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A NEW OCCUPATIONAL DISEASE
- CONSEQUENCES OF PROLONGED
EXPOSURE TO VINYL CHLORIDE

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Monochloroethylene ($\text{CH}_2 = \text{CHCl}$), commonly called vinyl chloride (VC), has been used in large quantities for nearly 40 years as the monomer in the production of polyvinyl chloride (PVC). Under ordinary conditions, vinyl chloride is a gas with 2.5 times the density of air, having a slightly sweet smell. With air in concentrations 4% to 22% v/v it gives rise to inflammable and explosive mixtures. (28,38)

From the toxicological point of view, it is an interesting fact that this substance was long considered only slightly toxic, resulting in severe poisoning only in cases of large concentrations, but numerous instances of disease in people chronically exposed to vinyl chloride in PVC production facilities have forced revision of that view.

Occurrences of severe VC poisoning are known both from experimental work and reports of poisoning in people under harmful conditions in industry. (11,30,38) Vinyl chloride's immediate effects are narcosis and weak irritation, resembling those of many other substances in the chlorinated aliphatic hydrocarbon group. In concentrations of 0.5 to 7.0% v/v vinyl chloride causes disturbances in the central nervous system, beginning with stupor and anesthesia, through dizziness, headache, nausea, disturbance of vision, and momentary "blacking out", all the way to actual loss of consciousness. Concentrations of 7 to 10% v/v bring about narcosis deep enough to fit the requirements of surgical operations. Concentrations around 20% v/v can result in sudden death.

Vinyl chloride has been considered as a substance suitable for surgical anesthesia, yet it was found unsafe primarily because of the possibility of its bringing about disturbances in heart rhythm. It is similar in this respect to other chlorinated ethylenes, e.g. trichloroethylene.

Besides its anesthetic effects in severe poisonings, vinyl chloride causes enlargement of the liver, as has been observed both in experimental animals and in individual cases of fatal poisoning in humans. In animals exposed to concentrations of 15% to 20% v/v swelling of the lungs has also been observed.

The long-term toxicity of vinyl chloride has been studied in conjunction with the introduction of this substance into industry, and the occupational exposure of considerable numbers of workers in polyvinyl chloride production facilities. (30,38) In consequence of exposure over a period of a few months to concentrations in excess of 100 ppm, corresponding to about 250 mg/m^3 , enlargement of the liver was observed in experimental animals, while with concentrations of 500 ppm, i.e. 0.5% v/v, or roughly 1250 mg/m^3 , enlargement of the kidneys was also observed.

The last 10 to 12 years have thrown a completely new light on the long-term toxicity of vinyl chloride. In connection with the wide-ranging adaptation of the so-called "suspension method" to the production of PVC, which creates the possibility of periodic exposure of certain groups of workers to concentrations on the order of several hundred to a few thousand mg/m^3 (41,25) there have been reports in many countries of the appearance of a new disease

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syndrome, unknown until now to occupational medicine. The symptoms of this syndrome transcend the framework of disease-induced changes observed in experimental animals. As early as 1963, Rumanian authors⁽⁴⁰⁾ described the results of observations on the clinical state of 168 workers employed in the polymerization of VC. In addition to disturbances in the nervous system and enlargement of the liver, they confirmed dysfunction of the thyroid, symptoms of Raynaud's syndrome, and changes in the skin similar to scleroderma. These authors were of the opinion that they were dealing with primary damage to the central nervous system, chiefly to the brain itself, which damage was finding expression in disturbances of the circulatory system. Later reports, mainly from western countries, concern themselves primarily with bone changes^(18,50,31,7,33,2,13,9,12). The disorder of workers employed in cleaning autoclaves where polymerization of VC takes place, is called "occupational acroosteolysis" since the most important symptom is recognized to be the transverse belt of osteolysis in the internodal bones of the palm. However, it may be pointed out that osteolysis is often accompanied by Raynaud's syndrome. The experimental work of the Italian investigator Violi⁽⁴⁷⁾, presented to the International Conference on Occupational Medicine in Tokyo (1969), had essential meaning to the further development of knowledge concerning long-term vinyl chloride poisoning, also termed "vinyl chloride disease". After year-long exposure of rats to vinyl chloride in 3% v/v concentrations, Violi succeeded in observing symptoms similar to those in human VC poisoning, i.e. osteolysis, changes in the skin, thickening of vascular walls, and additional degeneration in the brain and large musculature. Further elements essential to an overall

picture of chronic vinyl chloride poisoning were provided by clinical investigations carried out in the university center in Bonn^(19,49,34,20,25,26).

In addition to confirming symptoms already noted elsewhere, the German authors called attention to a process of fibrosis in the liver leading to an increase in pressure in the portal vein, with the clinical appearance of cirrhosis of the liver. Besides this, they advanced the suspicion that the overall disease also involves the respiratory system, leading to respiratory disturbances of the character of restrictions and partial irregularity in breathing⁽²⁵⁾. A new observation was the confirmation of thrombocytopenia.

..... The greatest anxiety was brought about by reports concerning significantly more frequent appearances, in long-time workers at PVC production plants, of a rarely encountered tumor, namely vascular sarcoma of the liver^(27,46,5,10). At present the chronic or long-term toxicity and carcinogenicity of vinyl chloride occupies the center of attention of toxicologists, specialists in occupational medicine, and investigators from other medical fields. The last two years have brought a wave of publications from various countries producing polyvinyl chloride. As background for the available literature and our own investigations at the Instytut Medycyny Pracy in Łódź, we can use the following approach of presenting the clinical aspect of the disease and offering suggestions as to its pathomechanism.

CLINICAL SYMPTOMS

Subjective Symptoms The earliest and most frequently encountered subjective symptom is oversensitivity to cold, especially in the palms and feet, as well

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as the feeling that the skin is "crawling", numbness, and weakening of the sense of touch. In advanced cases patients sometimes complain of spreading pains in the fingers. As general subjective symptoms, various authors cite sluggishness, worsening of memory, nausea, increased perspiration, vague intermittent pains in the epigastrium, headaches and dizziness, buzzing in the ears, spots before the eyes, lack of appetite, disturbance of sleep, loss of weight, and loss of sexual desire. Of course, it is necessary to take into account the coincidental convergence of VC exposure with ailments arising from other causes.

Objective Symptoms The objective symptoms of long-term vinyl chloride poisoning are manifold, embracing various organs and systems.

Changes in bone Bone changes characteristic of long-term VC poisoning, evident in radiological examination, depend on the appearance of a transverse belt of osteolysis in the ungual internode, due to which the appearance is given of dislocation or detachment of the finger tip. At times the finger tip seems to disappear and the finger gives the appearance, on an x-ray plate, of having been partially amputated. These changes can occur in individual fingers, or in all fingers of both hands. Similar changes, though more weakly manifested, are observed in the bones of the feet. Osteolysis as described above has been obtained experimentally⁽⁴⁷⁾. Histopathological studies of affected fragments of bones from extremities indicate decalcification in regions of osteolysis, transformation of bony material, and internal stratification. The hands of

workers with osteolysis have a characteristic appearance on physical examination. The finger tip is cylindrically swollen, giving the impression of being shortened. The skin of the fingers is usually bluish, thickened, and rather inelastic,^{to} which is due to a loss of dexterity.

Whether prolonged exposure to vinyl chloride leads to other changes in bone is a subject open to discussion. In persons exposed to vinyl chloride other changes have been observed which appear often in various pathological conditions, namely osteoporosis, local sclerotization, cysts, obliteration of the outlines of glenoid surfaces, and bony growths, but convincing proof is lacking for the toxic etiology of these changes. In our studies, these so-called "uncharacteristic" changes in bone correlated with the age of the subjects. It is worthwhile noting, however, that osteolysis is not peculiar to vinyl chloride poisoning and appears in other disorders, such as: hereditary osteolysis, advanced scleroderma, certain forms of leprosy, inflammations of the joints due to psoriasis, or a far-advanced form of Raynaud's disease.

Changes in the Skin In workers engaged in the polymerization of VC allergic dermatoses may appear, but these do not establish the existence of chronic vinyl chloride poisoning. The loss of elasticity already described with bone change appears, as well as thickening of the skin and the livid hue of the fingers. It is likewise possible to observe excessive keratoseness, increased perspiration of the palms, and inappropriate shape of the fingernails, i.e. convex with transverse ridges. The most characteristic changes

in the skin have the character of flat, thinly branching or knobby incrustations, somewhat distributed over the surface of the skin. These do not differ essentially in temperature or coloration from the surrounding skin. These changes are described by various authors as being similar to scleroderma. Histopathological examination of tissue samples taken from incrustations, as well as samples from areas without obvious macroscopic changes but from subjects with Raynaud's syndrome, yields the following observations: thinning of the epidermis with loss of appropriate structure; local hyperkeratosis; thickening of the actual skin through swelling and homogenization of unnucleated collagen bundles, obstruction of the pores and appendages of the skin (glands, organs of touch); simultaneously one may observe swelling in intercellular spaces, and lymphocytic flow about the vessels and among collagen accumulations. Under the epidermis were observed widened loops of capillaries with thickened endothelium; vessels of central and deeper layers of skin had clearly thickened walls^(19,25). As it appears from the above account, the histological aspect of chronic vinyl chloride poisoning has features in common with the histological aspect of scleroderma. The difference consists in the retention of elastic elements which, it is true, undergo fragmentation in both diseases. However, in scleroderma, their numbers as seen in the microscope are not diminished. To the contrary--the degenerative changes in the skin lead to apparent increase in elastic fibers, while in VC disease, besides fragmentation one observes their color dilution⁽²⁵⁾. The incrustations of the skin discussed earlier are localized mainly on the palms and inner forearms, but are also described on the face, neck and upper chest.

According to our observations, which have not, however, been numerous, obvious incrustations of the skin are expressions of significant advance in the progress of the disease. They accompany osteolysis and full manifestation of Raynaud's syndrome.

Vascular Changes

The earliest and most frequently occurring symptoms of chronic vinyl chloride are poisoning changes in the circulatory system, leading to disturbances in proper flow of blood through the tissues. These disturbances are observed above all in abducent sections of the extremities. In the beginnings of the disease, symptoms of these disturbances consist in the subjective oversensitivity to cold, while in more advanced cases Raynaud's syndrome appears with sudden loss of color, under the influence of cold, in individual or in all fingers, and even in the entire hands and feet. Loss of color is accompanied by paresthesia, and on warming, bothersome pain. Another type of reaction to cold, similar to that in vibration sickness, may be atonic opening of the vessels, leading to livid swelling. Between attacks, insufficient circulation may be observed in abducent sections of the extremities, which are characterized by bluish color and a drop in temperature. Objective, early appraisal of a vascular syndrome is very important to medical treatment. Unfortunately, the objective detection of early stages in vascular changes, in contradistinction to changes in bone and skin, involves large difficulties. The coexistence of the functional component with organic changes in the vascular system assures that suitable methods of examination

do not yield reproducible results. For the objective confirmation of disturbances in circulation to peripheral sections of the extremities, methods known from diagnosis of other circulatory disorders are adaptable dermal thermometry, cooling tests, thermography, capillaroscopy, plate counts, oscillometry, angiography. One can likewise adapt an examination of flow rates in very fine capillaries using isotopic methods.

Interesting observations were published by the German authors⁽²⁵⁾, who carried out angiography of the upper extremities in seven cases of chronic VC poisoning. Results of the angiographic examinations^(31,25) correlate with results of microscopic examination of tissue sections taken from subjects with VC disease^(19,25) and from animals given long-term exposure to vinyl chloride⁽⁴⁷⁾. Histological examination showed thickening of arterial walls, while angiographic examination showed lessening of light in the arteries of the curve of the palm and of the fingers.

The vascular syndrome in VC disease may then be connected not only with neurogenic circulatory disturbances, as is the opinion of Basalajew, et al.⁽³⁾ as well as Suciu et al.⁽⁴⁰⁾, but is likewise a manifestation of local organic changes in the vessels. Pathologically altered arteries may likewise dysfunction in a secondary fashion, due to disrupted neurological stimuli. The pathology of the angiographic aspect included cases without clinical symptoms of Raynaud's syndrome, but with oversensitivity to cold⁽²⁵⁾.

On the basis of the accumulated data to this point, we may begin to suspect that Raynaud's syndrome in chronic vinyl chloride poisoning is a manifestation of generalized angiopathy, which most obviously asserts itself in abducent segments of the extremities.

Changes in the Liver On the subject of damage to the liver in persons employed in the production of synthetic materials, including polyvinyl chloride, Soviet authors were already writing in 1949⁽⁴⁵⁾. Likewise, Rumanian authors⁽⁴⁰⁾ confirmed in 1963 enlargement of the liver in nearly 30% of the population observed by them to have been exposed to vinyl chloride./ insert Liver damage was observed in experimental work^(30,38). Nevertheless, in descriptions of VC disease over the last few years, emphasis has been placed on the bone, skin, and vascular alterations. Reports of vascular sarcoma of the liver in persons employed in VC polymerization have turned the alert attention of investigators to this organ. To the elucidation of the character of changes in the liver have applied themselves chiefly the German investigators^(34,4) who carried out complicated, specialized examinations combining scintillography, biopsy, and laparoscopy on 20 cases of suspected liver damage, and on 45 patients hospitalized out of a 120-worker complement in a plant producing PVC for many years. Analysis of this work and other similar reports⁽¹⁴⁾ allows extraction of the conclusion, that in chronic VC poisoning, it comes down to the development of fibrous alterations encompassing the area of the portal vein and liver sac, with relatively small changes in the flesh of the liver itself. Depositing of fat and other pathological aspects of Insert: This observation was confirmed in 1967⁽⁴¹⁾

the liver cells, as encountered in some reports, may be linked with disturbances in circulation with the advance of the fibrosis. Supporting this approach to the problem is the disproportion between laparoscopic evidence and clinical symptoms of pressure on the portal vein on the one hand, and the lack of or weak progress of dysfunction in the liver in laboratory tests on the other. Difficulties in appraising the ongoing disease process in the liver with the aid of commonly employed, so-called "liver tests" probably constituted the main cause of lack of early data on changes in this organ, sustained in the course of chronic vinyl chloride poisoning.

In advanced cases the bromsulphthalein test, and heightened activity of aminotransferase and other enzymes in the blood, have established themselves as additional indicators. Fibrosis around the portal vein and under the sac leads to stoppage of the circulation between the portal and the splenomegalia, the large veins of the esophagus and the bottom of the stomach, and later to dropsy. Regarding the spleen, it is not known whether its enlargement is solely due to blockage of the portal vein, or whether the organ itself is affected by the progress of the toxic disease.

Changes in Other Organs To the overall aspect of chronic vinyl chloride poisoning can be added certain symptoms, which may be expressions of disturbances in the central nervous system. Degenerative changes in the brain were found in experimental work^(47,3), chiefly in the cerebellum. Basalajew, et al. ⁽³⁾ interpret the vascular syndrome in terms of damage to the brain. In half their cases affected by skin changes, the Rumanian authors^(40,41,1)

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confirmed dysfunction of the thyroid, basing the identification of this disturbance on tests involving the granulation of iodine. When we come to the peripheral nervous system, one of the early symptoms is weakening of the sense of touch and paresthesia. These symptoms may be consequences of circulatory disturbances, but worthy of attention are the histological examinations of the skin of rats in the experiment of Violi⁽⁴⁷⁾, and of human skin sections in the German work^(19,25) where mention is made of obstruction, by connecting tissue of nerve endings and other systems localized in the skin. Changes in the nervous system and their part in the pathomechanism of chronic vinyl chloride poisoning demand further investigation. The pathogenesis of low platelet count, described by German authors^(19,34,25) as one of the easily assessed objective symptoms of the disease under discussion, is completely unknown, and the symptom itself demands independent confirmation with other methods, while the literature from other countries does not take thrombocytopenia into account. Our clinical investigations, involving mild cases of VC poisoning, it is true, have likewise not afforded significant deviations from the norm.

The suspicion of a disease process in the lungs of workers exposed to vinyl chloride, expressed as restriction in spirometric examination and partial unevenness of respiration in gasometric examination of the blood, is borne out in more than a few cases, so it is possible to draw some connecting conclusions. In addressing ourselves to this problem it is well to keep in mind that workers in VC polymerization plants are likewise exposed to poly-

vinyl chloride dust through inhalation, which may be the source of reactions, from the lungs, having no connection with chronic vinyl chloride poisoning. Instances of pneumopathy caused by inhalation of PVC dust, or hair spray preparations supported on some other synthetic polymer, are known from the literature^(24,29)

Damage to the kidneys was likewise observed in experimental work involving exposure of animals to large concentrations of VC⁽³⁸⁾. Reports of kidney damage in humans suffering from chronic vinyl chloride poisoning are lacking.

CARCINOGENICITY OF VINYL CHLORIDE

In 1971 Violi, Bigotti and Caputo⁽⁴⁸⁾ published reports of numerous instances of skin cancer, lung tumors, and osteomas in the extremities of rats exposed to vinyl chloride, in 3% v/v concentrations, for 12 months.

On the basis of epidemiological examinations, a link was proven between vascular sarcoma of the liver and long-time employment in vinyl chloride polymerization facilities. A statistical analysis from the United States, presented at the International Symposium in Lyons in 1974, shows that employment involving exposure to vinyl chloride increases the probability of contracting this type of tumor 300-fold in relation to the whole population of the United States. The time elapsed between the beginning of exposure and contraction of the tumor falls between the boundaries of 12 to 29 years.

Up to 1974, 13 cases of angiosarcoma of the liver were identified in the United States among persons employed in VC polymerization, while 6 cases were reported in Europe. Reports on the subject of links between chronic VC exposure and other tumors are lacking, but the results of the experiment of the Italian authors⁽⁴⁸⁾ have provided the stimulus for work on this problem, by investigators of various countries.

In our country, there have been no confirmed instances of vascular sarcoma of the liver, or other tumors, which would have been suspected as causal links with employment in vinyl chloride polymerization.

DISCUSSION

Long-term vinyl chloride poisoning has the character of an ailment with manifold symptoms. In spite of its embracing various organs and systems, the disease should be viewed as a whole. In all organs manifesting symptoms of the disease, changes affected connective tissue and connective vascular systems, respectively. Dermal changes in clinical and histopathological aspects testify that in chronic vinyl chloride poisoning, we are dealing with toxic collagenosis, similar to scleroderma. In advanced scleroderma there likewise appear osteolysis and Raynaud's syndrome. There are differences between scleroderma and VC disease involving not only etiology, but also the histopathological and prognostic aspects. There are no data to indicate that VC disease has immunological character. Testimony to this conclusion

is offered by negative results of LE cell examinations, examination of rheumatoidal agent fractions of gamma-globulin in the proteinogram, and immunoelectrophoresis carried out by us and other authors.

Changes in the liver are characterized by development of connective tissue especially in the vicinity of the entrance of the portal vein and below the sac. On dissection of the liver, changes in the walls of vessels and sinuses⁽¹⁴⁾ are observed. Disturbances to the function of the liver appearing rather late, after long periods of exposure, may be manifestations of toxic changes connected with the direct effects of VC on liver cells, or consequences of insufficient circulation of blood caused by development of fibrous tissue and thickening of internal membranes in the vascular system. Similar judgments may be applied to osteolysis, which may be a manifestation of neurogenic disturbance of normal processes, insufficient supply of blood, or indeed direct influence of the toxic agent on the transformation of materials in the bones.

The pathomechanism of the vascular syndrome likewise requires elucidation. Angiographic and histopathological aspects indicate thickening of the walls of small caliber arteries and lessening of their light. From examinations of this type it turns out that central dysregulation does not explain the entire mechanism of the vascular syndrome in VC disease. Moreover the fact presents itself, that the capillaries are not the only organic foundation for insufficient blood supply. The vascular syndrome in chronic vinyl chloride poisoning seems to be a generalized angiopathy equally in the anatomical and function-

al senses.

In the present state of knowledge regarding the disorder consequent to chronic exposure to vinyl chloride, the opinion may be expressed that the pathomechanism of this disease and its individual symptoms is completely unknown, and its clinical aspect is itself open to discussion on many points. For example, the matters of changes in the blood-producing system and the respiratory organs require elucidation. It is not known, moreover, in what relationships the individual syndromes of symptoms stand to one another. Can an isolated aspect of the liver appear without the other symptoms of chronic VC poisoning; do the changes associated with fibrosis in the liver precede the appearance of vascular sarcoma, or does the tumor arise independent of the development of other symptoms of VC poisoning, and is this independent of the development of fibrosis as well?

It would be possible to pose many questions, beginning with the metabolism of VC in the body and ending with prognosis, for which there are no answers as yet. The rather short time during which systematic observations have been made of persons affected, does not allow a firm statement on the subject of prognosis in chronic vinyl chloride poisoning. Observations are noted, in the literature heretofore available, of gradual subsiding of the changes in bone, skin, and vascular systems^(41,39) on cessation of exposure. Prognosis in vascular sarcoma of the liver is always unthinkable, and whether changes in working conditions or complete cessation of exposure help prevent the appearance of the tumor or lengthen the period of latency, are questions

which lend of no firm answers yet.

On the global scale, polyvinyl chloride is produced, transformed, and used in enormous quantities daily, and this may constitute a source of distribution for small quantities of vinyl chloride trapped with the polymer or perhaps arising from depolymerization. The problem thus arises not only of occupational exposure of workers producing PVC, but of the pollution of the environment and exposure of the entire populations of civilized nations to trace amounts of vinyl chloride. The chronic VC poisoning syndrome, disquieting in its clinical aspects, uncertain in prognosis, and with its carcinogenic aspect established, has caused widespread interest among scientific centers and in public opinion around the world. This produces certain chances for the elucidation of uncertainties and the taking of protective steps. The first step on the road to protection and prevention in the USA and Western Europe was changing the mandatory hygienic standards. At first, the concentration in air of VC considered safe was taken to be 500 ppm, i.e. 1250 mg/m^3 . In 1972 this maximum allowable concentration was lowered to 200 ppm and in 1974 to 50 ppm in the USA and 25 ppm on the European continent. In October, 1974 new standards were proposed, amounting to $1 \text{ ppm} = 2.5 \text{ mg/m}^3$. This standard aroused the opposition of producers as being technically unattainable. The nations of Western Europe variously adopted themselves to the American standard; in some the M.A.C. has been lowered and directives issued requiring the use of masks with aspirators in work involving higher concentrations of VC, while in others the matter is still in the discussion stage.

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In the Soviet Union and in Poland, from the beginning, the M.A.C. for vinyl chloride has been 30 mg/m^3 , corresponding to 12 ppm. Evaluating that figure from the point of view of risk of causing chronic VC poisoning, it seems safe. In the USA, operating under the 50 ppm figure, there have been no reports of poisoning. The chemical industry has the urgent task of discovering and quickly developing such methods of PVC production, as will guarantee VC concentrations within the bounds of safe values of M.A.C., and will guarantee the purity and stability of the polymer and nonpollution of the environment surrounding the production facility.

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Translation of the German, French, Italian, Polish, Romanian and Russian titles in the references.

Note: i wsp = and co-workers.

1. Clinical and pathological consideration on Raynaud's syndrome in vinyl workers.
2. English.
3. Pathogenesis of changes which take place upon prolonged exposure to vinyl chloride.
4. Sequence scintillography investigations of the liver and spleen in patients with VC-disease.
5. English.
6. Raynaud's syndrome in those employed in PVC production.
7. Acroosteolysis of occupational origin and of new observation (constant) in France.
8. - 14. English.
15. Chromosome investigation in exposure to vinylchloride.
16. Observation on certain biochemical modifications in workers in the vinyl-chloride synthesizing industry.
17. Hazards of vinyl chloride.
18. English.
19. Sclerodermal skin changes, Raynaud's syndrome and acroosteolysis in the PVC-producing industries.
20. The so-called vinyl-chloride disease.

21. Scleroderma and connective tissue problems related to it.
22. Acropathia extremicatum in vinyl chloride polymerization - a new occupational disease.
23. English.
24. New occupational diseases.
25. The so-called vinyl-chloride disease, - a new occupational system sclerosis?
26. On the occurrence of angiosarcomas of the liver in two workers in the PVC-manufacturing industry.
27. English.
28. Chemistry and toxicology of plastics.
29. Damage to health by plastics and their intermediate products.
30. English.
31. Acroosteolysis of occupational origin.
32. Unusual syndrome of acropathy as a new occupational disease.
33. English.
34. Chronic toxic liver damage in workers in PVC production.
35. English.
36. Liver and glandular damage (or lesions) of workers occupied in the production of certain types of plastics.
37. Infrared thermography in the diagnostics and monitoring of consecutive effects in operations in the polymerization of vinyl chloride.
38. Toxicology of vinyl chloride.
39. Band-shaped osteolysis in the end digits of the hand skeleton.
40. Contribution to the study of "imboluavirilor" produced by vinyl chloride.
41. Study of illnesses due to vinyl chloride.
42. Plant medical care and technical protective measures in PVC manufacturing plants.
43. Occupational medical ideas concerning the so-called vinyl-chloride disease.
44. Retrospective survey on the mortality and morbidity after VC exposure.
45. Working conditions and measures to improve hygienic conditions in the production and uses of vinyl chloride plastics.
46. - 48. English.
49. Dermatoses in the plastics industry. Occupational dermatoses.
50. English.

* Could not find this Romanian word. - Translator.