DOW CHEMICAL U.S.A.

MIDLAND, MICHIGAN 48640

August 22, 1977

ERRATUM

July 12, 1977 report titled "Resolution of Dose-Response Toxicity Data for Chemicals Requiring Metabolic Activation: Example - Vinyl Chloride" by P. J. Gehring, P. G. Watanabe and C. N. Park, Page 8, 2nd. paragraph, line 3 should read (95% confidence limits are from .03-8.7 ppm). Enclosed is the correct version.

P. G. Watanabe

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Using the foregoing equation, a projection below the levels of exposure producing an experimentally discernible response has been made (dashed line). Assuming no threshold for the induction of angiosarcoma in rats exposed to VC, the exposure concentration producing one angiosarcoma in 10,000 rats can be calculated. The probit percent representing an incidence of 0.01% is 1.28. Substitution of this value into the equation (4) yields:

$$\text{Log } v = 1.8827$$

$$v = 76.33 \text{ µg VC metabolized/4 hours}$$

Using equation 3, the concentration of exposure to VC needed to give this value for v is 11.66 µg/ℓ or 4.6 ppm (approximate 95% confidence limits obtained by substituting the upper and lower 95% confidence limits for v in equation 3 and solving for S are 0.03-8.7 ppm). Hence, exposure of rats to 4.6 ppm VC for 4 hours daily, 5 days/week for 1 year can be expected to produce one angiosarcoma per 10,000 rats if the dose-response curve remains valid at exposures less than those producing a discernible experimental response.

jap

The Goodyear Tire & Rubber Company

Akron, Ohio 44316

February 22, 1982

Mr L Husband
Mgr Industrial Relations
Goodyear - Niagara Falls, N Y

Dear Laurel:

Would you please pass the attached on to Dr Kim for his information. The purpose is to alert him of the potential for neurologic effects from vinyl chloride exposure.

I would doubt that employees at your plant would experience these findings since the levels of possible exposure at the Niagara Falls plant are far below the exposure levels described in this article.

Best regards,

C A Johnson M.D.

Corporate Medical Director

C A Johnson, M.D.
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JOURNAL OF HYGIENE, EPIDEMIOLOGY, MICROBIOLOGY AND IMMUNOLOGY 25, 1981, No. 3, 233—243

NEUROLOGICAL CHANGES IN VINYL CHLORIDE-EXPOSED WORKERS

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Vinyl chloride (VC) toxicity for the human organism is not still fully clear. The occupational exposure to VC is linked with the development of liver hemangiosarcomas, or with other malignant processes of varying locality. Some authors diagnose changes in terms of scleroderma, universally are described roentgenologically detected lesions of interphalangeal joints and zonal osteolysis. They are described in association with Raynaud's syndrome (12, 10, 1, 4, 5 and others). Lange with his colleagues (12) describes angiologically detectable constriction of digital arteries, stenosis or partial occlusion of phalangeal blood vessels. Described are also various types of dysesthesia in fingers, particularly cold and numbness sensations. Also Byczkowska (3) reports frequent occurrence of finger paresthesia, whitening of fingers, but also of palms and soles, and other symptoms of peripheral vasomotor disorders.

Neurological manifestations are described only sporadically. Spirtas and colleagues (16) emphasize particularly the narcotic action of VC at higher peak exposure concentrations. This manifests itself by vertigo, nausea and headache pains. Mentioned are also hand paresthesiae (princkling, formication). Langauer-Lewowicka (11) analyzes also the clinical symptoms in her group of 200 examinees who showed most frequently signs of cerebellar symptomatology. She recorded frequent occurrence of headaches and sleep disorders, but also trigeminal neuralgia.

Because of a lack of more detailed neurological studies among the VC-exposed persons, we conducted field investigations among the occupationally exposed workers in a plant where there was six years before put into operation a workshop with a considerable VC hazard.

MATERIAL AND METHODS

The group of examinees consisted of [illegible] workers (263 males and 30 females), age 18—58 years, mean age 32.8 years. Of these 76 % were below 40. The average time of exposure was 2.8 years (range from 2 months to 8 years). After consultations with plant physician and plant toxicologist the group was divided into two subgroups according to the level of exposure. The group of high-risk workers, in which the tentatively established maximum allowable concentration of 10 mg/m³ had been frequently and sometimes highly exceeded, included polymerization workers and some maintenance workers (a total of 109 persons). The group of lower risk category of workers included those engaged in drying and bagging operations, but even there they were sometimes exposed to high peak exposure concentrations during cleaning and sampling operations, and those from the other plant workshops — combustion, compressors, cracking, chlorination, regeneration — where the exposure risk was relatively low (a total of 154 persons).

All the workers were examined neurologically, some of them repeatedly. A more detailed analysis of subjective complaints was performed on the basis of EOD and N5 questionnaire surveys. Electroencephalographic examination with photostimulation was made in 232 persons (255 recordings). The group of controls consisted of 46 persons without exposure to toxic substances.

RESULTS

An overview of subjective complaints is presented in Table 1. Headaches occur frequently, but they are less frequent than in the control group. The incidence of gastrointestinal disorders and other neurovegetative disorders (palpitations, retrosternal pressure sensations) are significantly higher than in controls. Psychic disturbances were observed only in those exposed.

Table 1. Overview of subjective complaints in workers occupationally exposed to vinyl chloride, in a comparison with controls

Complaints	VC — exposed		Controls	
	number	%	number	%
Headache	46	15.7	9	19.6
Sleep disorders	16	5.5	2	4.4
GIT disorders	20	6.9	1	2.2
Vertigo	3	1.0	1	2.2
Psychic disturbation	13	4.5	0	0.0
Dysesthesia	9	3.1	1	2.2
Palpitations	14	4.8	0	0.0
Total number of examinees	293	100.0	46	100.0

Significant differences were observed between the two subgroups of exposed workers divided according to the level of exposure (Table 2). The subgroup of more exposed workers showed a higher incidence of headaches and

Table 2. Overview

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Table 2. Overview of subjective complaints in workers occupationally exposed to vinyl chloride, relation to the level of exposure

Complaints	Total		More exposed		Less exposed	
	number	%	number	%	number	%
Headache	46	15.7	19	17.4	27	14.7
Sleep disorders	16	5.5	7	6.4	9	4.9
GIT disorders	20	6.9	11	10.1	9	4.9
Vertigo	3	1.0	2	1.8	1	0.5
Psychic disturbation	13	4.5	6	5.5	7	3.8
Dysesthesia	9	3.1	7	6.4	2	1.1
Palpitations	14	4.8	5	4.6	9	4.9
Total number of examinees	293	100.0	109	100.0	184	100.0

gastrointestinal disorders, and furthermore, of dysesthesia of extremities. There was observed also a certain correlation with the length of exposure (Table 3). In persons with the exposure time longer than 4 years, the incidence of headaches was double the incidence in the group with a shorter time of exposed for more than 4 years. Sleep disorders, gastrointestinal complaints, vertigo and psychic disturbances were also more frequent in those with a longer time of ex-

Table 3. Overview of subjective complaints in workers occupationally exposed to vinyl chloride, relation to the length of exposure

Complaints	Total		Exposure longer than 4 years		Exposure shorter than 4 years	
	number	%	number	%	number	%
Headache	46	15.7	24	23.8	22	11.0
Sleep disorders	16	5.5	8	7.9	8	4.2
GIT disorders	20	6.9	9	8.9	11	5.7
Vertigo	3	1.0	3	3.0	0	0.0
Psychic disturbation	13	4.5	8	7.9	5	2.6
Dysesthesia	9	3.1	8	7.9	1	0.5
Palpitations	14	4.8	4	4.0	10	5.2
Total number of examinees	293	100.0	101	100.0	192	100.0

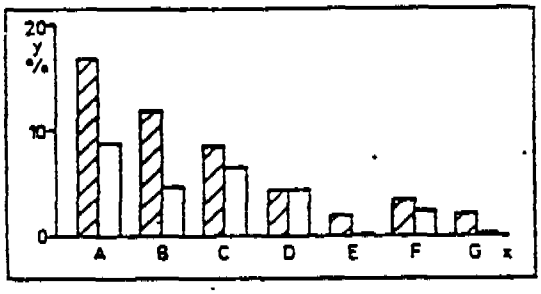
posed workers is characterized in Table 4. The group of more exposed workers showed a significantly lower per cent of normal findings and a higher per cent of more severe findings than the group of less exposed workers.

Graph 1 presents incidence of the most frequent, objectively diagnosed syndromes detected in exposed and control groups. The most frequent was the lesion of peripheral neurons, either motor or sensory, or both of them (16.8 % : : 8.7 %). Diagnosed were impairments of muscle tonus or trophicity, reduction or loss of tendon and bone reflexes, abnormal sensitivity. Compared to controls,

Table 4. Severity of objectively diagnosed changes in workers occupationally exposed to vinyl chloride in relation to the level of exposure

Severity of changes	Total		More exposed		Less exposed	
	number	%	number	%	number	%
Normal	165	57.0	50	46.0	115	62.5
Light changes	112	38.0	49	45.0	63	34.2
Manifest changes	16	5.0	10	9.0	6	3.3
Total number of examinees	293	100.0	109	100.0	184	100.0

the group of exposed workers showed also more frequently the symptomatology of peripheral neurovegetative disorders (12 % : 4.4 %), specifically acrohypothermy, acrohyperhidrosis or whitening of fingers, associated with dysesthesia.



Graph 1: Objectively diagnosed changes in VC-exposed workers in a comparison to controls. X-axis — objectively diagnosed changes: A — peripheral neuron lesions, B — peripheral neurovegetative symptomatology, C — cerebellar symptomatology, D — vestibular symptomatology, F — extrapyramidal symptomatology, G — disperse central symptomatology. Blank column — group of controls, hatched column — group of VC-exposed. Y-axis — % of examinees

Graph 2 shows objectively diagnosed symptoms in relation to the level of exposure. A marked difference is in the incidence of peripheral neuron lesions: more exposed workers are affected more than twice as often (25.6 % : 11.8 %). Celebellar and vestibular syndrome is also more frequent (10 % : 7.8 % and 6.4 % : 3.3 %, respectively). There are also certain indications of a correlation with the length of exposure, see Graph 3: those with more than 4 years of exposure have more frequently peripheral neuron lesions (22.8 % : 13.6 %) and cerebellar impairments (13.9 % : 5.7 %). Frequency of the vestibulocerebellar syndrome is also higher (5 % : 0.5 %) in these persons.

The results of EEG examinations of exposed and 81 non-exposed control workers are compared in Table 5. The per cent of abnormal EEG recordings in the group of exposed workers is higher than in the control group; the EEG abnormities detected in the controls were always least severe. Abnormities

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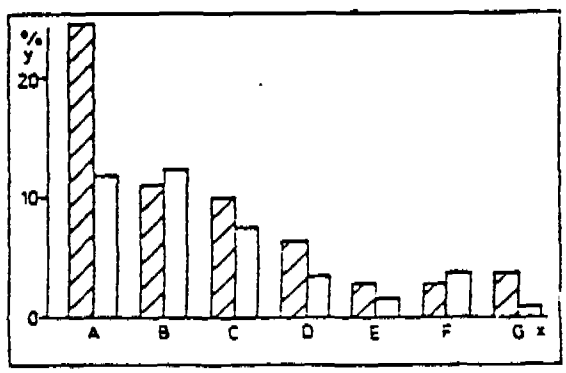
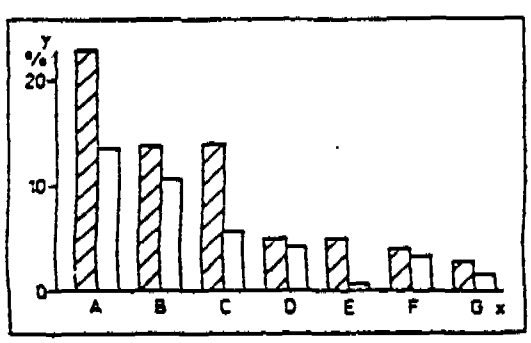
diagnosed in VC-exposed workers were predominantly episodic, in 5 cases combined with the diffuse abnormity. Three EEG recordings revealed only diffuse abnormities. Relatively frequent was also the presence of sleep waves (in 46.5 % of cases). In 16 % of workers the sleep activity manifestations were of



Graph 2: Objectively diagnosed changes in VC-exposed workers in relation to the level of exposure. X-axis objectively diagnosed changes: A-D see Graph 1. Blank column — lower exposure levels, hatched column — higher exposure levels. Y-axis — % of the total number of exposed subjects.

a higher degree of severity (2c to 3, according to Roth [13]). Significant differences were observed also in the photostimulation reaction that was normal only in 40 % of cases. The most frequent was extension of photic driving towards beta and theta waves (in 43.4 % of cases).

The additionally conducted N5 and EOD [6, 7, 8, 9] questionnaire surveys were used to improve analysis of subjective complaints and to complement



Graph 3: Objectively diagnosed changes in VC-exposed workers in relation to the length of exposure. X-axis — objectively diagnosed changes: A-D see Graph 1. Blank column — exposure shorter than 4 years, hatched column — exposure longer than 4 years. Y-axis — % of the total number of exposed subjects.

anamnestic data. The questionnaire N5 examines superficial personal traits as well as certain clinical symptomatology, particularly neurovegetative syndrome, neurasthenic, depressive and anxiety-phobic symptoms, and the so-called toxic syndrome. Data provided by this type of questionnaire were suggestive of

Table 5. Severity of EEG changes in workers occupationally exposed to vinyl chloride, in a comparison to controls

EEG changes		Exposed		Controls	
		number	%	number	%
Normal		148	63.8	57	70.4
Suspect		48	20.7	19	23.4
Abnormal	slight	30	12.9	5	6.2
	medium	6	2.6	0	0.0
	total	36	15.5	5	6.2
Total		232	100.0	81	100.0

a higher frequency of sleep disorders (36 %) than originally revealed by anamnestic data, highly frequent (5 % level of significance) was also somnolence (76 %), which had not been also indicated in personal histories. More frequent were also feelings of bad performance, fear of loosing life or health. Furthermore, the frequency of hyperhidrosis was also very high (73 %). The Eyseneck personality questionnaire examines neuroticism. It reveals subjective tendencies that are evaluated by the examinee and confronted with the objective reality. In the examined group there were not detected any significant deviations from the norm; increased neuroticism could not be demonstrated.

DISCUSSION

Clinical examination of VC-exposed workers revealed significant changes predominantly in neurologic symptomatology. Some of the subjective complaints, such as headaches, vertigo, sleep disorders or increased sleepiness during the day, as revealed by questionnaire N5, are suggestive of the narcotic action of VC, similarly as the occurrence of the cerebellar and/or vestibulocerebellar symptomatology. These changes have been already described by Spirtas and colleagues (16), Langauer-Lewowicka (11), but also by Schwartzová (15) and others. This characteristic symptomatology was also described in our previous studies concerned with the occupational exposure to trichloroethylene, benzene and other organic solvents (17, 18, 20). Here we also observed a high incidence of dysesthesia after exposures to some solvents, particularly to benzene. We ascribed it either to peripheral vasomotor changes, or — at least in some cases — to initial phases of polyneuropathy. In case of VC the presence of peripheral vasomotor changes is evidently very significant: according to literature data

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and to our own experience these changes are frequently associated with Raynaud's syndrome and may presumably lead to even more severe consequencies in terms of stenosis or occlusion, as described by Lange (12). Peripheral nerve lesions diagnosed in our group of VC-exposed examinees could be then explained by a direct neurotoxic action of VC, or as a consequence of hypoxia accompanying more severe vasomotor changes in the periphery.

The narcotic action of VC can be either transitory, inducing only reversible changes in the brain function, or persistent, causing more permanent, sometime irreversible changes in the CNS. Slight functional changes manifest themselves in EEG recordings by waves typical for various stages of sleep, as confirmed in a relatively high per cent (46.5 %) of cases in our group of examinees as well as in some of the examined subjects exposed to other organic solvents (19, 21, 22). Detection of episodic or diffuse EEG abnormalities is rather more serious and may be indicative of chronic changes in mediobasal and/or cortical brain structures. In our group of examinees, the joint episodic and diffuse abnormality occurred in 15.6 % of workers. This frequency is in agreement with the cited literature data as well as with our previous experience. This leads us to a conclusion that even VC, particularly at higher exposure concentrations, can produce neurotic changes in the above described brain structures.

VC-induced pathophysiological changes are believed by some authors to manifest themselves by the central neurovegetative dysregulation, as a result of changed hypothalamus functions (2). This localization, presumed by these authors on the basis of their experimental studies, seems to be in agreement with our findings of EEG episodic abnormalities.

We also believe that even FS reaction changes, recorded in our group of exposed workers, may be of importance in the early diagnosis of VC-induced damage. Comparable FS reaction changes were also described by Rousková (14) in persons exposed to other toxic agents.

CONCLUSIONS

1) Exposure to VC may lead, besides to other changes described in the literature, also to lesions of the nervous system. The onset and development of these neurologic changes depend on the VC exposure level and on the length of exposure.

2) Some of the neurologic manifestations are caused by the narcotic action of VC, such as certain subjective complaints and cerebellar and/or vestibulocerebellar syndrome. These symptoms can be transitory or persistent.

3) Among the important manifestations that are characteristic for VC action is, no doubt, the peripheral vasomotor symptomatology, sometimes in combination with the Raynaud's syndrome described in the literature. These vasomotor changes in the periphery may further develop, leading consequently to

more severe lesions of peripheral blood vessels. Equally important are the general neurovegetative manifestations (gastrointestinal and cardiovascular disorders, hyperhidrosis, etc.) that might result from the central neurovegetative dysregulation. Important are also symptoms of peripheral neuron lesions caused by a direct neurotoxic action of VC or by hypoxia-related mechanisms.

4) Episodic abnormality in EEG recordings seems to agree with the assumed involvement of hypothalamic structures (Basalajev and colleagues). It occurs even at exposure to the other types of organic solvents (15, 19, 21, 22) and may be indicative of a more diffuse affliction of mediobasal and cortical structures of the brain. Less severe manifestations of EEG sleep activity can be ascribed to the narcotic action of VC, more pronounced sleep manifestations accompanied with abnormal EEG changes may be suggestive of more persistent changes in the CNS.

5) Neurological changes have not been so far sufficiently accentuated in the professional literature and, therefore, the monitoring of workers at risk is not conducted systematically and by suitable methods. It is necessary to ensure a neurological prevention in these occupationally exposed workers. Of the supplementary methods of examination there are recommendable, both for prevention and research purposes, to use EEG examination with photostimulation, questionnaires N5 and EOD, and electromyographic examination.

SUMMARY

Neurological examinations were conducted in 293 workers occupationally exposed to vinyl chloride. Subjective complaints were evaluated on the background of N5 and EOD questionnare survey analysis. EEG examinations, including photostimulation, were performed in 232 persons. The control group comprised 46 nonexposed subjects. Average time of exposure was 2.8 years, the longest time of exposure was 6 years.

Among the most frequent subjective complaints were headache, neurovegetative disorders and dysesthesiae, among objective findings dominated cerebellar and/or vestibulocerebellar syndrome, lesions of peripheral neurons and peripheral neurovegetative symptomatology. Subjective and objective symptoms were found to depend on the exposure level and the time of exposure.

EEG examinations confirmed in 15.5 % of cases abnormities, predominantly episodic, sometimes combined with the diffuse abnormality. 46.5 % of the exposed showed presence of sleep activity as a consequence of VC narcotic action. The episodic EEG activity could be ascribed to lesions of mediobasal structures, or even to changes in brain cortex.

Our data have confirmed that vinyl chloride has a considerable impact on the human nervous system. Most frequent are lesions of vestibulocerebellar system and vigility disorders due to VC narcotic action. Frequent occurrence of peripheral symptomatology can be explained by a direct neurotoxic action of VC or as a consequence of hypoxia caused by peripheral vasomotor changes.

As a rule, regular check-ups of VC-exposed workers do not include systematic neurological examinations. The systematic neurologic prevention, based on the assessment of clinical, EEG and/or EMG examinations, should become obligatory. Supplementary use of N5 and EOD questionnaire surveys is highly advisable.

RESUMÉ

Stýblová, V., Lambl, V., Chumchal, O., Kellerová, V., Pašková, V., Vítovcová, J., Žlab, L.: **L'image neurologique chez les sujets exposés au chlorure de vinyle**

Il a été étudié d'une manière complexe l'image neurologique chez 293 sujets exposés au chlorure de vinyle. Des troubles subjectifs ont été analysés au plan détaillé à l'aide des anquêtes EOD et N5. Il a été mis en évidence un effet neurotoxique significatif produit par chlorure de vinyle qui dépend de la qualité et de la quantité de l'exposition subie.

Les troubles subjectifs rencontrés le plus souvent: maux de tête, symptômes végétatifs et dysesthésie. Les données objectives témoignent pour une affection du système vestibulocérébelleux et pour celle du neurone périphérique et de l'innervation périphérique végétative. La symptômatologie périphérique peut résulter de l'effet toxique direct produit par chlorure de vinyle aussi bien que du mécanisme d'hypoxie ayant lieu lors des changements vasomoteurs périphériques.

Des donnés issues de l'EEG témoignant une activité de sommeil chez 46,5 % de sujets démontrent un effet narcotique du chlorure de vinyle. L'activité épisodique (chez 15,5 %) associée parfois à l'anomalie de diffusion pourrait s'expliquer par une atteinte des structures médiobasales, même liée aux changements du cortex.

Les sujets exposés à l'influence du chlorure de vinyle ne se soumettent, jusqu'à ce jour, aux examens systématiques au plan neurologique. Il est nécessaire de poursuivre, dans ce cas, une prophylaxie neurologique étudiant l'image clinique, les données issues de l'EEG ou même de l'EMG. Il est utile d'employer les anquêtes EOD et N5.

ZUSAMMENFASSUNG

Stýblová, V., Lambl, V., Chumchal, O., Kellerová, V., Pašková, V., Vítovcová, V., Žlab, L.: **Neurologisches Bild bei den dem Vinylchlorid exponierten Arbeitenden**

Man beobachtete komplexerweise das neurologische Bild bei 293 Arbeitenden, die Vinylchlorid exponiert waren. Subjektive Schwierigkeiten analysierte man eingehender mit Hilfe der EOD- und N 5-Fragebogen. Dabei hat man eine signifikante neurotoxische Einwirkung von Vinylchlorid nachgewiesen, die von der Intensität und Dauer der Exposition abhängig ist.

Die häufigsten subjektiven Schwierigkeiten waren Kopfschmerzen vegetative Symptome und Dysesthäsie. Der objektive Befund zeugt von der Affektion des Vestibularzerebellarsystems, ferner von der Affektion des peripheren Neurons und der peripheren vegetativen Innervation. *Die periphere Symptomatologie kann man erklären* sowohl als direkte Einwirkung von Vinylchlorid, als auch den hypoxischen Mechanismus bei peripheren vasomotorischen Veränderungen.

In dem EEG-Befund stellte man bei 46,5 % Tiele der Gesamtheit die Schlafaktivität fest, die die narkotische Einwirkung von Vinylchlorid dokumentiert. Die episodische Aktivität (bei 15,5 %) manchmal in Verbindung mit Diffusionsabnormität könnte man durch Affektion von mediobasalen Strukturen, gegebenenfals durch Kortexveränderungen erklären.

Die Vinylchlorid exponierten Arbeitenden werden bisher systematisch vom neurologischen Standpunkt nicht beobachtet. Die Verfasser halten die gezielte neurologische

Vorbeugung in Verbindung mit Beobachtung des klinischen Bildes, des EEG- eventuell auch des EMG-Befundes für notwendig. Sehr geeignet ist die Anwendung der EOD- und N 5-Fragebogen.

RESUMEN

Stýblová, V., Lambl, V., Chumchal, O., Kellerová, V., Pašková, V., Vítovcová, V., Žlab, L.: El cuadro neurológico en trabajadores expuestos al vinilcloruro

Se ha examinado globalmente el cuadro neurológico en 293 trabajadores expuestos al vinilcloruro. Las dificultades subjetivos se las analizó detalladamente mediante los cuestionarios EOD y N 5. Se mostró el resultado neurotóxico marcado del vinilcloruro, el que dependía de la altura y duración de la exposición. Las dificultades subjetivas más frecuentes eran los dolores de la cabeza, los síntomes vegetativos así que la disestesia. El hallazgo objetivo muestra la afectación del sistema vestibulocerebelar, así que la de la neurona vegetativa periférica y de a inervación vegetativa periférica. La sintomatología periférica la puede explicar tanto por el afecto tóxico directo del vinilcloruro, como por el mecanismo hipóxico con los cambios vasomotóricos periféricos.

Se halló en hallazgos electroencefalográficos una actividad del sueño en el 46,5 p. c. del conjunto, lo que prueba el resultado narcótico del vinilcloruro. La actividad epizódica (en el 15,5 p. c.), a veces en la combinación con la anormidad difusa podría se explicar por afectación de las estructures mediobasales, eventualmente por cambios de la epidermis.

Los trabajadores expuestos al vinilcloruro no son aún examinados neurológicamente de manera sistemática. Hace falta que se haya realizado neurológica prevención encaminada incluso el cuadro clínico, el hallazgo electroencefalográfico, eventualmente el electromiográfico. Se recomienda usar los cuestionarios EID y N 5.

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