

Neurophysiological observations after chronic styrene exposure

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ROSÉN, I., HAEGER-ARONSEN, B., REHNSTRÖM, S. and WELINDER, H. Neurophysiological observations after chronic styrene exposure. *Scand. j. work environ. & health* 4 (1978): suppl. 2, 184—194. Thirty-three styrene exposed workers from three different industrial sites were examined with electroencephalography and motor and sensory neurography. The three groups had respective styrene exposures of clearly above the threshold limit value (50 ppm), at about this level, and clearly below it. The neurophysiological results were compared with those of a group of normal controls and a group of 17 patients judged to suffer from sequelae after long-term heavy exposure to organic solvents (mainly painters). Ten subjects in the styrene group presented signs of a mild sensory neuropathy with polyphasic sensory responses of a low amplitude. The same pattern was commonly found among the reference group heavily exposed to solvents. The ten subjects in the styrene group with mild polyneuropathy had a significantly higher age and significantly heavier styrene exposure than the rest of the group. Age difference could not explain the difference in the neurophysiological parameters, and therefore the contributing role of styrene exposure has to be considered. The electroencephalographic analysis showed no changes of the dominant alpha frequency. An increased amount of diffuse slow activity was seen in many of the heavily exposed mixed-solvent cases and was seen in some of the styrene-exposed cases without a clear relation to degree of exposure. An increased occurrence of fast activity in central and precentral areas of the brain was found in the styrene group, as well as in the mixed-solvent group. This pilot study indicates that the same type of neurophysiological changes from the strictly normal are seen among workers exposed to styrene as those found among a group of patients judged to suffer from sequelae after chronic exposure to various organic solvents. The neurophysiological "profile" is (a) sensory nerve responses with low amplitude and long duration, (b) somewhat low sensory conduction velocities, (c) close to normal motor neurographic findings, and (d) an increased amount of fast activity in central and precentral regions in the electroencephalogram in combination with normal occipital alpha activity.

Key words: electroencephalography, motor neurography, sensory neurography, styrene exposure.

Experimental studies on man (3, 7, 13) have demonstrated effects on central nervous function after acute styrene exposure. Concerning possible chronic effects of long-term styrene exposure, no statistically significant differences could be dem-

onstrated in a psychometric comparison between styrene workers and nonexposed controls (6). However, within the group of styrene workers a correlation was found between visuomotor inaccuracy and low psychomotor performance on one hand and styrene exposure on the other. Neurophysiological investigations of the same group of workers (10) demonstrated a somewhat higher frequency of electro-

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encephalographic (EEG) abnormalities in comparison with the normal frequency. No neurographic abnormality was found in this group of subjects.

Our study was made to compare groups of subjects differing in degree of exposure to styrene. The investigation had the character of a pilot study with the purpose of finding, if any, a profile of psychometric and neurophysiological deviations from the strictly normal as a result of chronic styrene exposure. Reference groups were formed from a group of normal nonexposed subjects and a group of patients considered to suffer from sequelae due to chronic exposure to mixed organic solvents.

MATERIAL

The air content of styrene was determined after personal sampling close to the inspiratory zone by absorption on active carbon. Gas chromatography was made after elution with carbon disulfide. The values given are the time-weighted averages for the total sampling periods.

The test groups comprised 33 styrene workers from three different plants.

Group A

Thirteen men, aged 24–66 years (mean 44 years) and employed for 1–21 years (mean 10 years) in the polyester resin boat industry formed group A. None of them had previously been exposed to organic solvents. Measurements during 1974 showed average levels among 11 employees varying from 74 to 175 ppm (mean 125 ppm). The company started production in 1963 with three workers under very bad conditions. In 1965 a new plant was built with improved conditions. In 1973 new facilities were acquired with conditions similar to those at the time of the investigation. The ventilation was considered much improved. From 1971 on, respiratory protective devices were commonly used

during the rolling and spraying processes. Their use is continuously improving. During the year of the investigation (1975) group A was presumably less exposed than group B. During the preceding years, however, group A had had a significantly higher exposure.

Group B

Ten men, aged 23–54 years (mean 43 years) and involved in production of polyester resin cisterns for 2–14 years (mean 7 years) made up group B. Nine of the subjects had not been exposed to organic solvents before, whereas one had been exposed to industrial solvents 22 years earlier. In 1974 large-scale measurements on 14 persons during 3 d showed average concentrations between 21 and 67 ppm (mean 47 ppm). Because of the rotating work task system used, we believe that the measurements were representative for the group. Massive exposure has occasionally occurred, and for some of the long-time employees the exposure may have exceeded the threshold limit value significantly.

Group C

Ten men, aged 29–65 years (mean 44 years) and having worked in polystyrene production for 5–15 years (mean 9), comprised group C. Measurements had been made on a number of occasions over the last five years. The styrene exposure did not exceed 5 ppm. Exposure is due to leakage from reactors. When the process started in 1948 no air evacuation was used. An improvement was made in 1955 when a ventilation system was installed. From 1966 on, no changes have been made that would affect the level of styrene exposure. From 1966 to 1974 a parallel production was introduced with exposure to aliphatic hydrocarbons, particularly isopentane in some cases. This exposure was judged to be of minor significance.

Based on measurements and judgement of the individual tasks, an evaluation of the degree of exposure was made for each subject on a 1–3 scale.

Table 1. Percentage distribution of symptoms, signs, and the occurrence of possible effect-modifying factors among the normal controls and styrene-exposed groups.

Parameter	Group			
	Normal control	Styrene-exposed		
		A	B	C
Symptoms				
Unusual tiredness	17	85	40	20
Reduced short-term memory	17	62	40	30
Giddiness	0	54	40	10
Headache	33	46	40	30
Paraesthesia in fingers/toes	0	23	30	10
Weakness in arm/leg	0	8	0	20
Signs				
Conjunctivitis	0	31	10	20
Skin irritation	17	38	20	40
Throat irritation	17	85	50	50
Minor decrease of muscle stretch reflexes	0	23	10	20
Possible effect modifying factors				
Smokers	33	46	60	50
"Higher than average" alcohol consumption	0	15	10	0
Head trauma	17	15	10	0
Diabetes mellitus	0	8	0	0

Normal control group

Six men from 22 to 57 years of age (mean 36 years), all employed in the hospital transportation service and never exposed to organic solvents to any significant degree, formed a normal control group (K).

Reference group

Seventeen men, 43–61 years of age (mean 53 years), referred to and examined at the Department of Occupational Health and considered to suffer from sequelae due to chronic exposure to mixed organic solvents, formed another reference group (MS).

The styrene exposed groups, as well as the normal controls, were asked to fill out a written questionnaire about symptoms, particularly with respect to the central

and peripheral nervous system. The complete results as well as the results of the clinical and laboratory investigations will be reported separately. The most relevant findings in the neurophysiological context are summarized in table 1. Among symptoms, increased tiredness, a reduction in short-term memory, and giddiness appeared to be related to the levels of exposure in the different groups. At the medical examinations signs of local irritation of conjunctivas and throat appeared, but no signs of polyneuropathy or central nervous lesion.

All subjects were investigated psychometrically with a wide spectrum of tests covering intellectual as well as psychomotor functions. The results of the psychometric investigation will be reported separately.

The styrene-exposed subjects were investigated with a minimum of 48 h of free interval after the last exposure. The purpose was to avoid possible acute effects of styrene on the test results (3).

METHODS

Sensory neurography

Motor neurography

Motor conduction velocities (MCV) were determined on a DISA 1500 electromyograph for the median, ulnar, fibular, and posterior tibial nerves by surface recording from the thenar, hypothenar, extensor digitorum brevis and abductor hallucis muscles, respectively. Electrical stimulation was applied at the wrist and elbow levels as well as at the ankle and knee levels. Only the velocity of the fast fibers was determined.

The sensory action potential (SAP) was recorded from the median nerve and in many cases also from the ulnar nerve. Electrical stimulation was applied to the first and third fingers (sometimes the fifth) with a constant current stimulator. A teflon-coated needle electrode was used for recording at the wrist, and the position was adjusted by electrical stimulation via the needle (threshold values < 0.8 mA). Surface electrodes were used for recording the median SAP at the elbow level. The upper frequency limit for SAP recording was 10 kHz. Signal averaging of at least

Table 2. Neurographic parameters, age, and exposure parameters of the various test and control groups. Mean values and standard deviations are given as well as the results of group comparisons with the normal controls.

Parameter	Normal control (N = 6)	Group			Mixed solvent-exposed (N = 17)
		A (N = 13)	B (N = 10)	C (N = 10)	
Age (years)	36 $\pm$ 15	44 $\pm$ 13	43 $\pm$ 12	44 $\pm$ 12	53 $\pm$ 6
Exposure					
Degree (0—3)	0	2.85 $\pm$ 0.38	2.30 $\pm$ 0.48	1 $\pm$ 0	—
Years	0	10.5 $\pm$ 4.7	6.7 $\pm$ 4.7	9.1 $\pm$ 3.9	—
Time-weighted exposure (degree $\times$ years)	0	30 $\pm$ 14.0	17.2 $\pm$ 15.1	9.1 $\pm$ 3.9	—
Motor neurography ^a					
MCV _M (m/s)	61.75 $\pm$ 7.38	60.92 $\pm$ 5.31	59.75 $\pm$ 3.85	58.85 $\pm$ 3.54	57.22 $\pm$ 5.96
Distal latency (M)	3.18 $\pm$ 0.38	3.88 $\pm$ 0.61	3.43 $\pm$ 0.43	3.40 $\pm$ 1.27	3.81 $\pm$ 0.54
MCV _U (m/s)	62.83 $\pm$ 6.38	60.69 $\pm$ 6.66	60.95 $\pm$ 2.63	56.05 $\pm$ 4.09	58.15 $\pm$ 6.33
MCV _F (m/s)	48.5 $\pm$ 3.39	48.23 $\pm$ 3.19	50.55 $\pm$ 3.79	46.85 $\pm$ 4.31	45.72 $\pm$ 4.60
MCV _T (m/s)	46.82 $\pm$ 2.38	44.73 $\pm$ 2.91	49.60 $\pm$ 3.43	45.95 $\pm$ 4.70	43.47 $\pm$ 6.50
Sensory neurography ^b					
SCV I dist (m/s)	49.00 $\pm$ 2.28	44.58 $\pm$ 6.61	48.15 $\pm$ 6.17	43.95 $\pm$ 3.67	45.65 $\pm$ 5.80
SCV III dist (m/s)	52.25 $\pm$ 3.19	48.47 $\pm$ 7.93	50.20 $\pm$ 6.14	47.05 $\pm$ 5.05	49.12 $\pm$ 6.34
SCV prox (m/s)	58.67 $\pm$ 4.40	62.85 $\pm$ 9.13	60.25 $\pm$ 3.24	58.20 $\pm$ 4.06	58.82 $\pm$ 5.19
SA I (uV)	26.67 $\pm$ 11.25	18.31 $\pm$ 9.64	18.10 $\pm$ 6.33	20.20 $\pm$ 8.99	17.35 $\pm$ 11.72
		0.1 > P	0.05 > P	n.s.	0.1 > P
SA III (uV)	16.00 $\pm$ 10.60	12.69 $\pm$ 7.38	11.20 $\pm$ 4.69	13.40 $\pm$ 10.11	13.12 $\pm$ 10.06
SD I (ms)	1.84 $\pm$ 0.16	2.71 $\pm$ 1.23	2.42 $\pm$ 1.11	2.24 $\pm$ 0.52	3.12 $\pm$ 0.96
SD III (ms)	2.03 $\pm$ 0.08	2.46 $\pm$ 1.00	2.23 $\pm$ 0.74	2.52 $\pm$ 0.84	3.31 $\pm$ 1.29
		0.05 > P	0.1 > P	0.025 > P	0.0005 > P

^a MCV = motor conduction velocity; M = median nerve; U = ulnar nerve; F = fibular nerve; T = posterior tibial nerve.

^b SCV = sensory conduction velocity; SA = sensory amplitude; SD = sensory duration; I = first finger; III = third finger; prox = from wrist to elbow.

Al.Br. ♂ 54y Exp. ~10y

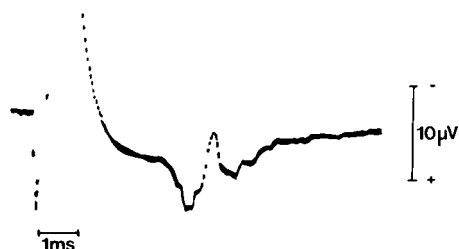
MEDIAN MOTOR

68.5 m/s



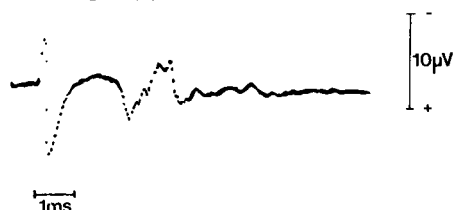
MEDIAN SENSORY

33.5 m/s



ULNAR SENSORY

44 m/s



To.Ni. ♂ 23y Exp. ~2y

MEDIAN MOTOR

60 m/s



MEDIAN SENSORY

44.5 m/s

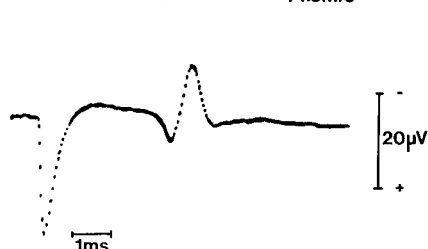


Fig. 1. Neurographic records from two styrene-exposed workers. Upper row of records shows the compound motor action potentials recorded from the thenar eminence by surface electrodes on stimulation of the median nerve at the wrist and elbow. Note normal conduction velocities in both cases. The lower rows of records show sensory action potentials recorded by needle electrodes from the median and, in the left case, also from the ulnar nerve. Additional details are given in the text. Note the polyphasic nature of sensory action potential responses in case Al.Br. in comparison with To.Ni. Time and voltage calibrations are given in the figure.

128 sweeps were regularly used for determining the SAP duration and amplitude. The skin temperature overlying the thenar was regularly checked and was not allowed to fall below 30°C.

Electroencephalography

The EEG examination was made as a routine record for 30 min with a 16-

channel Elema electroencephalograph with 21 electrodes placed according to the international 10—20 system. The mean frequency of the occipital dominant alpha (α) activity was determined, as well as the presence of faster (β) or slower (θ) activity. Localized EEG abnormalities were also noted. The effects of arousal, hyperventilation, and intermittent light were studied. All EEG records were made in wakefulness, and none of the subjects fell asleep during the recording.

Statistics

Group comparisons were made with the Student's *t*-test.

RESULTS

Neurographic results

The means and standard deviations of the various neurographic parameters are summarized in table 2. For the most commonly used parameter, i.e., MCVs, no differences between the groups were found. Also the distal motor latencies of the median nerve were similar. However, for the sensory neurography parameters certain differences were noticed. Particularly the duration of the SAPs at the wrist was significantly longer for the styrene-exposed groups when they were compared to normal controls. The difference was even larger for the MS group. Also for the SAP amplitudes an overall tendency towards lower values appeared among the styrene and MS groups when they were compared to normal controls. Due to the wide spread of the values and to the small control group, the difference was statistically significant only for group B. The sensory conduction velocities from the stimulus sites at fingers I and III to the wrist were somewhat lower in the styrene and MS groups, but the differences were not statistically significant.

An increased duration and a decreased amplitude of SAPs indicate increased dispersion of conduction velocities among the individual sensory axons and possibly a reduction of excitable fibers. This phenomenon is further illustrated in fig. 1. The neurographic records from a comparatively young subject with short exposure to styrene (group B) are shown in the right part of the figure. Motor and sensory conduction velocities are normal, and the sensory response has a normal amplitude and a short duration, around 2 ms. The left part of the figure illustrates the records from a subject with low amplitude and polyphasic sensory responses with long duration after more than 10

years of exposure to styrene. The sensory conduction velocity is reduced for the median nerve and within normal limits for the ulnar nerve. Note, however, that

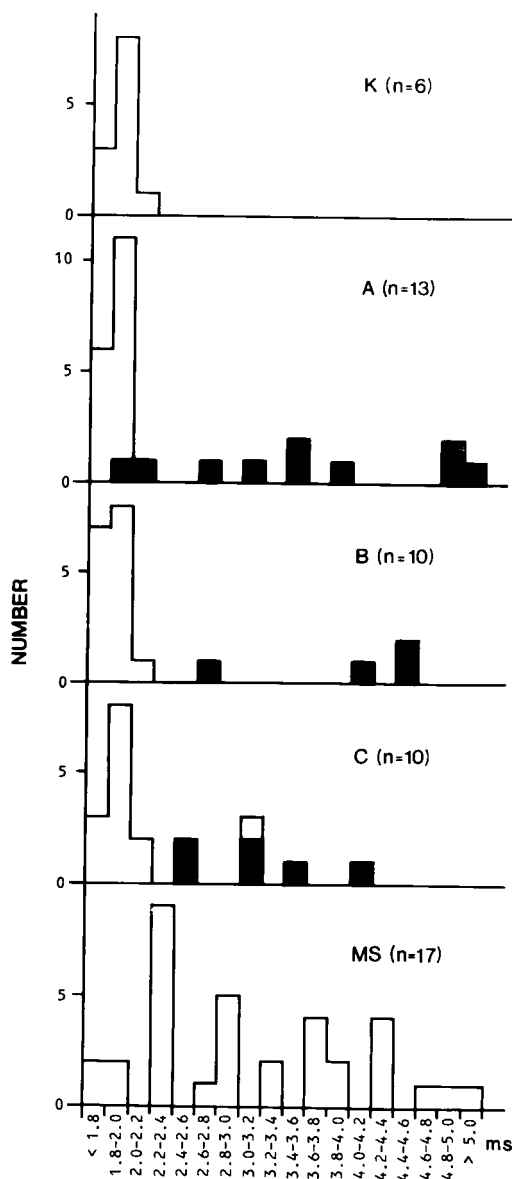


Fig. 2. Distribution of the duration of the sensory action potentials recorded from the median nerve at the wrist after the stimulation of fingers I and III. The values for the two fingers have been plotted together. Filled areas indicate the values obtained in subjects considered as possible cases of polyneuropathy. Such a subdivision was not made for the group exposed to mixed solvents (MS). (A, B, and C = styrene-exposed workers; K = normal controls)

Table 3. Comparison of one group of styrene workers with possible neuropathy and the remaining group of styrene-exposed subjects. Mean values, standard deviations and results of group comparisons are given. Values in italics refer to results of a group comparison with correction for age.

Parameter	Styrene-exposed subjects		
	Neuropathy group (N = 10)		Remaining group (N = 23)
Age (years)	54.9 ± 7.89	0.0005 > P	39.7 ± 10.64
Exposure			
Degree (0—3)	2.4 ± 0.97		2.0 ± 0.80
Years	11.50 ± 4.54	0.025 > P	7.80 ± 4.6
Time-weighted exposure (degree × years)	28.90 ± 17.16	0.01 > P	15.80 ± 12.16
Motor neurography ^a			
MCV _M (m/s)	58.30 ± 4.55	<i>n.s.</i>	60.67 ± 4.17
Distal latency (M)	4.11 ± 0.71	0.025 > P	3.55 ± 0.33
MCV _U (m/s)	55.5 ± 4.11	0.01 > P	61.24 ± 4.69
MCV _F (m/s)	46.75 ± 4.64	<i>n.s.</i>	49.28 ± 3.37
MCV _T (m/s)	44.75 ± 4.03	<i>n.s.</i>	47.37 ± 4.00
Sensory neurography ^b			
SCV I (m/s)	40.85 ± 6.18	0.025 > P	47.48 ± 4.49
SCV III (m/s)	42.91 ± 7.08	0.01 > P	51.02 ± 4.59
SCV prox (m/s)	55.35 ± 6.58	0.05 > P	62.96 ± 5.11
SA I (uV)	9.40 ± 3.41	0.0005 > P	22.91 ± 6.23
SA III (uV)	6.40 ± 2.72	0.0025 > P	15.09 ± 7.40
SD I (ms)	3.74 ± 1.03	0.0005 > P	1.93 ± 0.16
SD III (ms)	3.41 ± 0.94	0.0005 > P	1.97 ± 0.27
EEG ^c			
α (Hz)	9.60 ± 0.70		9.74 ± 0.78
β	8/10		1/23
θ	1/10		5/23
loc.	0/10		2/23

^a MCV=motor conduction velocity; M=median nerve; U=ulnar nerve; F=fibular nerve; T=posterior tibial nerve.

^b SCV=sensory conduction velocity; SA=sensory amplitude; SD=sensory duration; I=first finger; III=third finger; prox=from wrist to elbow.

^c EEG=electroencephalography; α=occipital alpha activity; β=increased fast activity; θ=diffuse slow activity; loc.=localized abnormalities.

also the SAP of the ulnar nerve is of low amplitude and widely dispersed in time.

From the hypothesis that the increased temporal spread of the SAPs might be an expression of sensory neuropathy, possibly related to styrene exposure, a further analysis was made. In fig. 2 the distribution of SAP durations were plotted for the different groups of subjects. Among the normal controls all sensory nerve responses had short durations. Among the styrene-exposed groups five subjects in group A, two in group B, and three in group C could be selected as possible cases of sensory neuropathy. Of considerable interest is also the observation that, among

the 17 subjects in the MS group, 11 exhibited clearly prolonged duration of their SAPs in comparison with the normal controls. One case in group C with a sensory duration of 3.1 ms from one finger and 2 ms from the other and the other neurographic findings completely normal was included in the normal group. One subject in group A with a normal SAP duration but with distinctly low SAP amplitudes and a decreased sensory conduction velocity from wrist to elbow was included among the cases of suspected sensory neuropathy.

An attempt to subdivide the styrene-exposed material into one group of 10 with

possible sensory neuropathy and one group of 23 without such signs is shown in table 3. When several parameters are included in this comparison a large difference of age appears with a difference of mean values of about 15 years. The extent to which the other observed differences between the two groups can be explained by difference in age should be considered. The mean values and standard deviations given in table 3 are based upon the actual measured values. The results of group comparison tests in italics were based upon values for each individual after correction for age according to the age regression factors given by Buchtal et al. (1). Even after compensation for age significant differences appeared between the groups in terms of sensory conduction velocities for all three sites measured. For finger I, only a sensory amplitude difference of 7 % can be attributed to age difference, compared with the observed 144 %; for finger III the respective figures are 19 % compared with 136 % (1). The long duration of the sensory responses in the first group is also far beyond the normal range. Thus, even when the difference in age is taken into consideration, the 10 selected subjects are seen to differ significantly in all sensory neurography parameters. The diffuse nature of the disorder disclosed is further emphasized by a significant difference in MCV for the ulnar nerve in the forearm and the median nerve at the wrist level. When the reason for this difference is considered, it is important to note that the two groups differed

significantly not only in age, but also in time of styrene exposure, as well as in the time-weighted styrene exposure (degree of exposure $\times$ years of exposure).

Electroencephalographic results

The results of the EEG investigation are summarized in table 4. The mean frequencies of the occipital alpha activity did not differ among the groups. An increased amount of fast activity within central and frontocentral areas was seen in one of the normal controls, in nine of the styrene-exposed subjects (27 %), and in ten of the 17 patients within the MS group (59 %). An increased amount of slow activity over both hemispheres was observed in one normal control, in six of the styrene-exposed subjects (18 %), and in ten of the MS patients (59 %). Somewhat unexpectedly the highest frequency of diffuse slow activity was found in group C, which had the lowest level of styrene exposure. The additional exposure of isopentane in this group might have contributed to this finding. Localized EEG abnormalities were observed in only two cases, of which one could be related to a previous head trauma. The other person was 57 years old, and a clinically silent cerebrovascular disturbance seems to be a likely explanation.

The EEG observations were also related to the two groups of styrene-exposed subjects with and without signs of polyneuropathy (table 3). No difference was found

Table 4. Summary of electroencephalographic findings among the various groups of subjects. The mean frequency of the occipital alpha (α) activity, as well as the percentage occurrence of increased fast activity (β), diffuse slow activity (θ), and localized abnormalities. The actual numbers of individuals are given within parentheses.

Parameter	Group				
	Normal control (N = 6)	Styrene-exposed			Mixed solvent-exposed (N = 17)
		A (N = 13)	B (N = 10)	C (N = 10)	
α (Hz)	10.0 ± 1.26	9.81 ± 0.56	9.85 ± 0.75	9.40 ± 0.94	9.46 ± 1.05
β (%)	17 (1)	31 (4)	20 (2)	30 (3)	59 (10)
θ (%)	17 (1)	0	20 (2)	40 (4)	59 (10)
localized abnormalities (%)	0	8 (1)	10 (1)	0	0

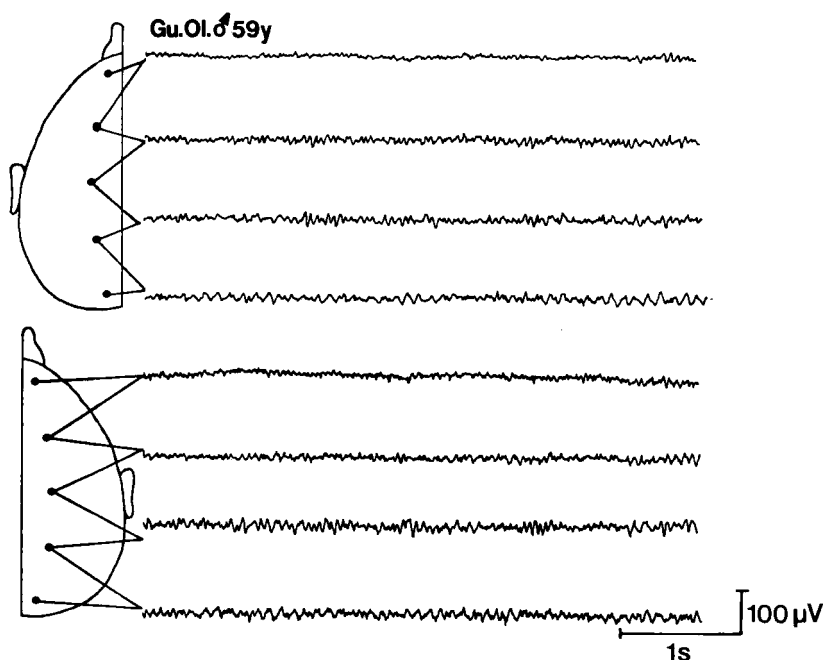


Fig. 3. Electroencephalogram from a styrene-exposed subject with an increased amount of fast activity in central and precentral areas. Additional details are given in the text.

in terms of the dominant occipital activity (α). One subject had had an EEG taken 10 years before and just before the commencement of the styrene exposure. The mean alpha frequency was exactly the same on the two occasions (11 Hz). The most striking difference between the two groups in table 3 is the occurrence of fast activity (β) within the rostral and central parts of the hemispheres. Eight out of ten in the polyneuropathy group and only one out of 23 in the normal group showed such an increased amount of fast activity. A representative EEG record from one subject in the polyneuropathy group is illustrated in fig. 3. This subject had been exposed to styrene for 10 years (group A) and was not taking any sedative or hypnotic drug.

DISCUSSION

Our study was undertaken with the purpose of finding a useful neurophysiological

test battery for the diagnosis and quantification of the neurotoxic effects of organic solvents with special consideration given to styrene. The test groups were styrene workers from three different plants with different levels of current and past exposure. The reference groups were a small group of normal subjects of comparable age and a group of patients considered by the occupational medical expert (B.H.-A.) to suffer from sequelae after heavy chronic organic solvent exposure of mixed type. Many among this latter group were former painters.

The study of the peripheral neurophysiological parameters indicates that detailed and quantified sensory neurography with needle electrode recording and signal averaging demonstrated signs of sensory neuropathy among many of the heavily exposed (MS) group and among some of the styrene-exposed subjects. The changes encountered were clearly beyond those which could be expected with increasing age even if the group of styrene workers with pathological signs had a higher mean age than the rest of the group. The analysis also demonstrated a

significantly longer time of exposure and heavier time-weighted exposure for the polyneuropathy group in comparison with the others.

Of great interest, theoretically and practically, is the possibility of synergistic effects of an aging organism and chronic styrene exposure. Returning to our material, it is worth noting that, among those with the highest degree of exposure (3) for 10 years or more, five out of six cases, who at the time of the investigation were 50 years of age or more, showed signs of neuropathy, whereas only one out of five persons under 41 years of age showed such signs. Currently much interest is being focused on free radicals, i.e., substances with a single electron in the outer shell, as mediators of endogenous cell damage. Their action is inhibited by antioxidants and "scavengers" such as vitamin E, vitamin C, and glutathione. The intracellular protective mechanisms presumably deteriorate with increasing age (11). Of great interest is the possibility that styrene or styrene oxide acts as an initiator of an increased production of free radicals as is the case for alcohol. The combination of an increased production of intracellular free radicals and a decrease of the scavenger capacity might explain a styrene-induced acceleration of the normal aging process.

The rather selective engagement of sensory neurons found in the present study suggests that sensory neurography with signal averaging and needle recording from an ideal position should be included in the neurophysiological examination of patients exposed to organic solvents. This has not previously been recognized to a sufficient degree. A possibility not tested in the present study is a change of function of slow motor fibers, although such change was not found by Seppäläinen and Härkönen (10). Selective affection of sensory neurons is common among hereditary neuropathies (2) but has also been demonstrated experimentally after methyl-mercury poisoning (12).

The EEG abnormality previously associated with chronic exposure to organic solvents is a diffusely spread slow activity (9). In the present study such changes were found in a majority of patients with

sequelae after long-term heavy exposure to mixed solvents. Also among the styrene-exposed subjects some cases with an increased amount of slow activity were found. Quantitative comparisons with other materials are very difficult to make. For example, our subjects were examined when fully awake, whereas one-third of the subjects examined by Seppäläinen and Härkönen (10) went to sleep during the examination. In agreement with these authors, we consider an increased amount of slow activity during hyperventilation a normal phenomenon (cf 8).

Particular interest was focused upon the presence of an increased amount of fast activity within the central and rostral parts of the hemispheres (fig. 3). The reason was that we have observed an increased occurrence of fast activity in EEG surveys of other groups of patients exposed to solvents even if sedatives could be excluded. We have also noticed that this activity remains for a long time despite the cessation of exposure (Ingvar and Rosén, unpublished observations). The possibility should be considered that the increased amount of beta activity is a sign of chronic brain effects produced by solvents. The observation of an increased amount of beta activity within central and rostral parts of the brain in combination with a normal and stable alpha activity within the posterior parts might indicate a regional specificity of the effects of organic solvents. A quantification of EEGs (5) recorded from multiple areas of the brain combined with regional studies of cerebral blood flow and metabolism (4) will presumably become useful tools for exploring such problems. The changes appearing in the routine EEG record after chronic styrene or organic solvent exposure are too small and too unspecific to contribute to a specific diagnosis.

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