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Time course of electrophysiological findings for patients with solvent poisoning

A descriptive study

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SEPPÄLÄINEN AM, ANTTI-POIKA M. Time course of electrophysiological findings for patients with solvent poisoning: A descriptive study. *Scand j work environ health* 9 (1983) 15-24. The time course of electroencephalographic (EEG) and electroneuromyographic (ENMG) findings was studied among 87 patients (40 men and 47 women) with a diagnosis of chronic solvent intoxication after occupational exposure. Neurophysiological studies were initially performed around the time of diagnosis, and all the patients were re-examined three to nine years later. No control group was available for the follow-up. Upon diagnosis 67 % of the patients had an abnormal EEG, the majority with diffuse slow-wave abnormalities. The reexamination showed improvement in the EEGs of 47 %, but the percentage of patients with paroxysmal abnormalities had increased from the initial 6 to 17. The percentage of patients with neuropathic findings was 62 for the first and 74 for the second ENMG. Upon reexamination fibrillations were seen in 46 % (in 38 % in the initial examination), and some loss of motor units was found for 61 % (for 54 % in the initial examination). Mild improvement in the neuropathic findings was noted for 60 %, but the neuropathic findings of 25 % showed slight deterioration during the follow-up period. The EEG findings showed a time course resembling that described after external head injuries. The ENMG findings resembled those described in hexa-carbon-induced or carbon disulfide-induced neuropathy. Controlled studies are recommended to examine the observed slight associations between electrophysiological findings and the type of chemical exposure.

Key terms: electroencephalography, electroneuromyography, solvent poisoning.

Various organic solvents and their mixtures are widely used in industry. Hydrocarbons, especially hexacarbons, have been shown to cause neuropathy (10, 18, 32, 34). In addition to paresthesia, muscular pain, or diminished strength patients with long-term occupational exposure to solvents often complain of headache, memory impairment, sleep disorders, and vegetative disorders; thus central nervous system effects also occur. Experimental studies (4, 19) have revealed central nervous system effects of 2,5 hexanedione and 2,5 hexanediol, metabolites of n-hexane and methyl n-butyl ketone. Electroencephalographic (EEG) abnormalities

and evoked potential changes have been found both among patients with solvent poisoning and among occupationally exposed groups (7, 9, 20, 25, 26, 27).

Most studies have been cross-sectional ones, and little follow-up information is available (1, 5, 16, 31). The aim of the present study was to describe the frequency, the types, and the time course of EEG and electroneuromyographic (ENMG) abnormalities among persons for whom chronic solvent poisoning had been diagnosed three to nine years earlier.

Subjects and methods

From 1970 to 1974, 106 patients who had come to the Institute of Occupational Health because of neurological symptoms and suspected chronic solvent poisoning

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were diagnosed as having chronic poisoning caused by exposure to tri- or perchloroethylene or solvent mixtures. They fulfilled the following diagnostic criteria, which have been presented in more detail elsewhere (12): (i) verified exposure to neurotoxic solvents, (ii) clinical picture of organic damage of the nervous system, and (iii) other diseases reasonably well excluded. These 106 patients were invited for reexamination in 1978–1979. The 87 patients who participated in the reexamination (40 men and 47 women) comprised the subjects of the present study. The mean age of the patients at the time of diagnosis was 38.6 (range 20–59) years, and the mean duration of follow-up was 5.9 (range 3–9) years. All the patients underwent electroencephalography, and 73 of them also had an ENMG examination around the time of diagnosis. Upon reexamination, all the patients underwent both electroencephalography and electroneuromyography.

Exposure

The mean duration of exposure was 10.7 (range 1–33) years. Twenty-one patients had been exposed to trichloroethylene (9 patients) or perchloroethylene (12 patients), 53 to solvent mixtures, and 13 to both solvent mixtures and tri- or perchloroethylene. The mixed exposure of the last-mentioned 13 occurred mostly at the same worksite. The most common solvent mixtures to which the patients were exposed contained aliphatic hydrocarbons (petroleum benzine) with variable amounts (up to 20 %) of aromatic hydrocarbons, mainly ethyl toluenes and trimethylbenzenes. In most cases the patients had also been exposed to thinners, the main component of which was toluene and which usually also contained methyl isobutyl ketone, isobutanol, and ethylene glycol, or sometimes petroleum benzine, isopropanol, acetone, xylene, and butyl acetate.

The exposure of 54 patients had terminated after their intoxication had been diagnosed. The remaining 33 patients had continued to work under conditions which involved exposure for different lengths of time. Twelve of them had stopped working in solvent exposure within one year after their intoxication had been diagnosed. At

the time of the reexamination, only five persons were still working under conditions of some exposure to organic solvents.

Electroencephalography

Later in this report the initial EEG examination is referred to as EEG I, and the EEG examination upon reexamination is referred to as EEG II. All the EEG recordings were done with silver-silver chloride electrodes placed according to the international 10–20 system. The recording time was 25 to 30 min, and hyperventilation for 3 min and intermittent photic stimulation were used as the activation methods. The EEG I examinations were done with an 8-channel Elema mingograph, and the EEG II examinations with a 17-channel van Gogh electroencephalograph.

Both records of the 87 patients were reinterpreted in connection with examination II. The same neurophysiologist (AMS) interpreted all the EEGs according to general clinical principles (13).

An increased amount of theta and/or delta activity was denoted as slow-wave abnormality. Paroxysmal abnormalities consisted of sharp waves, spikes, or spike-and-wave discharges.

The EEG abnormalities were graded with a six-point scale (21). The value 6 denoted severe abnormality, 4 moderately severe abnormality, 2 slight abnormality, 1 very mild abnormality, 3 transition between 2 and 4, and 5 transition between 4 and 6 (21). If the EEG abnormalities between two recordings had changed only one point on the scale, the change was designated as slight. If the change was at least two points, the change was designated as clear. In the actual comparison both EEG recordings were visually interpreted side by side.

In order to estimate the consistency of the interpretation, a neurophysiologist from another laboratory also read a sample of 54 EEG records (EEG I and II of 27 persons). The sample was selected by one of the authors (MA-P), who first chose every fifth record. Thereafter, some selected records were used to complete the sample so that there were at least four records representing each degree of severity, each type of abnormality, and each degree of change in the abnormality. The concordance between the two interpretations was good

on the whole. Three of the original borderline findings were interpreted by the second neurophysiologist as slightly abnormal, and one originally normal finding was reinterpreted as a borderline finding. No originally abnormal records were reinterpreted as normal; only one borderline finding was reinterpreted as normal. The ratings of changes (improved, similar, deteriorated) between the two records of the same individual agreed 100 %. All the findings interpreted as paroxysmal by the first neurophysiologist were interpreted as paroxysmal by the second one also. There were minor deviations in their opinions of the localization of the slow-wave abnormalities. The first interpreter emphasized the local character of slow waves in three records, and the second in one record where the other had seen more diffusely distributed slow waves. In the Results section the results of the first interpretation (made by AMS) are considered.

Electroneuromyography

The initial ENMG examination is hereafter referred to as ENMG I, and the ENMG examination upon reexamination as ENMG II. The ENMG II examination included measurement of the maximal motor conduction velocity of the median, ulnar, deep peroneal, and posterior tibial nerves (30); the conduction velocity of the slower motor fibers of the ulnar and peroneal nerves (23); sensory conduction velocity in the forearm region (SCV); and the distal sensory conduction velocity of the median and ulnar nerves orthodromically with finger stimulation (24). Also included were the sensory conduction velocity and distal sensory conduction velocity of the sural nerve and the distal sensory conduction velocity of the radial nerve antidromically (24). These measurements were taken with a Disa electromyograph and a Disa multistim, skin electrodes being used for recording and stimulation. A Nokia LP 4840 pulse analyzer was used for the computerized averaging of the measurements of the orthodromic sensory conduction velocities and distal sensory conduction velocities. Electromyography with a concentric needle electrode was performed for the extensor digitorum communis, the first dorsal interosseus, the opponens pollicis, the tibialis anterior, and

the gastrocnemius muscles on the patient's dominant side.

The ENMG I examination included measurement of the nerve conduction velocities of four to five nerves for 92 % of the patients. Needle electromyography of several muscles was included in the ENMG I examinations for all but four patients. (All the nerve conduction velocities of these four patients had been normal.)

The individual nerve conduction velocities were scrutinized. Each patient was given a score (hereafter referred to as the NF score) based on the number of nerve conduction velocities below the lower limit of normality determined in the laboratory (24). A patient with normal conduction velocities was thus given an NF score of 0; another with, eg, three abnormally slow nerve conduction velocities was given an NF score of 3.

In the electromyography the muscles were studied during maximal and weak voluntary contraction and at rest. During maximal voluntary contraction the observed pattern was graded as an interference pattern (no loss of motor units), a reduced interference pattern (slight loss), a mixed pattern (moderate loss), or as a pattern of single motor units (severe loss) (11). During weak voluntary contraction the individual motor unit potentials were visually analyzed for their duration and amplitude. While the muscle was at rest, spontaneous activity was sought, and possible fibrillations, monophasic positive potentials, fasciculations, bizarre high-frequency bursts, or myotonic bursts were recorded.

On the basis of usual criteria, the ENMG findings were classified as normal, myopathic, rhizopathic, or neuropathic. Subjects who had normal motor unit potentials and no denervation activity although showing a reduced interference pattern in concordance with reduced force applied during their maximal contraction were considered to suffer from upper dysfunction. Neuropathy was graded as (i) mononeuropathy, ie, only one nerve involved, (ii) slight polyneuropathy, ie, at least two nerves with abnormally slow nerve conduction velocities and/or neurogenic abnormalities (mild loss of motor units, increased duration of motor unit potentials and/or denervation activity) in two or more muscles supplied by two or more nerves,

(iii) moderate polyneuropathy, ie, at least one nerve with a nerve conduction velocity 20 % less than the lower normal limit of the laboratory and moderate loss of motor units involving three to five muscles, and (iv) afterstate of polyneuropathy, ie, mild

to moderate loss of motor units or other neurogenic abnormalities in electromyography, nerve conduction velocities within the normal limits, but abnormally slow conduction velocities in previous studies. The change in ENMG was graded as (i)

Fig 1. Electroencephalographic (EEG) findings in the two examinations of 87 patients with solvent poisoning. The numerals at the top of the columns are the numbers of cases.

Fig 2. Types of electroencephalographic (EEG) abnormalities in the two examinations of 87 patients with solvent poisoning. The numerals at the top of the columns are the numbers of cases.

Fig 3. Comparison of the neurophysiological findings of the reexamination with those of the initial examination of patients with solvent poisoning. The numerals at the top of the columns are the numbers of cases. (EEG = electroencephalography, ENMG = electroneuromyography)

mild improvement, ie, the nerve conduction velocities of one or two nerves had returned to the normal range from abnormally slow values and/or a slight increase was noted in the number of motor units, (ii) clear improvement, ie, the conduction velocities of three or more nerves had returned to a normal level and/or a clear increase was noted in the number of motor units (three to five muscles with moderate loss changed to one to two muscles with only a mild loss of units), (iii) mild deterioration, ie, the previously normal conduction velocities of one or two nerves had decreased to abnormally slow values and/or the loss of motor units had slightly increased, and (iv) clear deterioration, ie, the previously normal conduction velocities of at least three nerves had decreased to abnormally slow values and/or a clear decrease of motor units was noted. If the only difference between the two ENMG examinations was a decrease or increase of nerve conduction velocities within normal limits without electromyographic changes, the ENMG findings were graded as unchanged. Similarly, if the loss of motor units was slightly changed in only one muscle, the ENMG findings were graded as unchanged.

Statistical methods

The differences between independent groups were tested with the chi-square test, or hypergeometric distribution was applied. When testing the significance of the changes from examination I to examination II, the McNemar test (29) was used.

Results

Electroencephalographic findings

The distributions of the EEG findings in examinations I and II are presented in fig 1. Most of the abnormalities were slight in degree. Severe abnormalities were rare. During the follow-up period the prevalence of slow-wave abnormalities decreased and the prevalence of paroxysmal abnormalities increased ($p < 0.01$) (fig 2). The types of EEG abnormalities among some patients changed during the follow-up period (table 1). Especially diffuse slow-wave abnormalities changed, often to normal findings, but several to paroxysmal findings. Similarly, among those with initially local slow-wave abnormalities, there were several changes to paroxysmal abnormalities. The EEG findings of 41 patients had improved, and the EEG findings of 18 patients had deteriorated. For 28 patients the EEG findings had remained similar, but 19 of these patients had had normal EEGs at both recordings (fig 3).

Electroneuromyographic findings

Actual ENMG abnormalities were found more often in ENMG II than in ENMG I ($p < 0.001$) (fig 4). Polyneuropathy of slight degree was the most usual finding. Findings compatible with root compression were rare; they were found for four patients in ENMG I and for six patients in ENMG II.

The findings of ENMG II were similar to those of ENMG I for 18 patients. Six of

Table 1. Changes in the electroencephalographic (EEG) findings of examinations I and II.

EEG findings in examination I	EEG findings in examination II				Total
	Normal	Diffuse slow-wave abnormalities	Local slow-wave abnormalities	Paroxysmal abnormalities ^a	
Normal	20	5	1	3	29
Diffuse slow-wave abnormalities	20	7	2	5	34
Local slow-wave abnormalities	7	—	7	5	19
Paroxysmal abnormalities	3	—	—	2	5
Total	50	12	10	15	87

^a Patients with paroxysmal abnormalities often had slow-wave abnormalities at the same time.

them had normal findings in both examinations. Improvement was found for 43 % of the patients, while 33 % showed deterioration, usually mild (fig 3).

The frequency of patients with abnormally slow nerve conduction velocities remained relatively similar in both the

Fig 4. Types of electroneuromyographic (ENMG) findings in the initial examination and in the reexamination of patients with solvent poisoning. The numerals at the top of the columns are the numbers of cases.

Table 2. Frequency (%) of abnormal nerve conduction velocities (NF score) in the two electroneuromyographic (ENMG) examinations of 87 patients with solvent poisoning.

Examination	NF score					
	0	1	2	3	4	5-7
ENMG I (N = 73)	43	26	16	6	7	3
ENMG II (N = 87)	38	29	18	7	2	6

examinations (table 2). In the ENMG II examination the sensory action potential of the sural nerve was not recordable in the foot area of 12 patients, five of whom had no recordable sensory action potential at the ankle either. The proportion of abnormal EMG findings was higher in examination II than in examination I ($p < 0.02$) (table 3). It is notable that even the results of ENMG II showed denervation activity (fibrillations) in some of the muscles of 46 % of the patients. ENMG I detected such findings for 38 % of the patients. Nine patients had fibrillations in ENMG I but not in ENMG II. Conversely 17 patients who had not had fibrillations in ENMG I (ten had had a normal EMG then) had fibrillations in ENMG II. The loss of motor units was also frequent in both ENMG I (53.5 %) and ENMG II (61 %).

Neuropathic findings were noted in 45 patients (62 %) in ENMG I and in 64 patients (74 %) in ENMG II. The difference was statistically significant ($p < 0.05$). Ten of the 21 patients who had had a normal ENMG I had neuropathic findings in ENMG II. Two of four patients who were interpreted to have rhizopathy in ENMG I showed neuropathic findings in ENMG II. Some improvement in the neuropathic findings was noted for 27 of 45 patients (60 %); the improvement was clear only for seven patients, one of whom had normal results in ENMG II. The neuropathic findings of 11 patients (25 %) deteriorated slightly during the follow-up period. All of them had at least four nerves studied in ENMG I (on the average 4.4 nerves), as did the other patients with neuropathy (on the average 4.5 nerves studied in ENMG I).

Neuropathic findings were usually noted in both the upper and the lower limbs. ENMG I revealed distally accentuated neuropathic findings in nine patients, and ENMG II revealed such findings in

Table 3. Types of electromyographic findings among patients with solvent poisoning.

	N	Findings				
		Normal	Loss of motor units		Fibrillations	Fibrillations + loss of motor units
			Slight	Moderate		
Initial examination	69	25	10	8	7	19
Reexamination	87	20	25	2	14	26

25 patients. The other patients with neuropathic findings also had findings in the forearm and/or the leg areas.

Correlations between the electroencephalographic and electroneuromyographic findings

All the patients had abnormal findings in either their EEG or ENMG at least once during the follow-up period. In examination I they quite often (32 patients) had both neuropathic findings and an abnormal EEG. Thirteen of 25 patients with normal EEGs had neuropathic findings in their ENMGs. The EEG abnormalities tended to improve more often than the ENMG findings ($p < 0.08$) (table 4). In examination II, 26 patients had both an abnormal EEG and neuropathic findings, whereas 38 of 51 with a normal EEG still had neuropathy.

Background characteristics and neurophysiological findings

Alcoholic habits showed no relationship to the EEG or ENMG findings. In ENMG II, for example, those patients who admitted the regular use of alcohol tended to have less neuropathic findings than those who never used alcohol or used it only occasionally.

The duration and level of exposure or the immediate termination of exposure after the diagnosis had no clear relationship to the prevalence, the type, or the

time course of the EEG or ENMG findings.

Some tendencies in the distribution of the ENMG findings were noted that were dependent on the type of chemical exposure (table 5). Those patients who had been exposed both to a mixture of solvents and to tri- or perchloroethylene tended to have neuropathic findings more often than the patients exposed either solely to tri- or perchloroethylene or to a solvent mixture without chlorinated hydrocarbons. The former patients' abnormal findings were also noted more often for both the motor and sensory nerves and both the distal and proximal portions of the nerves than the other patients' findings were. The only statistically significant difference concerned the more widespread location of the findings ($p < 0.003$, hypergeometric distribu-

Table 4. Ratings of changes in the electroencephalograms (EEG) and electroneuromyograms (ENMG) of 73 patients with solvent poisoning at the end of the follow-up period in comparison with the corresponding ratings in the initial examination. The mean length of follow-up was 5.6 years. (The rating "similar" also includes patients whose EEGs or ENMGs remained normal throughout the follow-up period.)

ENMG	EEG		
	Improved	Similar	Deteriorated
Improved	18	9	4
Similar	9	5	4
Deteriorated	9	9	6

Table 5. Electroneuromyographic (ENMG) findings and types of chemical exposure for 87 patients.

	Type of chemical exposure								
	Tri- or per- chloroethylene			Mixture of solvents			Mixture of solvents and tri- or perchloroethylene		
	Number given ex- amination	Cases of neuropathy		Number given ex- amination	Cases of neuropathy		Number given ex- amination	Cases of neuropathy	
		N	%		N	%		N	%
ENMG examination I	18	9	50	44	26	59	11	10	91
ENMG examination II	21	15	71	53	38	72	13	11	85
Location of neuropathy									
Proximal		4	27		6	16		—	0
Distal		6	40		17	45		1	9
Proximal and distal		5	33		15	39		10	91 ^a
Type of neuropathy									
Sensory		1	7		6	16		—	0
Motor		4	27		14	37		3	27
Sensory and motor		10	67		17	45		8	73

^a $p < 0.003$ (hypergeometric distribution) when compared with the other groups of solvent exposure combined.

tion). The patients exposed to both a solvent mixture and tri- or perchloroethylene also had fibrillations in ENMG II slightly more often (69 %) than the other patients (42 %) ($p > 0.05$).

Abnormal findings in EEG I were found for 47.5 % of the patients exposed solely to tri- or perchloroethylene, whereas 71 % of those exposed to a solvent mixture or to a solvent mixture and to tri- or perchloroethylene had abnormalities in EEG I ($p < 0.05$). Abnormalities in EEG II were equally distributed among all types of exposure. Improvement in the EEG findings was noted for 29 % of those exposed to tri- or perchloroethylene, whereas improvement was found for 53 % of the patients with other types of exposure ($p < 0.05$).

Discussion

The present study is descriptive, and only cautious conclusions can be drawn from its results. The main problems in the evaluation of the results concern the uncertainty of the initial diagnosis of the patients (12) and the lack of a control group during the follow-up. Even if some statistically significant differences and associations were noted, the results still should be considered as suggestive. The comparison of the separate examinations, all of which had been done by the same investigator (AMS), may be subject to some factors of uncertainty. However, in clinical practice, comparisons are always made to previous recordings, and our laboratory had well established standards already when the first ENMG I studies were performed. The good concordance between the interpretations of two neurophysiologists supports the absence of any systematic error in the interpretation of at least the EEGs. In addition the change of the EEG apparatus during the follow-up could not affect the results in such a way that it would not be differentiated in determinations made by an experienced neurophysiologist.

Although most studies of patients with solvent poisoning have been cross-sectional ones, some authors have described the short-term evolution of the disease. Often the clinical state and neurophysio-

logical findings have further deteriorated within one to four months after the termination of occupational exposure (1, 5, 31). Similar observations have also been reported for neuropathy induced by glue sniffing (2, 14, 17, 33). Even cases with initially severe neuropathy and/or myelopathy leading to tetraplegy have shown some recovery (2) although some patients have been confined to wheelchairs and one fatality has been reported (17).

Recovery to a normal or an almost normal state has been common among patients with occupationally induced neuropathy (1, 31). However the electromyographic examination of workers who had suffered from neuropathy due to toxic exposure in shoe factories still revealed a loss of motor unit potentials and fibrillations for some patients even after the motor conduction velocity had returned to the normal level (16). In addition Giuliano et al (8) reported that electromyographic abnormalities disappeared in eight of their fifteen patients with shoemaker's neuropathy, whereas the abnormalities of four subjects became slighter and those of three patients remained similar. Neurological examination also revealed some deterioration for a certain portion of our patients in the clinical reexamination (12), and mild neurological signs were frequent three to nine years after the diagnosis.

Our patients with solvent poisoning often had neuropathic findings, as well as abnormal EEGs. This result was somewhat expected since neurophysiological tests were part of the diagnostic procedures. However only 4 of the 14 patients who had undergone only ENMG II (ie, they were thus examined only several years after their diagnosis) had a normal ENMG, nine of them had neuropathic findings, and one had signs of upper dysfunction. These findings did not differ from those of the other patients.

The EEG findings were evaluated to have improved for a large portion of the patients during the follow-up period, and the change was often to normal. Especially diffuse slow-wave abnormalities improved to normal in 59 % of the cases, but some changed into paroxysmal abnormalities. Local slow-wave abnormalities also changed in some cases to paroxysmal abnormalities. Actual epileptic seizures did not appear during the follow-up

period. Thus the changes in the EEG findings among patients with solvent poisoning were similar to the changes common among patients with external head injuries, like concussions or contusions (6).

The neuropathic findings were evaluated to have improved somewhat during the follow-up period, but abnormally slow nerve conduction velocities were still frequent in ENMG II. Fibrillations were common findings even of ENMG II, a result which would indicate that some active neuropathic processes had been continuing. Conduction velocities were only slightly reduced; together with fibrillations this occurrence would suggest axonal changes rather than segmental demyelination.

On the basis of this study it is impossible to define selective differences between the effects of various solvents. Some trends were seen however. The disturbances in the peripheral nervous system tended to be more common and widespread among patients exposed to both solvent mixtures and tri- or perchloroethylene than among those exposed to solvent mixtures or to tri- or perchloroethylene alone. EEG abnormalities on the other hand were more common among patients exposed to solvent mixtures than among those exposed to tri- or perchloroethylene alone. However, the EEG abnormalities in connection with the exposure to tri- or perchloroethylene seemed to be less reversible. The present knowledge on the metabolic and pharmacokinetic mechanisms of various solvents singularly and in combination is so limited that these differences are not readily explained. Nevertheless they are interesting and deserve more extensive research.

The duration and level of exposure or the termination of exposure after diagnosis was not clearly related to the neurophysiological findings. This lack of correlation probably depends on the patients' individual susceptibility. In addition inaccuracy in the determination of the level of exposure may partly be responsible for the lack of correlation. The various aspects of individual susceptibility, like preexisting traits of ill health, may also have contributed to the fact that some patients showed deterioration while others improved during the follow-up.

It has been thought that n-hexane and some other hydrocarbons cause neuropathy which cannot pathologically be distinguished from the neuropathy associated with triorthocresyl phosphate and acrylamide (15, 18). However, Cavanagh & Bennetts (4) claimed that hexacarbon-induced neuropathy (which is characterized by the accumulation of 10-nm neurofibrils within the axons) was different from dying-back neuropathy (a primarily degenerative form) induced by acrylamide and thus resembled carbon disulfide-induced neuropathy and the hereditary disease giant axonal neuropathy (3). The ENMG findings of our study resembled those of patients with carbon disulfide neuropathy (22, 28).

Our study still detected electrophysiological abnormalities within the peripheral and the central nervous systems of patients with previously diagnosed chronic solvent poisoning. The EEG findings showed a time course similar to that followed after external head injuries, and a substantial number of patients still had abnormal EEGs three to nine years after their diagnosis. The ENMG findings resembled those in hexacarbon-induced or carbon disulfide-induced neuropathy, and abnormal findings were still frequent in the follow-up examination, with less amelioration than for the EEG findings. Some slight associations between the type of neurophysiological findings and the type of chemical exposure were also noted. Controlled studies would be motivated to focus on these questions.

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