}})$, where ampl_{aff} = amplitude on the 'affected' side and ampl_{non} = amplitude on the 'non-affected' side (both as microvolts); side-by-side differences in the latencies were calculated $\text{lat}_{\text{aff}} - \text{lat}_{\text{non}}$, where lat_{aff} = latency on the 'affected' side and lat_{non} = latency on the 'non-affected' side. Normal limits of amplitudes as microvolts and normal limits of side-by-side differences in the amplitudes as amplitude ratios (higher amplitude per lower amplitude) are in parentheses.

	Statistics in the control group				Limits of normality		
	Mean	S.D.	Median	Range	Mean - 3 S.D.	Mean + 3 S.D.	3 S.D.
<i>Affected side</i>							
P40-N75 IPL (msec)	40.9	3.8	39.5	34.5-49.0		52.4 msec	
log(N32-P40 amplitude)	0.36	0.28	0.42	-0.50-0.91	-0.48 (0.33 μV)	1.20 (15.8 μV)	
log(P40-N48 amplitude)	0.54	0.25	0.49	0.03-1.11	-0.21 (0.62 μV)	1.29 (19.5 μV)	
log(P57-N75 amplitude)	0.67	0.21	0.65	0.17-1.07	0.04 (1.10 μV)	1.30 (20.0 μV)	
<i>Side-by-side differences</i>							
P40 latency (msec)	-0.31	1.31	0	-3.5-2.0			3.93 msec
N48 latency (msec)	-0.12	1.72	0	-4.0-4.0			5.16 msec
P57 latency (msec)	-0.64	1.81	-1.0	-3.5-3.5			5.43 msec
N75 latency (msec)	-1.34	4.28	-1.0	-14.0-8.0			12.84 msec
N32-P40 amplitude (dB)	0.13	1.99	0.41	-4.1-3.9			± 5.97 dB (3.95*)
P40-N48 amplitude (dB)	0.46	2.06	0.28	-3.7-4.4			± 6.18 dB (4.15*)
P57-N75 amplitude (dB)	-0.38	1.37	-0.45	-2.9-3.5			± 4.11 dB (2.57*)

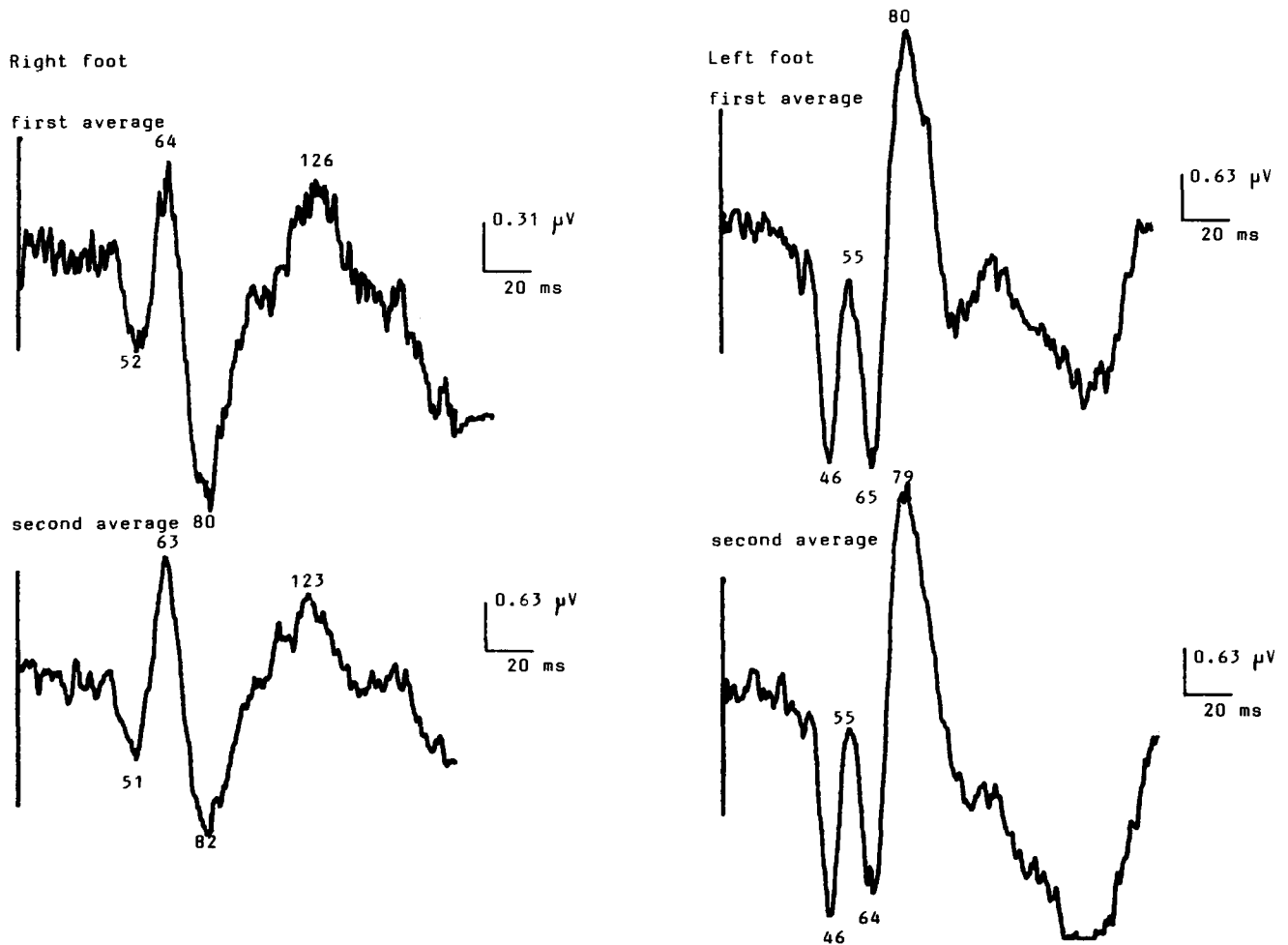


Fig. 1. Tibial nerve SEPs of a 33-year-old male (the second patient in group g in Tables I, IV and V) recorded at C_2' (in the midline, 2 cm behind C_2) with A_1A_2 reference. The responses to stimulation of the right foot are on the left and of the left foot on the right of the figure. The first average on each side is in the upper half and the second average is in the lower half of the figure. The P40-N75 IPL, and also the side-to-side differences in the P40, N48, P57 and N75 peak latencies were abnormal when the right foot was stimulated. The amplitudes were normal.

were transformed to decibels by the formula: $10 \times \log(\text{ampl}_{\text{aff}}/\text{ampl}_{\text{non}})$, where ampl_{aff} = amplitude in the affected side, ampl_{non} = amplitude in the non-affected side. These transformations resulted in normal (i.e., gaussian) distributions (the Shapiro-Wilk test for normality). In 5 control subjects, a second tibial nerve SEP record was made after an interval of 2 years 8 months (32 months) to 3 years 11 months (47 months) from the first recording (Table II). In these subjects, the latency values were slightly more stable than the amplitude values, and the consistency of the later waves (P57 and N75) was about as good as that of the earlier waves (P40 and N48).

Group differences in the peak latencies were studied by analysis of covariance adjusted for height, age and gender, and in the P40-N75 interpeak latency and the amplitudes by analysis of covariance adjusted for age and gender. The changes in peak latencies, interpeak latencies and amplitudes between the first, second and third examinations were analysed with a paired *t* test, and the changes in the number of abnormalities with a chi-square test. Statistical analyses were performed with

a PC/XT compatible microcomputer and a SAS/STAT™ version 6.03 statistical software package. The interpeak latency P40-N75 on the affected side, the side-to-side differences of P40, N48, P57 and N75 peak latencies, the peak-to-peak amplitudes N32-P40, P40-N48, P57-N75 on the affected side, and the side-to-side differences of N32-P40, P40-N48, P57-N75 amplitudes were used as criteria of normality. The limits of normality were: mean + 3 S.D. in the interpeak latencies, mean - 3 S.D. in the amplitudes (after logarithmic transformation), zero \pm 3 S.D. in the side-to-side differences (where the values were distributed symmetrically around zero) calculated from the control group. The absence of a peak was classified as amplitude abnormality. The normal limits are presented in Table III.

Results

The tibial nerve SEPs recorded in the scalp channel of 3 patients are presented in Figs. 1-3.

Case 1 (Fig. 1): the patient was a 33-year-old male

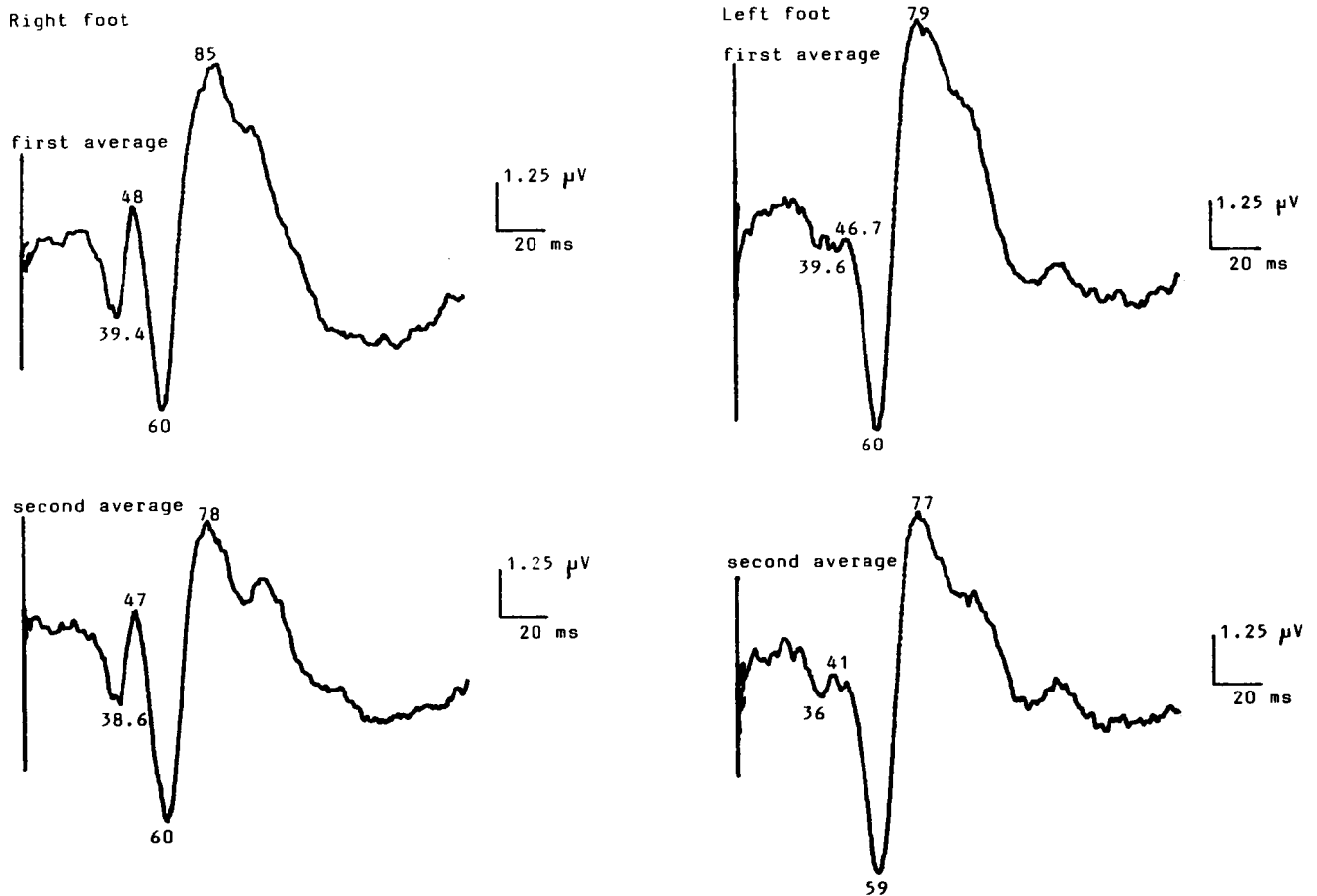


Fig. 2. Tibial nerve SEPs of a 30-year-old male (the first patient in group i in Tables I, IV and V) recorded at C_2' with A_1A_2 reference. The arrangement as in Fig. 1. The P40-N75 IPL and also the side-to-side differences in peak latencies were normal. The P40-N48 peak-to-peak amplitude was abnormal when the left foot was stimulated (both the absolute value and the side-to-side difference were abnormal). The P57-N75 amplitude was normal.

(the second patient in group g in Tables I, IV and V). The CT was done 2 days after the onset of the symptoms. There was extensive infarction in the left hemisphere: in the capsula interna, in the prefrontal, temporal, precentral and postcentral white matter, and in the temporal, precentral and postcentral gray matter. A neurological examination was performed 10 days after the stroke. The patient had total aphasia (which precluded sensory examination), and there were no voluntary movements in the limbs of the right side; the biceps and patellar reflexes were enhanced on the right side, and the Babinski sign was positive on the right side. In the tibial nerve SEPs recorded 11 days after the stroke,

the P40-N75 IPL was abnormal when the right foot was stimulated, and the side-to-side differences in the P40, N48, P57 and N75 peak latencies were abnormal, but all amplitudes were normal.

Case 2 (Fig. 2): the patient was a 30-year-old male (the first patient in group i in Tables I, IV and V). The CT was done 1 day after the onset of the symptoms. There was infarction in the right hemisphere: of the postcentral white matter, the white matter of the temporal lobe, and of both the white and gray matter in the occipital lobe. A neurological examination was performed 4 days after the stroke, the only abnormal sign being a defect in the visual field of the left side. The

TABLE IV

Abnormalities of the latencies of the tibial nerve SEP during the acute stage in relation to the findings in the CT (see Table I). abn = abnormal, m = male, f = female.

CT class	Age	Sex	P40-N75 IPL (aff. side)	Side-by-side differences			
				P40	N48	P57	N75
a: thalamus	16	m		abn			
	68	f					
	29	f				abn	
b: capsula int.	54	f					
	58	f					
c: capsula int. + PREC.W	45	m					
	60	m					
d: capsula int. + POSTC.W + G	60	m					
e: capsula int. + PREC.W + POSTC.W	59	m		abn	abn	abn	abn
	58	m					
	65	f	abn			abn	abn
f: capsula int. + PREC.W + POSTC.W + G	64	m					
	56	m					
	40	m					
g: capsula int. + PREC.W + G + POSTC.W + G	53	m					
	33	m	abn	abn	abn	abn	abn
	64	m				abn	abn
	47	m	abn	abn	abn	abn	abn
	39	f	abn	abn	abn	abn	abn
66	f						
55	m				abn	abn	
h: PREC.W + G	33	m					
i: POSTC.W	30	m					
	53	m	abn				abn
j: POSTC.W + G	44	m					
k: PREC.W + POSTC.W	71	m	abn				
	57	f					
l: PREC.W + G + POSTC.W + G	54	m					abn
	66	m				abn	abn
	36	f					
m: occipital l.	52	m	abn			abn	abn
	41	f					
n: normal	55	m		abn	abn	abn	
	68	m					
	52	m					
	53	f					
	56	m					
	39	m					
	64	m					
	54	m					

TABLE V

Abnormalities of the amplitudes of the tibial nerve SEP during the acute stage in relation to the findings in the CT (see Table II). att = attenuated, abs = absent, enh = enhanced, aff.side = affected side, s.-b.-s.d. = side-by-side difference, m = male, f = female.

CT class	Age	Sex	P40-N48 amplitude		P57-N75 amplitude	
			aff. side	s.-b.-s.d.	aff. side	s.-b.-s.d.
a: thalamus	16	m				
	68	f				
	29	f				
b: capsula int.	54	f				
	58	f				
c: capsula int. + PREC.W	45	m				
	60	m				
d: capsula int. + POSTC.W + G	60	m	abs	abs		att
e: capsula int: + PREC.W	59	m				
	58	m	att		att	
	65	f				enh
	64	m				
f: capsula int. + PREC.W	56	m	abs	abs	abs	abs
	40	m				
g: capsula int. + PREC.W + G	53	m	abs	abs	abs	abs
	33	m				
	64	m	abs	abs		att
	47	m	att	att		att
	39	f				
	66	f	abs	abs	abs	abs
	55	m	abs	abs		
h: PREC.W + G	33	m				
i: POSTC.W	30	m	att	att		
	53	m				
j: POSTC.W + G	44	m				
k: PREC.W + POSTC.W	71	m				
	57	f				
l: PREC.W + G + POSTC.W + G	54	m	abs	abs		
	66	m	abs	abs	att	att
	36	f	abs	abs	abs	abs
m: occipital l.	52	m				
	41	f				
n: normal	55	m				
	68	m				
	52	m				
	53	f				
	56	m				
	39	m				
	64	m				
	54	m				

tibial nerve SEPs were recorded 5 days after the stroke: the P40-N75 IPL and also the side-to-side differences in peak latencies were normal, but the P40-N48 peak-to-peak amplitude was abnormally low when the left foot was stimulated (both the absolute value and the side-to-side difference were abnormal).

Case 3 (Fig. 3): the patient was a 66-year-old female (the sixth patient in group g in Tables I, IV and V). The CT was done 21 days after the stroke. There was extensive infarction of the left hemisphere: of the capsula interna, the prefrontal, temporal, precentral and postcentral white matter, and of the prefrontal, temporal, precentral and postcentral gray matter. A neuro-

logical examination was performed 6 days after the stroke: the level of consciousness was still reduced; the patient had deficiency on the right side of the sense of light touch, temperature, joint position and vibration, and of stereognosis, and there were no voluntary movements in the limbs of the right side; the biceps reflex was enhanced on the right, and the Babinski sign was positive on that side. The tibial nerve SEPs were recorded 7 days after the stroke: all cortical peaks were absent when the right foot was stimulated.

Abnormalities in the peak latencies and in the P40-N75 IPLs of the tibial nerve SEPs during the acute stage are presented in Table IV (the classification of the

CT findings is described in Table I). Altogether 15 patients (38%) had abnormalities of latency. Most frequently they occurred in the side-to-side differences of the P57 or N75 peak (13 patients). Only 4 patients with normal side-to-side differences of the N75 latency had abnormalities of the other latencies, and only 1 patient with normal P57 and N75 side-to-side differences had abnormal side-to-side differences of the earlier components. As many as 13 (87%) out of 15 patients with abnormalities of the latencies had involvement of the subcortical white matter in the postcentral region.

During the acute stage altogether 13 patients (33%) had abnormalities of amplitudes (Table V). All patients with abnormal N32-P40 amplitudes also had abnormalities of the P40-N48 or P57-N75 amplitudes, and hence the N32-P40 amplitude was not included in the table. The P40-N48 amplitude was abnormal in 12 patients (30%) and the P57-N75 amplitude in 10 patients (25%). All patients with an absent P40 peak in the first examination (9 patients) had evidence of involvement of the cortical gray matter in the postcentral area in the CT.

When both the latency and the amplitude abnormalities were taken into account, 22 patients (55%) during the acute stage, 20 patients (51%) about 2 months later and 15 patients (43%) about 12 months after the stroke

had abnormal tibial nerve SEPs (Fig. 4). The changes in the number of amplitude abnormalities and in the total number of abnormalities were not significant (chi-square test), but nearly significant decreases between the second and the third examination ($P = 0.034$ with chi-square test), and between the first and the third examination ($P = 0.023$ with chi-square test) were found in the number of latency abnormalities (Fig. 4).

All 6 patients with a lowered level of consciousness at the time of the first recording showed abnormal SEPs in all the 3 examinations (Fig. 5). In the subgroup of 15 patients with mass displacements as many as 13 patients (87%) had abnormal SEPs during the acute stage. Furthermore, 12 (80%) out of 15 patients with involvement of both the cortical gray matter and the subcortical white matter in the rolandic region had abnormal SEPs during the acute stage, whereas in the subgroup of 13 patients with involvement of the subcortical white matter but not the cortical gray 8 patients (62%) had abnormal SEPs.

The differences in the means of P40 and N75 peak latencies, P40-N75 interpeak latency (IPL), P40-N48 and P57-N75 amplitudes between the control group and certain CT groups for all the 3 examinations were tested with analysis of covariance. Patients with absent peaks could not be included in these statistical analyses. How-

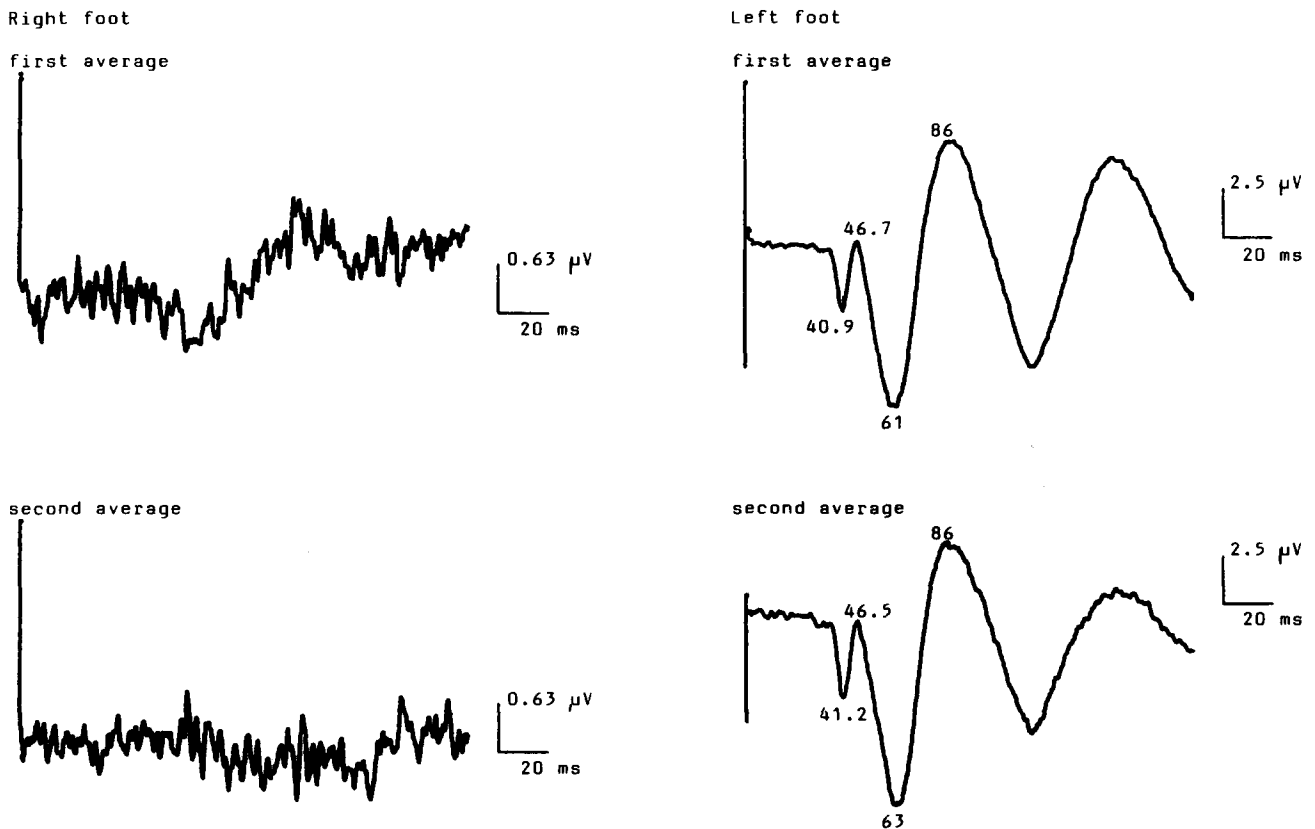


Fig. 3. Tibial nerve SEPs of a 66-year-old female (the sixth patient in group g in Tables I, IV and V) recorded in C_2' with A_1A_2 reference. Arrangement as in Fig. 1. The P40, N48, P57 and N75 peaks were absent when the right foot was stimulated.

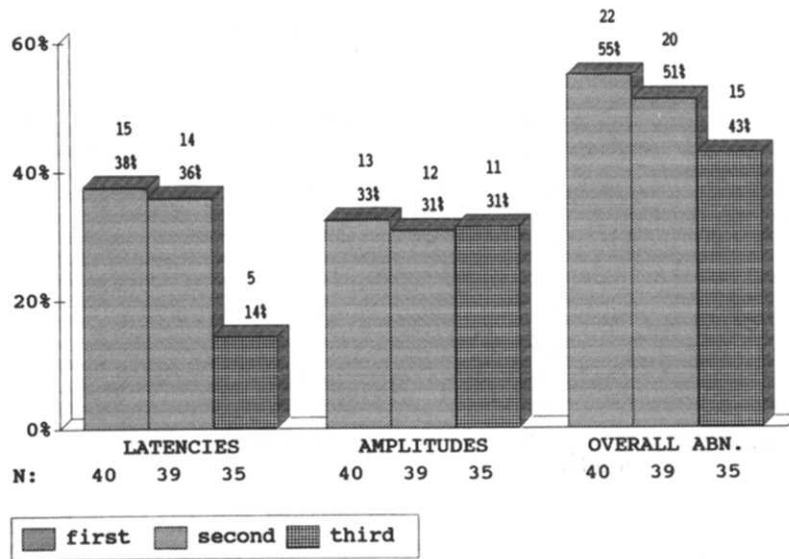


Fig. 4. The percentages of abnormal SEP findings in latencies and amplitudes as well as the total number of abnormal findings in the entire patient group (OVERALL. ABN.) in the first examination (days 4–19), in the second examination (days 56–100) and in the third examination (days 348–393).

ever, some significant differences were observed between the control group and those patients with measurable peaks (Figs. 6–9). The mean of the P40 latency was still, about 1 year after the stroke, significantly longer in the group of patients with involvement of the rolandic cortex (mean 44.3, S.D. 3.8) than in the control group (mean 39.8, S.D. 3.6, $P = 0.007$ by the analysis of

covariance, adjusted for height, age and gender). In the P40-N75 IPL, only a nearly significant difference was found during the acute stage between the entire patient group and the control group (Fig. 6). In this acute stage the subgroups of patients with mass displacements and patients with involvement of the rolandic cortex differed highly significantly from the control group in the

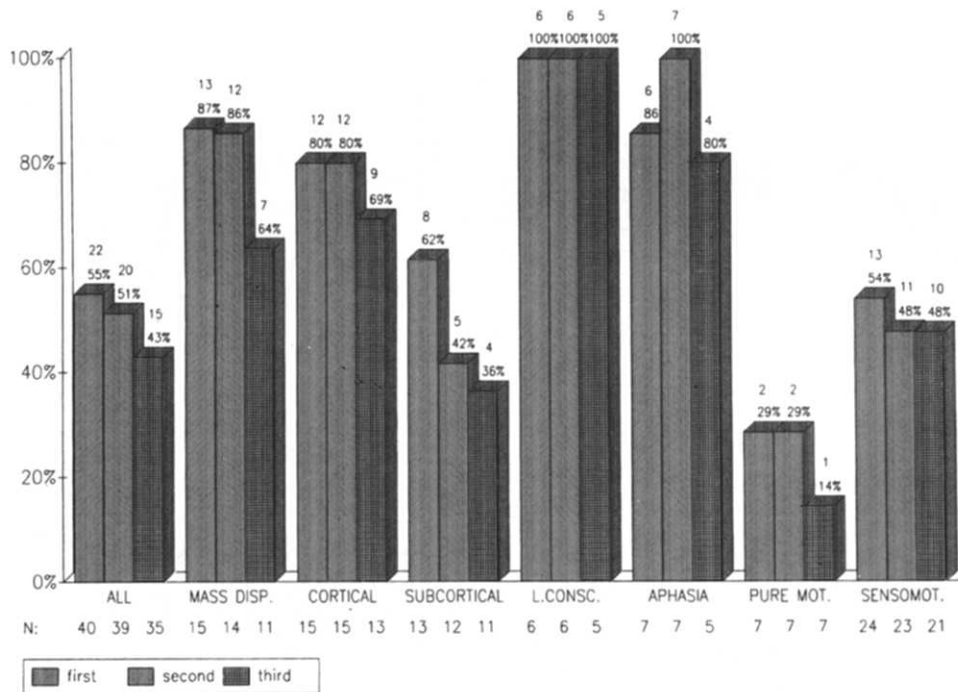


Fig. 5. The total number of abnormal SEP findings in relation to the CT and the clinical examination in the first, second and third examinations. ALL = all patients, MASS DISP. = patients with evidence of mass displacements in the CT, CORTICAL = patients with involvement of both the cortical gray matter and the subcortical white matter in the rolandic region, SUBCORTICAL = patients with involvement of the subcortical white matter but not the cortical gray matter in the rolandic region, L.CONSC. = patients with lowered level of consciousness, APHASIA = patients with right-sided hemiplegia and severe aphasia, PURE MOT. = patients with pure motor hemiparesis, SENSOMOT. = patients with sensorimotor stroke.

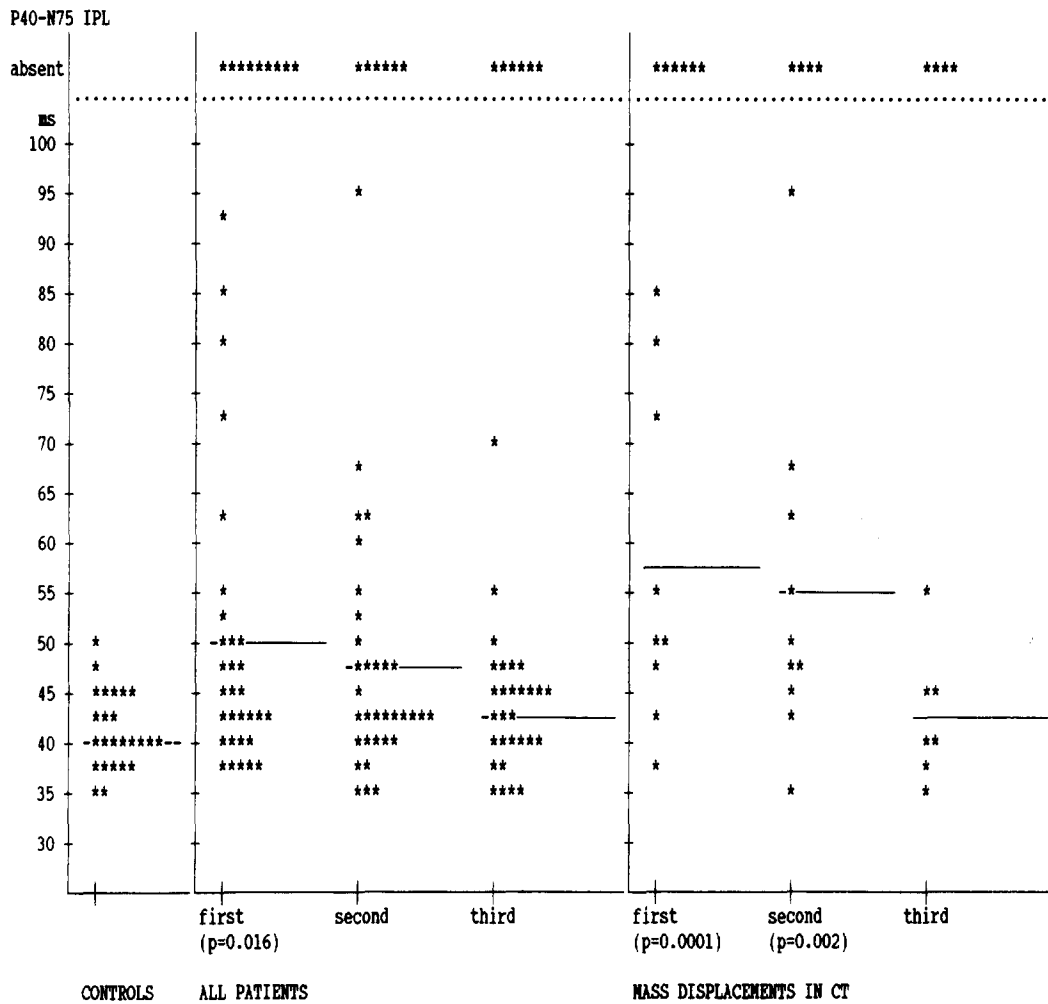


Fig. 6. The scatter of the P40-N75 IPLs in the control group, in the entire patient group, and in the subgroup of patients with evidence of mass displacement in the CT. * = one patient, first = the first examination (days 3-15), second = the second examination (days 56-99), third = the third examination (days 347-392). The levels of significance were tested by analysis of covariance adjusted for age and gender.

P40-N75 IPL (Figs. 6 and 7). During the follow-up period these differences diminished; in the second examination the patients with mass displacements differed significantly and the patients with involvement of the rolandic cortex only nearly significantly from the control group, and in the third examination no significant differences were noted any more (Figs. 6 and 7). Thus, the prolongations in the P40-N75 IPL were observed mainly during the acute stage.

When considering the amplitudes, the changes in the P40-N48 amplitude were more profound than those of P57-N75. The difference between the subgroup with mass displacements and the control group in the P40-N48 amplitude was still significant in the third examination (Fig. 8). The subgroup of patients with involvement of the subcortical white matter in the rolandic region differed significantly from the control group in the P40-N48 amplitude during the acute stage and in the second examination, and the difference was nearly significant in the third examination (Fig. 9). The

P40 peak was absent during the acute stage in 9 out of 15 patients (60%) with both cortical and subcortical involvement in the rolandic region, but in the subgroup of patients with only subcortical involvement, none had an absent P40 peak in the first and second examinations, and only 1 patient had an absent P40 peak in the third examination (Fig. 9).

The changes in the P40 and N75 latencies, in the P40-N75 IPL and in the P40-N48 and P57-N75 amplitudes between the first, second and third examinations were tested with paired *t* test in the same subgroups of patients as in Figs. 6-9. In the entire patient group there were no significant changes. In the subgroup of patients with mass displacements there was a nearly significant ($P = 0.037$) decrease in the N75 peak latency between the second and the third examinations: in the third the N75 latency was on average 7.5 msec (S.D. 7.4 msec) shorter than in the second. In the subgroup of patients with involvement of the subcortical white matter in the rolandic region there was a small but significant

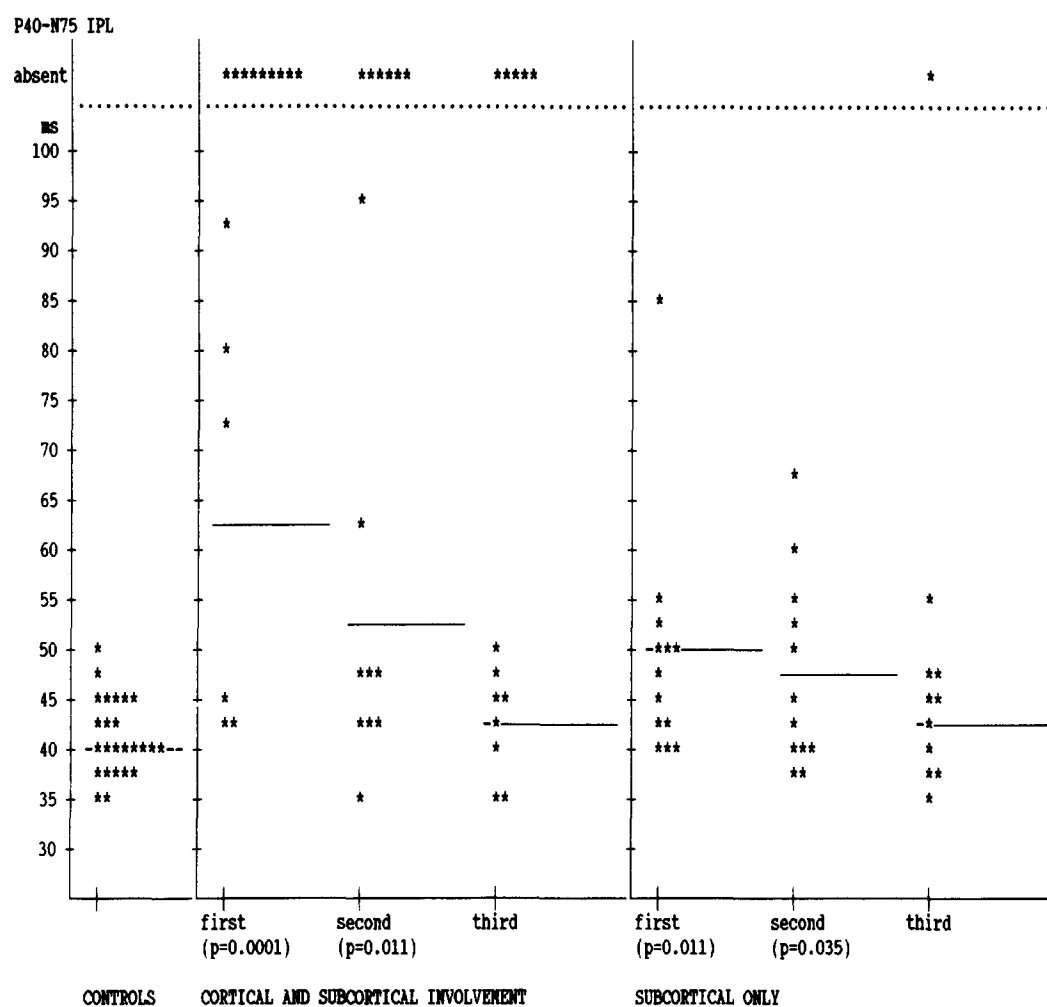


Fig. 7. The scatter of the P40-N75 IPLs in the control group, and in the subgroups of patients with involvement of both the cortical gray matter and the subcortical white matter in the rolandic region and patients with involvement of the subcortical white matter but not the cortical gray matter in the rolandic region. * = one patient, first = the first examination (days 3–15), second = the second examination (days 56–99), third = the third examination (days 347–392). The levels of significance were tested by analysis of covariance adjusted for age and gender.

diminution of the P57-N75 amplitude from the first to the second examination (the mean change in the logarithmic amplitude = -0.11 , S.D. = 0.12 , $N = 12$, $P = 0.007$) and from the first to the third examinations (the mean change in the logarithmic amplitude = -0.14 , S.D. = 0.13 , $N = 10$, $P = 0.006$), but no significant changes between the second and the third examinations.

Discussion

The most frequent abnormal findings in the tibial nerve SEPs were high side-to-side differences in the P57 or N75 peak latencies and absence of the P40 peak. The latency abnormalities were associated with involvement of the subcortical white matter in the rolandic region when related to the findings in the CT scan. On the other hand, the absence of the P40 peak was closely connected with extension of the infarcted area into the cortical gray matter of the rolandic region. It has been

established in earlier studies of the generators of the tibial nerve SEPs that the source of the P40 peak is a cortical generator located in the interhemispheric fissure (Cruse et al. 1982; Berić and Prevec 1983; Seyal et al. 1983; Desmedt and Bourguet 1985; Lesser et al. 1987), that the generators of the P57 peak are probably situated more posteriorly in the parietal lobe, and that the N75 peak has a widespread negative field over both the frontal and posterior scalp (Desmedt and Bourguet 1985). The absence of the P40 peak in some patients might thus reflect neuronal damage in the cortex of the rolandic area. High side-to-side differences in the P57 and N75 peak latencies could, in contrast, indicate involvement of the subcortical white matter (possibly in the cortico-cortical connections or in the successive thalamo-cortical loops). This suggestion confirms the observation of Chu (1986a) that in subcortical infarcts mainly mid- and long-latency SEP components are affected. Similar findings have been reported in the literature about median nerve SEPs: according to Mauguière

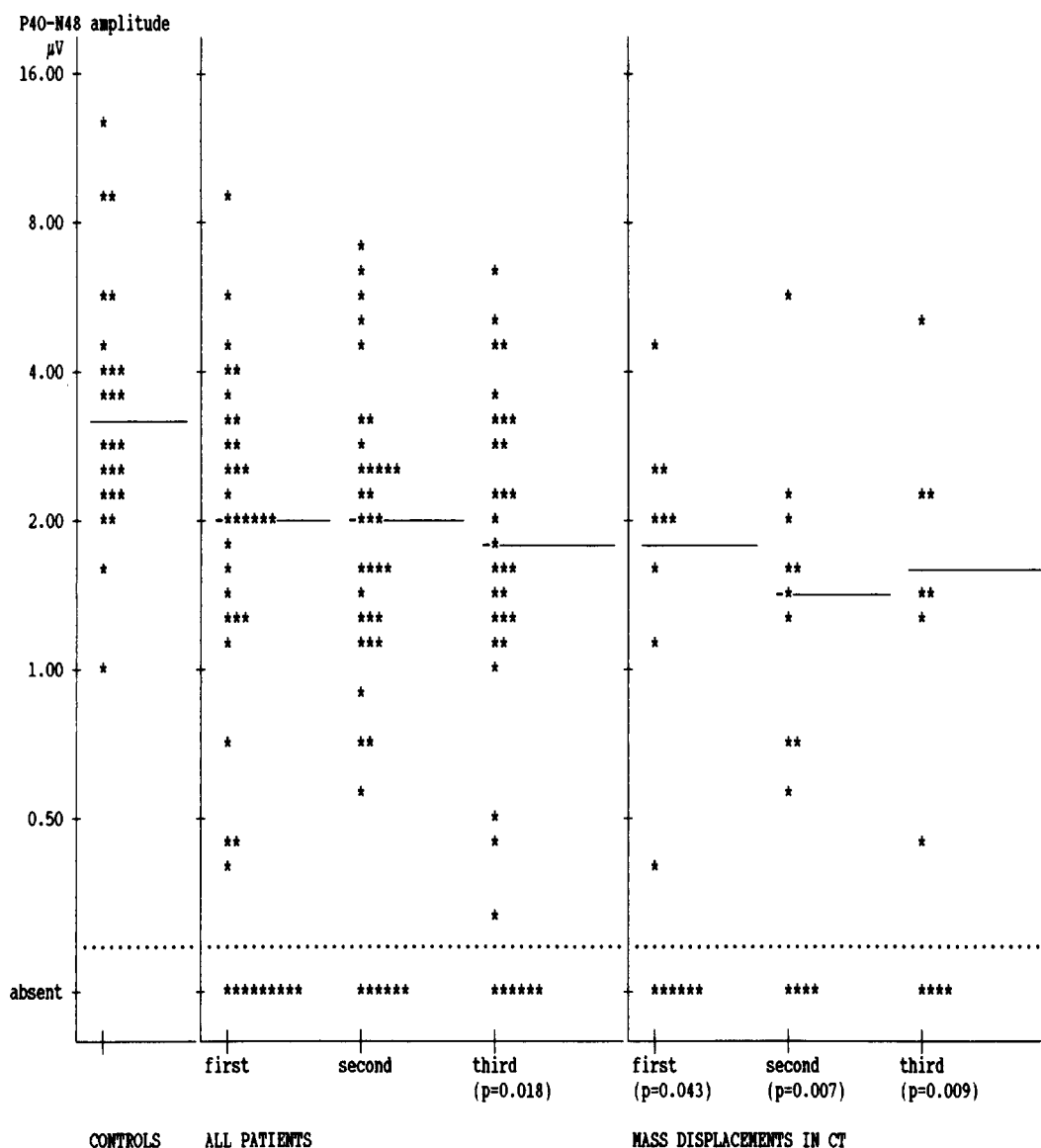


Fig. 8. The scatter of the P40-N48 peak-to-peak amplitudes in the control group, in the entire patient group, and in the subgroup of patients with evidence of mass displacement in the CT. * = one patient, first = the first examination (days 3-15), second = the second examination (days 56-99), third = the third examination (days 347-392). The levels of significance were tested by analysis of covariance adjusted for age and gender.

et al. (1983) typical abnormalities in patients with cortical lesions are changes in amplitude or changes in duration of cortical SEP components, whereas the onset latencies are either equal to those of the controls or only slightly longer. The hypothesis that the absence of the P40 peak might reflect cortical neuronal damage is further supported by our finding about the persistence of the amplitude abnormalities during the 1 year follow-up period (Fig. 4).

When the measured latencies and amplitudes of the control group and the patient groups are compared by analysis of covariance, it can also be noted that the differences in the N75 peak latency and the P40-N75 IPL were concentrated in the acute stage, whereas the differences in the early components were more perma-

nent. This is in accordance with our hypothesis that the changes in the N75 peak latency indicate involvement of the subcortical white matter.

The height of the subject correlates highly with the peak latencies of tibial nerve SEPs. Using multiple correlation and regression analysis, Chu (1986b) has observed that height provides the best prediction of the remaining latencies, whereas age alone does not correlate with latencies. In addition, Kakigi (1987) noted that the interpeak latencies of cortical potentials following "P35" (our P40) do not correlate with age, but that the amplitudes of short- and middle-latency cortical potentials are slightly smaller in aged subjects than those in young subjects. In the present study the problem of interpreting the role of height in abnormalities was

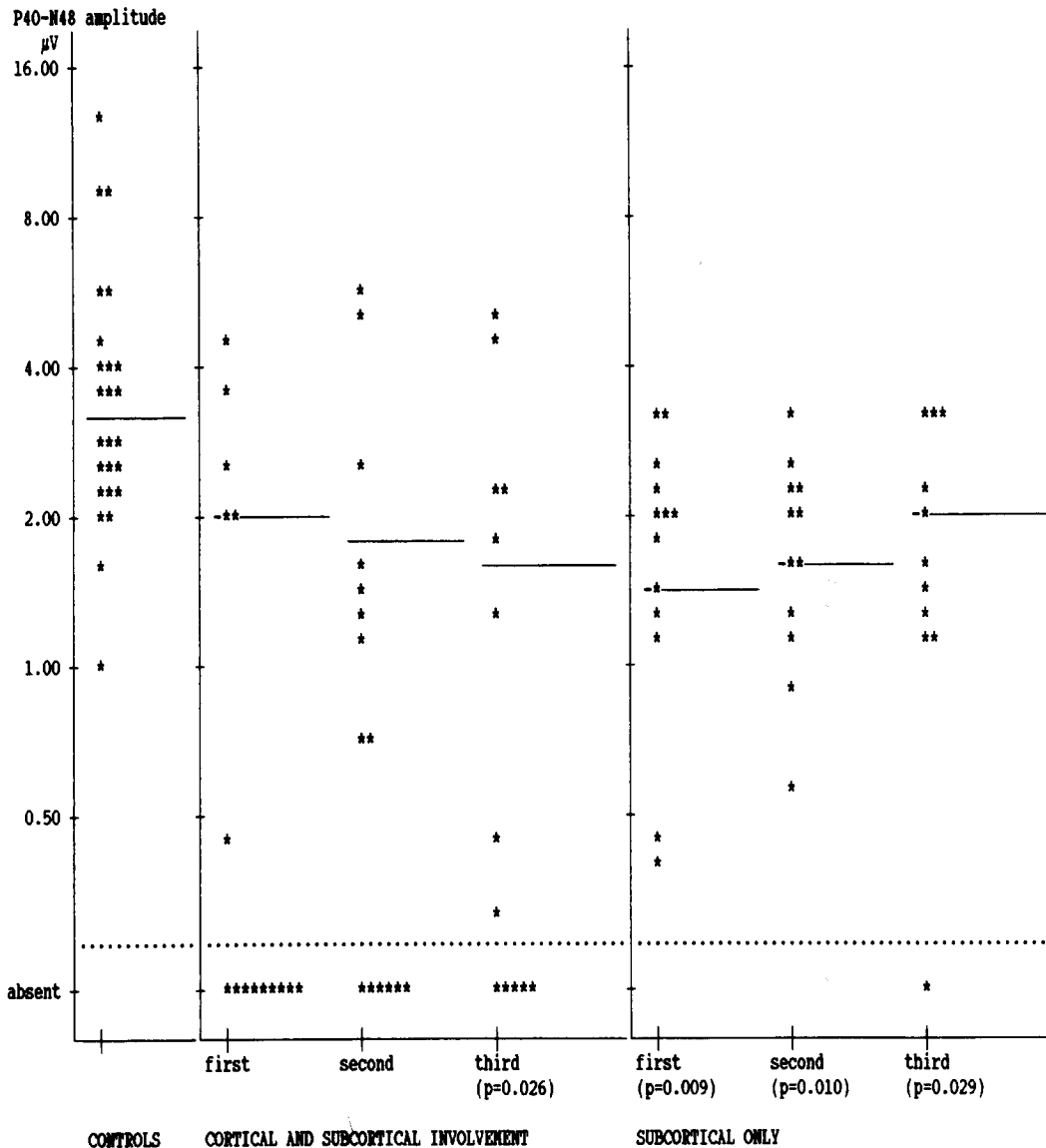


Fig. 9. The scatter of the P40-N48 peak-to-peak amplitudes in the control group, and in the subgroups of patients with involvement of both the cortical gray matter and the subcortical white matter in the rolandic region and patients with involvement of the subcortical white matter but not the cortical gray matter in the rolandic region. * = one patient, first = the first examination (days 3–15), second = the second examination (days 56–99), third = the third examination (days 347–392). The levels of significance were tested by analysis of covariance adjusted for age and gender.

avoided by using only the P40-N75 IPL and side-to-side differences of the peak latencies as criteria of abnormality of the latencies. In the statistical analysis of differences of measured values between different subgroups and the control group we used analysis of covariance adjusted for height, age and gender for the peak latencies, and adjusted for age and gender for the P40-N75 IPL and the amplitudes.

Studies have been published which indicate that SEPs may have prognostic significance in cerebral infarct (La Joie et al. 1982; Regli and Despland 1982; Pavot et al. 1986; De Weerd et al. 1987).

Although only 55% of patients with cerebral infarction had abnormal tibial nerve SEPs, in certain subgroups a substantial proportion of patients revealed

abnormal findings. A greater number of abnormalities would have been detected if narrower limits of normality than ours (± 3 S.D.) had been used. However, as narrower limits would inevitably lead to false positive findings (altogether 11 parameters were used as criteria of abnormality in 3 successive examinations) we preferred the use of the ± 3 S.D. limits.

Prolongation of the peak N75 latency and P40-N75 IPL of the tibial nerve SEPs correlated with damage in the subcortical white matter of the rolandic area, observed mainly during the acute stage. The abnormalities of amplitude, especially the absence of the P40 peak and attenuation of the P40-N48 peak-to-peak amplitude, were associated with the extension of the infarcted area into the cortical gray matter of the rolandic region.

The amplitude abnormalities were relatively permanent throughout the 1 year follow-up period.

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