

Neurophysiological effects of long-term exposure to a mixture of organic solvents

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SEPPÄLÄINEN, A. M., HUSMAN, K. and MÄRTENSON, C. Neurophysiological effects of long-term exposure to a mixture of organic solvents. *Scand. j. work environ. & health* 4 (1978) 304—314. Neurophysiological effects of long-term exposure to a mixture of organic solvents was studied among 102 car painters from 27 car repair garages in Helsinki. The reference group consisted of 102 age-matched railroad engineers from the Finnish State Railways. The mean age was 35 years and the exposure time ranged from 1 to 40 years (mean 14.8, SD 8.5). According to measurements the mean concentration of the solvent mixture was relatively low in the garages, namely, 31.8 % of the Finnish threshold limit value (TLV), the range of separate components varying from 4 to 212 % of the respective TLVs. The main components of about 20 organic solvents of the mixture were toluene, xylene, butyl acetate and white spirit. Electroencephalograms (EEGs) of all the 102 exposed and 102 nonexposed subjects were studied, but electroneuromyographic measurements were made of only 59 car painters and 53 referents with a similar age distribution. Motor (MCV and CVSF) and sensory conduction (SCV) velocities, as well as motor distal latencies, were recorded from nerves in the upper and lower extremities. Abnormal EEGs were encountered in 32 car painters and 37 referents. The frequency of abnormal EEGs was in both groups higher than expected on the basis of EEG literature (about 10 %). Twenty-six car painters had a complex of four common symptoms of disturbances in the central nervous system; the same symptom complex was found in 12 engineers. Forty-six percent of the car painters with this symptom complex had an abnormal EEG, while only 26 % of those without this symptom complex had an abnormal EEG. Railroad engineers did not show such a tendency. Abnormally slow MCVs or SCVs and/or prolonged motor distal latencies were found in 12 of the 59 car painters but in none of the 53 engineers studied. Other authors have stressed that many solvents primarily cause neuropathy, while objective signs of central nervous involvement have been minor, if any. Our findings are similar; they showed slight positive signs of slowed nerve conduction velocities among the car painters and no increase in EEG abnormalities in comparison to the reference group of railroad engineers.

Key words: electroencephalography, electroneuromyography, organic solvent mixture, polyneuropathy.

Exposure to mixtures of solvents is a common occupational hazard, especially in spray painting, gluing and degreasing.

Central nervous system (CNS) dysfunction in man, with electroencephalographic abnormalities after exposure to organic solvents, has been described by several authors (5, 12, 24, 27, 35). Also peripheral neuropathy in humans who have been exposed to volatile hydrocarbons (e.g., hexane and methyl-n-butyl ketone) used as solvents has been reported many times (1, 8, 16, 25, 44), and in experimental animals similar lesions have been produced (40).

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In most studies the exposure levels have been higher than the threshold limit values (TLVs) (4) or ceiling values (4) of single compounds or of mixtures of organic solvents. For example in cases of glue sniffing exposure is about 50–100 times higher than the respective TLV, although for shorter periods at a time.

The objective of this study was to determine whether long-term, low level exposure to a mixture of organic solvents can damage the nervous system. Neurophysiological methods have proved sensitive in showing the neurotoxic effects of several industrial chemicals (8, 34). We decided to apply these methods in our study, which was focused on car painters exposed to a mixture of organic solvents.

MATERIALS AND METHODS

Subjects

The subjects comprised 102 male car painters from 27 car repair garages in Helsinki. The sample was drawn from all active car painters in Helsinki ($N = 166$) by stratified sampling so that subjects with different exposure times became evenly represented (table 1). The exposure times ranged from 1 to 40 years (mean 14.8, SD 8.5); and the age from 20 to 65 years (mean 35, SD 11).

The reference group was chosen from engineers of the Finnish State Railways.

They were age-matched (± 2 years) with the exposed subjects. However there were no engineers older than 54 because the age of retirement for such workers is 55 years.

The health histories of all the subjects were collected with a questionnaire. The subjects were also given a clinical, psychological, neuroophthalmological and neurological examination, and a number of laboratory tests were performed, the results of which have been reported elsewhere (14, 26).

Electroencephalograms (EEGs) of all the subjects were recorded, but it was possible to perform the nerve conduction velocity measurements only on 59 car painters and 53 reference subjects because of a lack of personnel. The exposure time of the 59 car painters examined electroneuromyographically varied from 1 to 30 years (mean 12.9, SD 7.2), and their age from 21 to 64 years (mean 33, SD 10) (table 1).

Exposure

Solvent concentrations were measured in the air at six randomly selected car repair garages, in which a total of 40 car painters were employed. Because the exposure has been described in detail before (14), only a brief summary is included in this report (table 2).

The range of concentrations of separate components was 4 to 212 % of the respective TLVs. The variation, with time, of the 1-h average concentrations during a workday was evaluated by repeated sampling. The average standard deviation of

Table 1. Number of car painters in Helsinki and those examined with electroencephalography (EEG) and electroneuromyography (ENMG) by length of exposure.

Length of exposure (years)	Car painters in Helsinki	Car painters in the sample	
		EEG	ENMG
1–5	37	21	10
6–10	44	21	17
11–15	45	20	13
16–20	15	15	7
21–25	11	11	10
≥ 26	14	14	2
Total	166	102	59

Table 2. Mean concentrations of organic solvents in the breathing zone of 40 car painters. (Sampling period = 1 h; number of car repair garages = 6; number of samples = 54)

Solvent	Mean concentration (ppm)	Percentage of the TLV ^a	TLV ^a (ppm)
Toluene	30.6	15.3	200
Xylene	5.8	5.8	100
Butyl acetate	6.8	4.5	150
White spirit	4.9	2.5	200
Methyl isobutyl ketone	1.7	1.7	100
Isopropanol	2.9	0.7	400
Ethyl acetate	2.6	0.7	400
Acetone	3.1	0.3	1,000
Ethanol	2.9	0.3	1,000
Total		31.8	

^aTLV = threshold limit value in Finland in 1976.

the logarithms of the sum of the percentages was 0.27.

There were no marked differences in work conditions and solvent exposures between the six car repair garages since the work methods, ventilation equipment, and paints used were essentially the same. Because there were no differences and because the random sampling method was used, we assumed that the six car repair garages were representative of all the 27 car repair garages which employed the exposed workers (All garages were visited, however).

Methods

For the elimination of acute effects the neurophysiological examinations were always administered at least 16 h after the cessation of the last exposure.

EEGs of the 102 car painters and 102 referents were recorded with an 8-channel Elema Mingograph. Silver-silver chloride electrodes were placed according to the international 10–20 system, and nine different montages were utilized that consisted of scalp-to-scalp recordings, as well as of scalp-to-ipsilateral ear and scalp-to-common average reference recordings. During the main part of the recording the time constant was 0.3 s and the upper band pass filter was set at 70 Hz. The recordings lasted 25 to 30 min, and hyperventilation for 3 min and photic

stimulation for 2 to 3 min were used as activation methods.

The EEGs were interpreted according to usual criteria (18); the interpreter was unaware of whether the subject was exposed or nonexposed. The EEG abnormalities were graded according to previously published criteria (32). The occurrence of different stages of sleep was noted and assessed according to Dement and Kleitman (9).

Electroneuromyographic (ENMG) measurements were made of 59 car painters and 53 referents with a similar age distribution. The measurements included maximal motor conduction velocity (MCV) of the median, ulnar, deep peroneal and posterior tibial nerves, conduction velocity of the slower motor fibers (CVSF) of the ulnar and deep peroneal nerves, maximal sensory conduction velocity (SCV) of the median, ulnar and sural nerves in the forearm and calf regions, respectively, as well as in the distal sections (dSCV) of the same nerves from finger to wrist or ankle to foot, respectively. The motor distal latencies were also recorded.

All nerve conduction velocities were measured with the use of skin electrodes and a Disa electromyograph and stimulator according to routine methods (36, 39). During the measurements of the SCVs and dSCVs of the arm nerves orthodromically, the stimulus was applied to the finger nerve and a Nokia Pulse Analyzer was used for signal averaging (37). The

temperature around the nerve was monitored as skin temperatures at the proximal stimulation points; it was always well within the normal range of the skin area in question. No systematic difference could be detected in the skin temperatures of the car painters as compared to the referents.

RESULTS

Electroencephalography

Abnormal EEGs were encountered among 32 car painters and 37 referents. The prevalence of abnormal EEGs was clearly higher than expected for a normal population [10% according to EEG literature (18)] in regard to both the car painters ($p < 0.001$) and the railroad engineers ($p < 0.001$). The proportion of abnormal EEGs was similar in all age groups except the youngest, in which the referents had a somewhat higher frequency of abnormal EEGs (table 3). Local and paroxysmal EEG abnormalities were as frequent among the car painters as among the referents, while the frequency of diffuse abnormalities tended to be higher among the engineers.

A history of head trauma was reported by 26 car painters and 37 engineers. The subjects with a history of head injury

had abnormal EEGs in the same proportion as other subjects, namely, 8 abnormal EEGs among 26 car painters and 13 among 37 engineers.

The mean frequency of the background activity was similar in both groups, i.e., 9.7 ± 0.9 c/s among the car painters and 9.5 ± 0.8 c/s among the referents. Moreover, it was similar in normal EEGs and in EEGs showing different types of abnormalities. The main features of the organization and development of the background activity were similar, although the car painters tended to have both well-organized and poorly organized background activity more frequently than the referents, who tended to have a moderately well-organized background rhythm.

Certain psychotropic drugs induce beta activity in the EEG. Prominent beta activity in the EEGs of subjects without drug intake were noticed among 32 car painters and 29 engineers. In response to the photic stimulation the car painters showed good driving (following response in the EEG rhythms) in 35 cases of 102, i.e., slightly more often than the referents (in 24/102). Good driving was noted especially in the normal records (in 30 out of 35) of the car painters, while among the referents no such connection was found. Out of 24 EEGs with good driving, only 13 were normal in the latter group (table 4).

Hyperventilation increased the abnormality of 19 of the 32 car painters and 32 of the 37 engineers with abnormal

Table 3. Electroencephalographic findings in different age groups of 102 car painters and 102 referents.

Age (years)	Normal	Borderline	Abnormal		
			Diffuse	Focal	Paroxysmal
<i>Car painters</i>					
≤ 24	10	3	5	2	0
25—49	41	5	16	6	1
≥ 50	10	1	0	1	1
	61	9	21	9	2
<i>Referents</i>					
≤ 24	6	1	7	3	0
25—49	41	7	18	4	2 ^a
≥ 50	9	1	3	1	0
	56	9	28	8	2

^aOne referent had a slight diffuse + a slight paroxysmal abnormality.

Table 4. Photic driving in the normal and abnormal electroencephalograms (EEGs) of 102 car painters and 102 referents.

Photic driving	Car painters		Referents	
	Normal EEG	Abnormal EEG	Normal EEG	Abnormal EEG
Poor	4	6	8	4
Moderate	36	21	44	21
Good	30	5	13	11
S & W discharges	0	0	0	1
Total	70	32	65	37

EEGs. Focal abnormality increased almost invariably, namely, in 7 of 9 car painters and in all 8 referents with focal EEG abnormality. Diffuse slow wave abnormalities increased often in referents (in 24 out of 29) and in half of the car painters (11 out of 21) during the hyperventilation.

During the EEG registration sleep stage I or II (drowsiness and light sleep) was noted slightly more often among the engineers (56 out of 102) than among the car painters (47 out of 102). However, the EEGs of the referents were mainly recorded in the afternoon (on the average at 1300) after lunch and a morning filled with psychological tests, etc., while the EEGs of the car painters were mainly recorded in the morning (on the average at 1000).

The evaluation of subjective symptoms according to the self-administered questionnaires revealed a symptom complex of four general symptoms which was found more often among the car painters than among the referents. The symptom complex consisted of the following complaints: sleep often interrupted, absentmindedness, easy to fall asleep when watching TV, frequent headaches. The EEG of car painters with this symptom complex, tended to be abnormal more often (in 12 cases out of 26 = 46 %) than that of those without this symptom complex (in 20 cases out of 76 = 26 %). This difference was not statistically significant, however ($\chi^2 = 2.68$). Twelve engineers had the same symptom complex; among them three had an abnormal EEG. The EEG was abnormal in 34 cases out of 90 (= 38 %) without this symptom complex.

Electroneuromyography

The means and standard deviations of the nerve conduction velocities of the car painters and the referents are presented in table 5 and those of the motor distal latencies in table 6. The means were almost identical in both groups. Moreover, the mean nerve conduction velocities were very close to those recorded earlier in our laboratory for workers exposed to styrene and their referents (35).

In several instances individual nerve conduction velocities were lower than the normal limit of our laboratory. Abnormally slow MCVs in at least one nerve were noted in seven of the 59 car painters and in none of the 53 engineers ($\chi^2 = 4.84$, $df = 1$, $p < 0.05$). An abnormally slow MCV and/or an abnormally slow SCV of the arm nerves was found in nine car painters and none of the referents ($\chi^2 = 6.85$, $df = 1$, $p < 0.01$). Twelve car painters had an abnormally slow MCV and/or an abnormally slow SCV of the arm nerves and/or prolonged motor distal latency, while none of the engineers showed any of these abnormalities ($\chi^2 = 10.04$, $df = 1$, $p < 0.005$). Abnormally low values in the CVSF of the ulnar or deep peroneal nerves, in the SCV of the sural nerve, or in the dSCV of the median, ulnar or sural nerves were found among the car painters as well as among the referents. If all the nerve conduction velocity measures were taken into account and one point given to each abnormal value, a neurophysiological score (NF score) could be established from 0 to maximally 16; the highest score

actually recorded was 4. The car painters had higher NF scores, as can be seen from table 7.

The neurological examination revealed slight deficits in sensitivity to pain and light touch more often among the car painters than among the engineers. However, these deficits showed no correlation to the EEG findings or abnormalities in the nerve conduction velocities. On the other hand deficits in vibratory sense showed some correlation to abnormalities in the conduction velocity measures, but none to the EEG findings. Thus deficit in the vibratory sense in the lower extremities was found among car painters in six out of seven subjects with an abnormal MCV of any nerve and/or an abnormal SCV of their arm nerves. Among 28 car painters with an NF score of 1 or more, 22 persons had diminished vibratory sense in their legs, while this was the case among 12 out of 21 with an NF score of 0. Similarly, the vibratory sense in the arms was lowered in 10 out of 28 subjects with an NF score of 1 or more, while the vibratory sense in the arms was lowered in 4 out of 21 subjects with an NF score of 0. Diminished vibratory sense was noted in the arms of 4 out of 22 engineers with an NF score of 1 or more and never among the 27 engineers with an NF score of 0. Diminished vibratory sense in the legs was found in 8 out of 22 engineers with an NF score of 1 or more and in 3 out of 27 with an NF score of 0.

The medical histories of the subjects revealed that some of them suffered or had suffered from various diseases. Of these, the following were considered to have possible nervous system effects: diabetes, tuberculosis (possible side effects of the medication), epilepsy, and other neurological or psychiatric diseases. The neurophysiological findings of these cases are presented in table 8. Diabetes seemed to affect the NF score of peripheral nerve function, although one car painter with a diabetic glucose tolerance test had normal ENMG findings. However, his EEG displayed paroxysmal abnormalities compatible with epilepsy; he also had a history of convulsions. The other diseases recorded may also have affected the NF score, and the one car painter suffering

from a neurological disease also had a local slow-wave abnormality in his EEG. The subjects with these diseases were not excluded from the series, because their effects on the statistical calculations was considered negligible.

Table 5. Nerve conduction velocities (m/s) of the car painters (N = 59) and referents (N = 53).

Nerve	Car painters		Referents	
	Mean	SD	Mean	SD
Median				
MCV	59.7	5.0	61.4	5.5
SCV	62.0	4.5	63.7	4.0
distal SCV	46.5	6.0	47.1	5.3
Ulnar				
MCV	61.2	5.8	61.6	5.6
CVSF	44.4	4.3	44.3	5.2
SCV	60.2	5.2	60.5	5.0
distal SCV	53.8	5.4	54.5	5.1
Deep peroneal				
MCV	53.9	6.3	53.9	4.8
CVSF	39.4 ^a	3.6	39.7	3.3
Posterior tibial				
MCV	48.9	6.5	48.6	4.7
Sural				
SCV	48.4 ^a	5.7	49.8	6.5
distal SCV	33.4 ^b	4.8	33.0	3.4

^aN = 58.

^bN = 57.

Table 6. Motor distal latencies (ms) of the car painters (N = 59) and referents (N = 53).

Nerve	Car painters		Referents	
	Mean	SD	Mean	SD
Median	3.9	0.8	3.8	0.5
Ulnar	3.4	0.4	3.4	0.4
Deep peroneal	4.9	0.9	4.7	0.7
Posterior tibial	5.5	0.8	5.2	0.8

Table 7. Neurophysiological score of 59 car painters and 53 referents.

Group	Score				
	0	1	2	3	4
Car painters	28	19	7	4	1
Referents	35	15	2	1	0

Scores 2 and higher more frequent among car painters ($\chi^2 = 4.00$, $p < 0.05$).

Table 8. Diseases with possible neurological effects and neurophysiological findings among 102 car painters and 102 referents.

Disease	Neurophysiological score	MCV or SCV abnormality ^a	Electroencephalogram
<i>Car painters</i>			
Diabetes	4	Present	Normal
Diabetes + epilepsy	0	Absent	S & W discharges
Tuberculosis	1	Absent	Borderline
Tuberculosis	1	Absent	Normal
Cerebral contusion	1	Absent	Abnormal
<i>Referents</i>			
Diabetes	2	Absent	Normal
Tuberculosis	0	Absent	Normal
Paresis of right leg (1 year previously)	1	Absent	Normal
Tuberculosis	Not done	Not done	Normal
Neurotic disorders	Not done	Not done	Abnormal

^aMCV = maximal motor conduction velocity, SCV = maximal sensory conduction velocity.

DISCUSSION

Recently several cases of toxic neuropathy after exposure to organic solvents have been described. Many of these patients have suffered from clear neuropathy (3, 6, 13, 16, 38, 44, 45), while some patients have suffered from lesions of the spinal cord or from a syndrome of the Guillain-Barré type (25, 27). Exposure to solvents has been high in these cases. The most severe manifestations have developed among young men who have sniffed thinner (25), or after strong accidental exposure to trichloroethylene (27). Poor work conditions and lacking ventilation in small workshops have led to motor neuropathy (1, 13, 16, 38, 45). The toxic substance has often been a glue or a solvent mixture used for cleaning purposes. All these contain different chemicals, and some authors have incriminated triortocresylphosphate as the causative factor (1). However, triortocresylphosphate seems to be lacking from or present only in minimal amounts in the chemical used by most patients. Abbritti et al. (1) thus concluded that different volatile aliphatic and aromatic hydrocarbons may cause polyneuropathy.

Methyl-n-butyl ketone caused several cases of polyneuropathy (3, 6), and n-

hexane seems to have similar properties (16). Animal studies support this view (17, 28, 46). However, in many of the cases of occupational neuropathy it is impossible to identify one single causative factor, as has been critically discussed by Scala (29).

Case reports of occupational neuropathies have usually been based on clinical observations and at times also on electrophysiological findings. Nerve conduction velocities and electromyographic findings have been published by Italian authors (1, 8), and American authors (3, 6) have used ENMG methods as screening tests for cases of methyl-n-butyl ketone-induced neuropathy. The results have been used for the grading of polyneuropathy, and relationships between exposure and occurrence of neuropathy have also been elaborated.

In the present series unselected car painters and railroad engineers of similar age were studied. It should be stressed that the subjects had not come to the examinations because of any alleged occupational disease. Nevertheless nerve conduction velocity studies among them showed abnormalities in the maximal motor and/or sensory conduction velocities and/or motor distal latencies in 20 % of the car painters (12 out of 59 subjects),

while these signs were found in none of the 53 referents similarly studied. The abnormalities were slight if concluded from the fact that the mean conduction velocities of the car painters did not differ statistically significantly from those of the engineers.

Even among the railroad engineers some nerve conduction velocities were abnormally low; this was the case concerning dSCVs and CVSFs. The CVSF is a sensitive indicator of mild partial lesion in the peripheral nerves, and abnormal CVSF values can be found in a certain proportion of industrial workers without any toxic exposure (37). The distal branches of the peripheral nerves, especially the sensory fibers, are often the first site of beginning toxic neuropathy (dyingback neuropathy) (30). Slowing of dSCVs is a sign of lesion in the distal portions of sensory nerve fibers. Local vibration from hand-held tools may cause traumatic vasospastic disease and may also induce peripheral nerve lesions, especially a slowing of the CVSF of the ulnar nerve and also a slowing the dSCV of the median nerve (2, 31). Whole body exposure to vibration may have increased the prevalence of slight abnormalities in the CVSF and dSCV measured among the referents, although other possible causes for slight nervous dysfunction cannot be excluded. No evidence of neurotoxic chemical exposure was found in the work environment of the railroad engineers.

When even the most sensitive measures were taken into account, the car painters showed more abnormalities in the ENMG studies than the referents (NF score, table 7). The fact that slight peripheral nervous lesions could be shown in a group of car painters in comparison to railroad engineers indicates that even at a relatively low level of exposure a mixture of organic solvents may affect the nervous system in an undesirable manner.

Organic psychosyndrome and other psychic symptoms have been described as effects of long-term exposure to organic solvents (5, 7, 20, 45). In deliberate inhalation, acute symptoms of CNS depression arise, and in late CNS depression changes occur in the EEG as well (47). Symptoms reported by workers exposed to solvents often consist of relatively

common, nonspecific complaints which may indicate dysfunction in the CNS. These include headache, dizzy spells, memory and sleep disturbances, etc. (5, 7, 20). Although these symptoms may be purely psychogenic or neurotic in type, part of them may arise from dysfunctions or lesions in the CNS, especially in the deep structures.

Cortical lesions and dysfunctions are relatively well reflected in the EEG from the scalp, while deeply located ones may escape unnoticed. A case report from Sweden (5) described 10 patients with psychoorganic syndrome after exposure to a mixture of solvents. Six of them had abnormal EEGs, and in four cases the EEG showed borderline findings. Hanke et al. (15) studied electroencephalographically 11 floorlayers selected from a group of 32. All of these workers not only showed an abnormal EEG shortly after exposure, but also after an interval of at least one weekend.

Larger series of EEG studies on workers exposed to mixtures of solvents are few. Preliminary studies from our institute revealed an exposure-related increase in the frequency of abnormal EEGs among workers with exposure to different types of mixtures of organic solvents. In slight to moderate exposure abnormal EEGs were found in about 40 % of the cases, while among workers with an exposure level close to the TLV level or higher the prevalence of abnormal EEGs was up to 70–75 % (33). Giuliano et al. (12) published EEG findings of 125 shoe factory workers exposed to adhesive solvents. Abnormal EEGs were found in 27 % and borderline findings in 32 %. The main features were signs of diffuse brain damage, often with involvement of the mesodiencephalic structures. Studies on workers with long-term carbon disulfide exposure have shown abnormal EEGs in 37 to 39 % in both Czechoslovakia (43) and Finland (37), while in the Finnish series abnormal EEGs were found in 12 % of the reference population from a papermill. An exposure-response relationship was also found between styrene exposure and the prevalence of abnormal EEGs. At a low level of exposure to styrene abnormal EEGs were found in about 10 % of the subjects, whereas the frequency of

abnormal EEGs was over 30 % among workers whose styrene exposure exceeded a certain threshold level (35).

The present study revealed a relatively high frequency of abnormal EEGs among both the car painters and railroad engineers. The prevalence of abnormal EEGs was similar to that found earlier among workers exposed to low or moderate levels of solvents (33). The overall exposure to solvents in car repair garages was mainly low to moderate, probably not high enough to separate the car painters from the reference population with respect to EEG results. Among the car painters the frequency of abnormal EEGs was roughly one-third of the cases in all age groups; the same proportion was found among the railroad engineers in all the age groups except the youngest, i.e., age below 25 years. These young railroad engineers had abnormal EEGs in almost 60 % of the cases; the group was not large, however. This number differs greatly from the frequency of abnormal EEGs found among 185 Swedish normal, healthy adolescents (11). Among these normal adolescents 77 % showed an EEG without any phenomenon ever categorized as abnormal in the EEG literature. The criteria for abnormality in the series studied do not seem to differ from ours; e.g., EEG samples published by Eeg-Olofsson (11) and our categorizing are similar.

A history of head injury was common among car painters and railroad engineers, but those with head injury did not have abnormal EEGs more often than the other subjects. Railroad engineers had no relevant chemical exposure in their work; e.g., measurements showed that carbon monoxide levels were relatively low in their work environment (21). Railroad engineers are exposed to vibration while working in moving locomotives (41). Some authors have claimed that even local vibration may cause pathological changes in the spinal cord and encephalopathy (10, 19, 22, 23, 42), and EEG abnormalities in the form of reduced amplitude or reduced reactivity have been connected to later stages of vibration syndrome. However, it is unlikely that whole body vibration would be the etiologic factor for the EEG ab-

normalities of especially the youngest railroad engineers with the shorter exposure to vibration.

No definite EEG features separated the car painters from the railroad engineers. The frequency and type of EEG abnormalities were similar in both groups. Good driving response in photic stimulation seemed to be connected to normal EEGs among the car painters, but not among the railroad engineers. Good driving in photic stimulation may reflect good reactivity of the brain, while encephalopathy might decrease the reactivity, which would explain the finding of poor or moderate driving response in the abnormal EEGs of the car painters (table 4). At our laboratory we have recently studied EEGs of 35 factory workers without chemical exposure; 4 EEGs were abnormal and 31 normal. In another population of factory workers without chemical exposure we found 15 abnormal EEGs out of 83 examined. In our study both the exposed and nonexposed had more abnormal EEGs than the number reported elsewhere in a "normal working population" (37). Chronic exposure to organic solvents might be the reason for the car painters' abnormal EEGs, but the railroad engineers' abnormal EEGs remain unexplained.

Other authors have stressed that many solvents primarily cause neuropathy, while objective signs of central nervous involvement have been minor, if any (8, 48). Our findings of slight positive signs of slowed nerve conduction velocities among the car painters and no increase in EEG abnormalities in comparison to the reference group of railroad engineers is similar.

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