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 BRIEF REPORTS

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Tumors in Patients with Angiosarcoma of the Liver

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ANGIOSARCOMA of the liver is an exceedingly rare vascular tumor that has been associated with exposure to thorium dioxide, arsenic, and vinyl chloride monomer (1, 2). Using methods previously described (1), the Bureau of Occupational Health, New York State Health Department, developed a registry of all histologically confirmed cases of angiosarcoma of the liver diagnosed in residents of New York State from 1970 through 1975. Detailed medical, social, occupational, familial, and therapeutic histories on each patient were obtained from their closest living relatives and physicians. Slides of all patients included in the registry were reassessed blindly for diagnostic accuracy by a panel of pathologists. All other slides relating to diagnoses of other malignancies in these patients were also reassessed.

Of the 26 patients included in the registry, six were found to have nine histologically confirmed additional primary neoplastic tumors. Table 1 indicates the probable types and duration of exposure for these six patients. In general, persons with a first cancer are at 1.29 times increased risk overall of developing a new independent nonsimultaneous primary cancer compared with persons who never had cancer (3).

These findings may well be fortuitous, and the small number of patients (and person years) involved limits the value of statistical analysis. However, the rarity of angiosarcoma of the liver (annual incidence rates ranging from 0.14 per million [2] to 0.25 per million population) (1), the high frequency of other primary tumors observed, and the very short interval between the diagnosis of angiosarcoma of the liver and most of the other tumors observed (Patients 1 to 4, Table 1) support the hypothesis that exposure to these three carcinogens might be of etiologic importance in the pathogenesis of many of the different types of tumors observed in each patient. Moreover, evaluation of the past medical, social, and therapeutic histories of the six patients did not suggest that other factors (such as smoking or radiation) could account for these observations. None of the patients had a family history of any of the tumors listed in Table 1. Patient 1 had a sister in whom breast cancer was diagnosed; Patient 2 had a father in whom stomach cancer was diagnosed; and the father and mother of Patient 4 had diagnoses of Hodgkin's disease and myeloid leukemia, respectively.

Several mortality studies (4, 5) have found a slight to moderate excess of digestive tract, lymphatic, urinary tract, and brain cancers among cohorts with occupational exposure to vinyl chloride. Other studies (6), however, have failed to document these results. Findings of experi-

Table 1. Descriptive Data on Patients with Angiosarcoma of the Liver and Other Primary Malignancies

Patient Number	Sex	Angiosarcoma of the Liver		Other Primary Malignancies			Year of Death
		Patient Age at Diagnosis	Year of Diagnosis	Type	Year of Diagnosis	Probable Exposure History and Duration	
1	F	40 yrs	1965	Squamous cell carcinoma, left finger	1965	Lived less than 1 mile from a vinyl chloride factory (15 years)	1965
2	M	52	1968	Giant follicular lymphoma, breast	1968		1968
				Adenocarcinoma, prostate	1968	Thorotrast*	1968
3	M	68	1971	Cholangiocarcinoma, liver	1968		
				Lymphosarcoma, small intestine	1955	Unknown, chemist-teacher (1 year)	
				Transitional cell carcinoma, bladder	1971		1971
4	M	63	1975	Transitional cell carcinoma, bladder	1973	Unknown, cleaning and maintenance (40 years)	1973
5	F	43	1970	Meningioma, brain	1959	Lived less than one mile from a vinyl chloride factory (22 years)	1974
6	F	64	1973	Fibromyxoliposarcoma, knee	1969	Arsenical pesticides (45 years)	1975

* Angiography 30 years before diagnosis of angiosarcoma of the liver.

mental studies have shown that this chemical induces adenoma, adenocarcinoma of the lung, brain tumors, lymphomas, and other tumors in various animal species (7). Arsenic is generally accepted as a cause of Bowen's disease and probably certain respiratory tract cancers (8). Other neoplasms that have less frequently been attributed to chronic arsenic exposure include cancers of bladder, prostate and liver and leukemias and lymphomas (8). Chronic exposure can produce lymphomas experimentally (9). Thorium dioxide has been associated with cancer of the maxillary sinuses, meningioma, and bile duct and acute and chronic myelogenous leukemia (10).

Further study of the possible spectrum of malignant disorders associated with exposure to each of the three agents clearly is warranted. Because most patients with well-documented angiosarcoma of the liver have no documented exposure to any of these three chemicals (1), some consideration should also be given to the possibility that other agents structurally similar to vinyl chloride (such as chloroprene, trichloroethylene) might play a role in the cause of this and possibly other malignant disorders.

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Estrogen Receptor Activity in a Case of Hodgkin's Disease

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THE DISCOVERY by Jensen and colleagues (1) and others (2) of the presence of estrogen receptors in human breast cancers and the subsequent correlation of the presence of hormone receptors and response to therapy have had profound effects on the modern management of patients with advanced cancer of the breast. Estrogen receptor

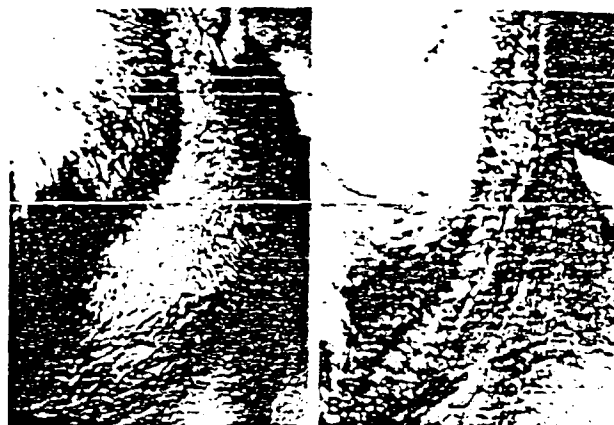


Figure 1A. Large submandibular mass present on presentation. B. Neck mass has disappeared entirely after 1 week of high-dose diethylstilbestrol therapy.

activity has also been identified in normal liver, endometrium, and pancreas; in neoplasms other than those of breast, including adenocarcinoma of endometrium, colon, and ovary; and in malignant melanoma (3). To date these observations have been of only modest therapeutic significance. Presented here is the case of a patient with Hodgkin's disease in whose tumor estrogen receptor activity was found to be very elevated and whose lymphadenopathy regressed dramatically after fortuitous estrogen therapy.

A 72-year-old man presented to his otolaryngologist with a 1 month history of a left submandibular mass. He was otherwise totally asymptomatic. Past medical history was essentially unremarkable. Findings of physical examination including careful evaluation of the structures of the oral cavity were unremarkable except for a 4- by 4- by 6-cm firm, nontender fixed mass in the left submandibular area obliterating the angle of the mandible (Figure 1A). The rest of the findings were essentially normal except for diffuse induration of the prostate extending into the seminal vesicles. He was referred for urologic evaluation: Biopsy of prostate showed poorly differentiated adenocarcinoma. Intravenous pyelogram, technetium pyrophosphate bone scan, prostatic and total acid phosphatase fractions (sigma), and alkaline phosphatase were all within normal limits. The patient had no symptoms on voiding, and a cystoscopy therefore was not done.

Diethylstilbestrol therapy was initiated at 25 mg/d as a therapeutic trial. At the end of 1 week the cervical mass had disappeared totally (Figure 1B). The dose of diethylstilbestrol was then reduced to 1 mg/d with regrowth of the mass to its original size over 10 to 14 days. Administration of diethylstilbestrol, 5 mg daily, was then begun, and although the mass did not disappear as it had done on the higher dose, it did regress significantly. However, on maintenance estrogen therapy with diethylstilbestrol, 5 mg/d, severe angina developed with T wave changes on electrocardiogram, fluid retention, and epigastric and substernal burning. Diethylstilbestrol was therefore withdrawn, and the mass enlarged to its original size. Because of intolerance to estrogen therapy the patient underwent bilateral orchiectomy. The mass did not regress, however, despite a fall in serum testosterone level to less than 10 ng/dL (normal, 300 to 900 ng/dL). During this period serial rectal examinations showed that the local prostatic lesion had completely disappeared.

Because of the inconsistent hormonal response, biopsy of the mass was done. Histologic studies showed Hodgkin's disease, nodular sclerosis (pathologic studies reviewed by Dr. Costan Berard, National Cancer Institute). Estrogen receptor analysis

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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 (18) 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 (prickling, 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 293 workers (263 males and 30 females), age 18–53 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 6 years). After consultations with plant physician and plant toxicologist the group was divided into two subgroups according to the level of exposure. The subgroup of high-risk workers, in which the tentatively established maximum allowable concentration of $10 \text{ mg} \cdot \text{m}^{-3}$ had been frequently and sometimes highly exceeded, involved polymerization workers, and some maintenance workers (a total of 109 persons). The subgroup of lower-risk category of workers included those engaged in drying and bagging operations, but even here 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 184 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 45 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 disturbance	13	4.5	0	0.0
Dysaesthesia	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 disturbance	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 exposure 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 disturbance	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

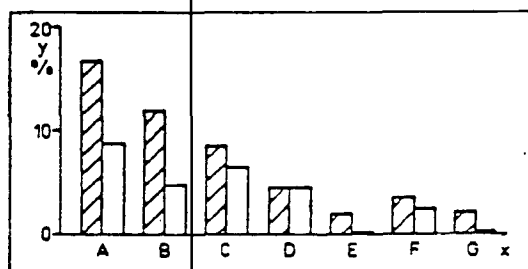
posed workers is characterized in Table 4. The group of more exposed workers showed a significantly lower per cent of normal findings and a higher per cent of more severe findings than the group of less exposed workers.

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 acrohypo-thermy, 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 %). Cerebellar and vestibular syndrome is also more frequent (10 % : 7.6 % 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 abnormalities detected in the controls were always least severe. Abnormalities

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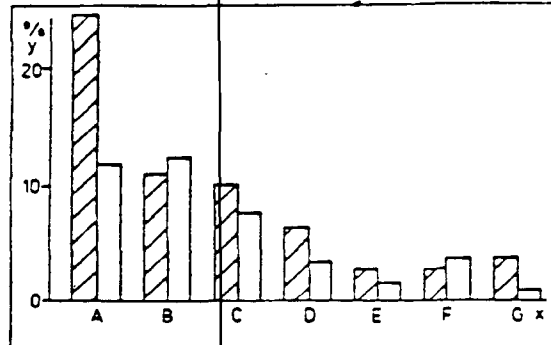
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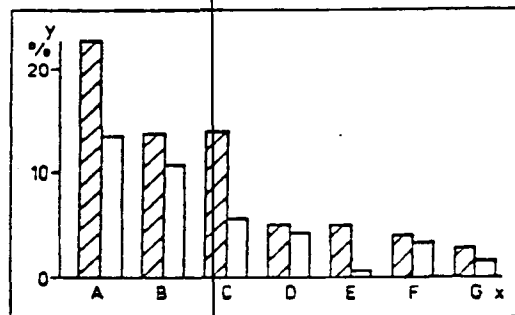
diagnosed in VC-exposed workers were predominantly episodic, in 5 cases combined with the diffuse abnormality. Three EEG recordings revealed only diffuse abnormalities. Relatively frequent was also the presence of sleep waves (in 46.5 % of cases). In 16 % of workers the sleep activity manifestations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

anamnesic 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	number	Exposed		Controls	
		number	%	number	%
Normal	148		63.8	57	70.4
Suspect	48		20.7	19	23.4
slight	30		12.9	5	6.2
Abnormal	6		2.6	0	0.0
medium	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 losing life or health. Furthermore, the frequency of hyperhidrosis was also very high (73 %). The Eysenck 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 Spirias 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 consequences 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 questionnaire 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 8 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 abnormalities, predominantly episodic, sometimes combined with the diffuse abnormality. 48.5 % 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 vigilance 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.

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RÉSUMÉ

Stýblová, V., Lambl, V., Chumchal, O., Kellerová, V., Pašková, V., Vítovcová, J., Zlab, 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 enquêtes 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 symptomatologie 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 données 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 enquêtes EOD et N5.

ZUSAMMENFASSUNG

Stýblová, V., Lambl, V., Chumchal, O., Kellerová, V., Pašková, V., Vítovcová, V., Zlab, 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 Vestibulärzerebellarsystems, 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 % Teile 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, gegebenenfalls 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.

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RESUMEN

Stýblová, V., Lambi, V., Chumchal, O., Kellerová, V., Paškova, 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 subjetivas 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 síntomas vegetativos así que la disestesia. El hallazgo objetivo muestra la afectación del sistema vestibulocerebelar, así que la de la neurona vegetativa periférica y de la innervación vegetativa periférica. La sintomatología periférica la puede explicar tanto por el efecto 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 estructuras 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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