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#251. INTOXICATION BY VINYL CHLORIDE POLYMERS AND/OR THEIR ADDITIVES

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Translation of: "Zur Intoxikation durch Vinylchlorid-Polymerisate und/oder deren Begleitstoffe," Z. Haut-Geschl. Kr., Vol. 48, No. 11, 1973, pp. 425-436

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The complex pattern of damage as the result of handling a plastic became known to us when a situation arose involving difficulties in differential diagnosis. The synopsis of the individual findings, job history data, and the literature on the subject enabled us to discover an occupational disease caused by long-term skin contact with vinyl chloride polymers (PVC) and/or their additives.

Juehe and Lange [6] published in 1972 a short article on "Skin Changes of the Sclerodermal Type, Raynaud's Syndrome and Acroosteolysis in Workers in the PVC Manufacturing Industry" (see also [7]). They were able to show that nothing of the kind had been reported in the German literature (see also [15]).

The case histories we have chosen as examples should contribute to propagating knowledge of damage caused by a particular set of industrial circumstances in the attempt to ensure that intoxication by vinyl chloride polymers and/or their additives, which occurs today with variegated symptoms, including extracutaneous symptoms, no longer escapes diagnosis — as we must suppose it does at the present time.

Patient observation: Mr. E. B., Identification No.: 22 28 06 021; telephone installer, age 50, admitted to FUB Steglitz Skin Clinic in December 1972; admission No. 1376610.

Case history: First difficulties about 4 years ago on the hands: swelling mainly on the left, paresthesia, weakness; continuous intensification of these symptoms over the years, increasingly painful radiating sensations in the hand and forearm. Two years ago, clear tendency to hardening of the thus far edematous swellings of hands, in particular the forearms, with discomfort upon moving fingers and hands and motoricity when writing; whitish finger color after cold stimulus. Arthralgia at the lower extremities, discomfort of the upper abdomen, severe discomfort through sinistral acoustic phenomena such as hissing and whistling, etc., with damage to the right ear and hearing impairment which had been present since childhood. For years, symptomatic treatment under the diagnosis of a rheumatic disease.

Hospitalized for in-patient investigation with progressive sceloderma suspected.

Skin findings upon admission: Both hands with tight, pronounced, pittable edema, more distinct on the left than on the right side, and the skin was firmly attached to the underlying tissues; diffuse skin patches; pileous areas unaffected. Pale whitish-yellowish appearance of the integument of the hands, striking intensification of the pale color tone when the fist was clenched. Clear

dermatosclerosis of both forearms with relatively sharply defined ramifications of these phenomena toward the distal upper arm. Remaining integument and frenulum of the tongue without striking changes; in the center face and over the cheek bones discrete telangiectasis which apparently had existed for some years (Figures 1 and 2).

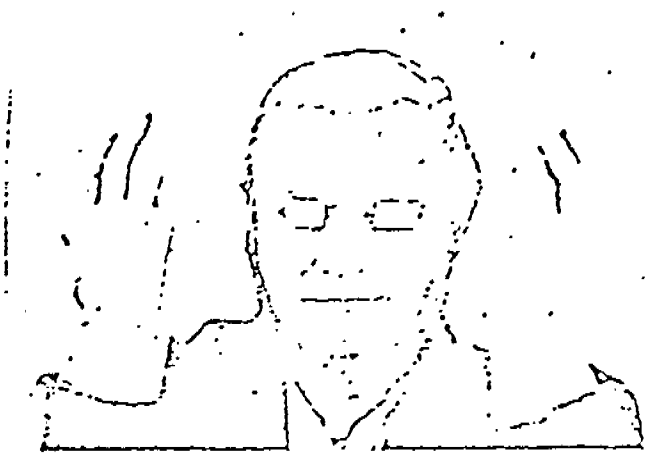

Figure 1. Edema of the Hand Which Can Still be Pitted, Skin Attached to Underlying Tissues. Pale color of the region involved, face and frenulum of tongue without changes.

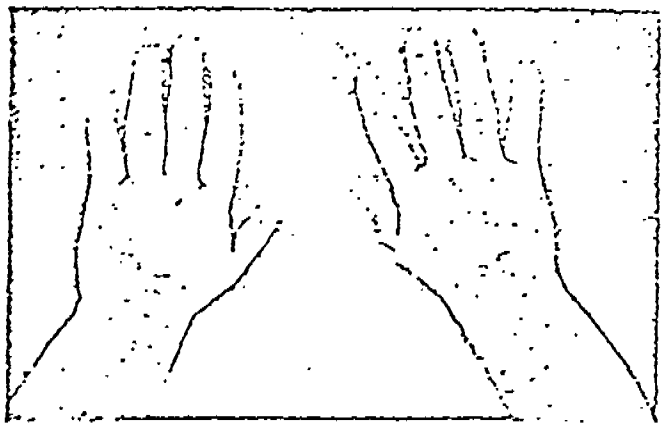

Figure 2. Edema of the Hands More Noticeable on the Left Than on the Right, Diffuse Skin Patches, Pileous Regions Predominantly Intact. Dermatosclerosis on both forearms.

Tentative diagnosis upon admission: Progressive scleroderma: edemotous or sclerosing stage. Disease of the rheumatic type with pseudoscleroderma in the upper extremities.

Further findings:

No abnormal results or values in the following: ESR/differential blood count/electrophoresis/IV glucose/IS uric acid/IS total cholesterol/IS triglyceride/IS creatinine/IS GOT/IS GPT/IS alkaline phosphatase/Quick test/IS iron/IS CPK/ urine status/total porphyrin, porphobilinogen delta-aminolevulinic acid, hydroxyproline in the urine/IS sodium/IS potassium/IS total calcium/IS inorganic phosphate/Wassermann reaction/rheumatoid factor/AST/Quantitative immunoglobulin/C_3 and C_4 complement/antinuclear antibodies/direct immunofluorescence investigation of the skin area involved for IgG, IgA, IgM, C_3, fibrin inclusions.

X-ray investigation: Area kymogram of heart/thoracic organs/shoulder joints, elbow joints, knee joints, ankle joints, and iliosacral joints, esophagus/ /gastroduodenal tract/excretion urogram/kidney x-ray/EKG/lung function test/ /capillaroscopy.

Neurological and ophthalmological consultant investigation.

Pathological results:

X-ray investigation: Both hands with striated translucence near the joints in the region of the proximal and middle phalanges, arteriography of

the hand: all finger arteries visualized only after intra-arterial administration of Priscol; no morphological vascular changes.

Visual lymphography of the upper extremities: Condition of primary lymph nodes more obvious on the left than on the right.

EMG: Somewhat myopathic form of the mU potential of the digital flexor, attributable to the edema.

Histological investigation (file No.: 1563/72; He[1]/vG[1]/tunica elastica/ /toluidine blue/ alcian blue/Giemsa (Figures 3 and 4): epidermis with lamellar orthohyperkeratosis, circumscribed acanthosis and [Translator's Note: 4 words illegible] cutis and subcutis clearly edematized, occasional visualization of metachromatized material. Impressive damage of the tunica elastica with rupture of fibers and swelling of fiber fragments. Broadening of the collagen bundle of fibers and dissociation of the connective tissue by the interstitial edema. In the subepidermal region striated zones with incipient homogeneous visualization of the connective tissue. Striking packing of individual dermal arterioles, sometimes broadened and evident homogenized media of smaller vessels, with umbonate prominences, [Translator's Note: half of a line illegible]. There was sometimes a perivascular round-cell proliferation, consisting mainly of lymphocytes, with infrequent histiocytes and mast cells. Dilated lymph capillaries. Pronounced swelling of dermal nerves, with clear formation of vacuoles inside them. Appendages of the skin without change.

Figure 3. Edematized Corium, Dissociated and Sometimes Widened Collagen Bundles of Fibers. Thickened tunica media of smaller vessels, swollen endothelia; perivascular round-cell proliferation. Dilation of lymph capillaries. Large increase in volume of dermal nerve cords. Magnification X 90.

Figure 4. Increase in Volume of Fiber Bundles, Clear Vacuolization. Magnification [Translator's Note: a 3 digit number beginning with 7, last 2 digits are illegible].

[1]Translator's Note: expansion unknown.

Electron microscope findings: Considerable lymph dilation with a high degree of flattening of the endothelium and thickening of the lymph (Figure 5a). High degree of interstitial protein-rich edema with relaxation and, in parts, considerable dissociation of the previously paraplastic substances (Figure 5b); interfibrillar edema of the collagen fibers, no destruction of collagen (average period approximately 640 Å), swelling and local destruction on and in the elastic fibers (moth-eaten appearance) and clumping of elastic fiber material (Figures 6a and b). [Translator's Note: illegible word] blood vessels (arterioles and capillaries) with swelling of the endothelium, sporatic small perivascular round-cell infiltrates (lymphocytes and histiocytes), also disseminated mast cells, partially triggering the typical granula. Pronounced perineural and endoneural edema (Figure 7a): axon edema in medullated and non-medullated nerves, also local destruction and vacuoles in the axon. No destruction in the myelin sheath, no cell reactions (Figures 7b and c).

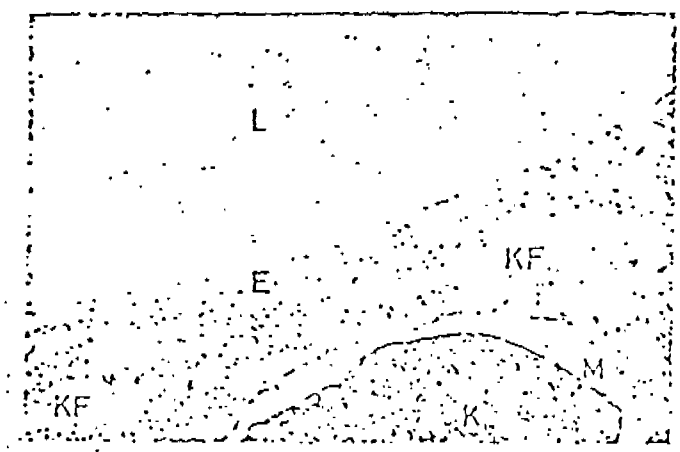

Figure 5a. Electron Microscope of a Strongly Dilated Lymph Capillary Filled with Thickened Lymph (L) with Considerably Flattened Endothelium (E). Pericapillary slanting and diagonal sections of collagen fibrils (KF), moderately intense interfibrillar edema. At the bottom, section of a mesenchymal cell (M) with nucleus (K). Electron microscope magnification X 16,000.

Figure 5b. Electron Microscope Magnification of a Section of the Corium: Considerable Protein-Rich Edema (EW) with Dissociation of the Previously Existing Fibers and Fibrils. Destruction in and on the elastic fibers (EF) looking moth-eaten. Electron microscope magnification X 12,800.

Audiogram: December 1972 combined bilateral hearing difficulty; late January 1973 clear damage to the cochleae as well.

Further x-ray investigations: vertebral block, cervical vertebrate 2/3; osteochondrosis and spondylosis deformans C5 to C7; slight degree of scoliotic false posture, slight osteochondrous changes and spondylosis deformans of the thoracic vertebrae; marked changes in the area of L 5 to S 1; upper and lower jaw merely lacunar.

Orthopedic consultant investigation: Incipient degenerative changes of the spine; no connection could be made with the skin changes.

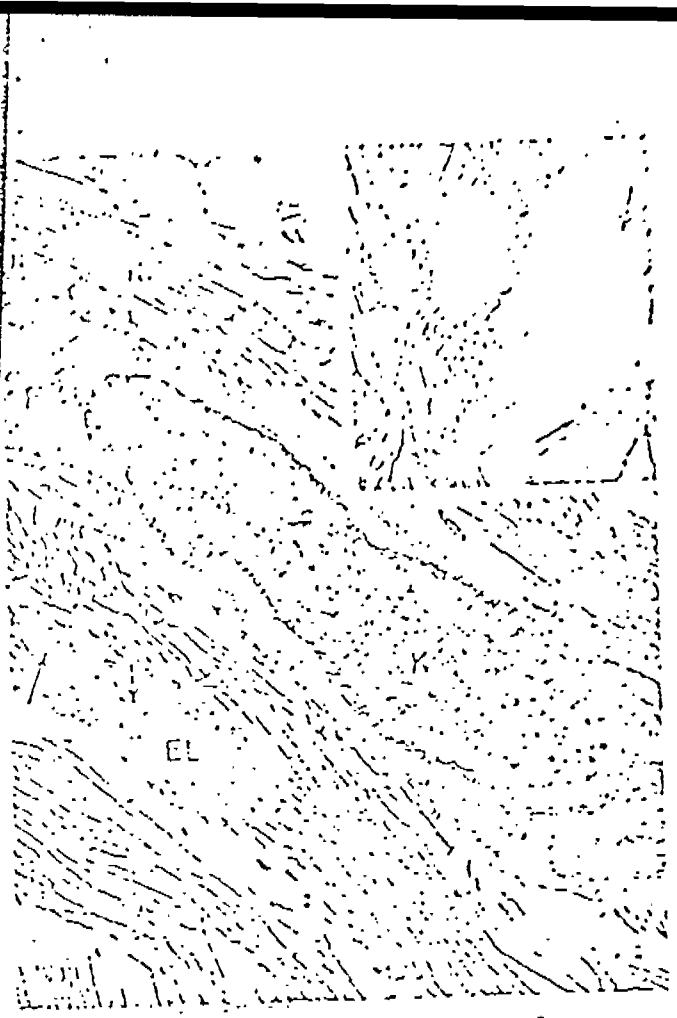

Figure 6a. (Upper Right) u.b.[2]: Electron Microscope Magnification of a Section of the Corium: Considerable Interstitial Edema with Dissociation of Collagen Fibrils (KF) and Elastic Fibers (EF). Cytoplasm and karyoplasm (K) of the fibrocytes (F) normal. Electron microscope magnification X 12,000. Inset: Magnification of destruction in and on the elastic fibers (↑). Electron microscope magnification X 19,000.

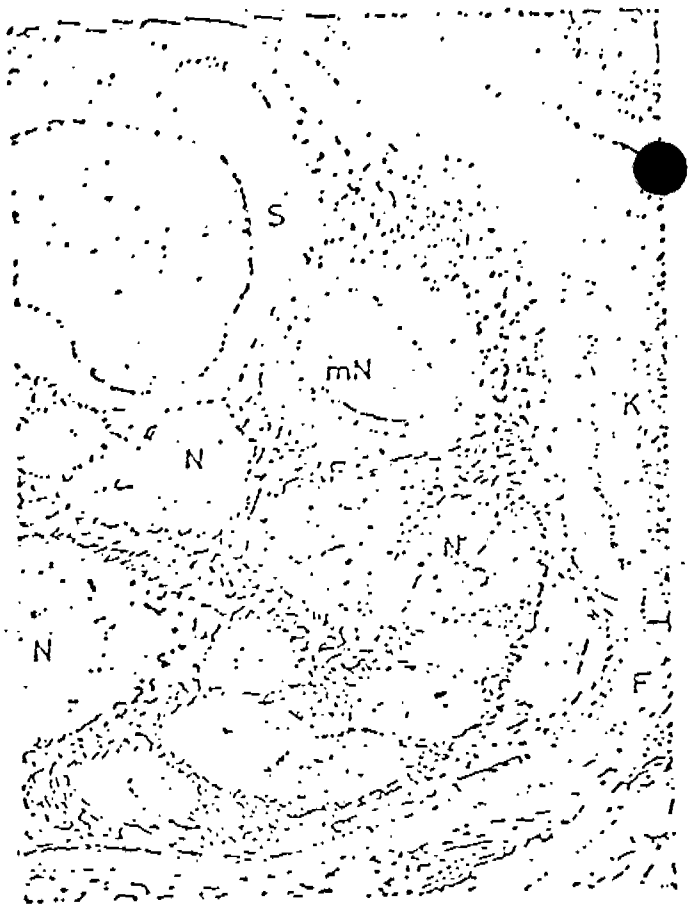

Figure 7a. Electron Microscope Magnification of a Section of the Corium with Medullated (mN) and non-medullated (N) nerves: clear perineural and endoneural edema. Schwann's cells (S) are normal. Perineurally, one normal fibrocyte (F) and collagen fibrils (KF). Electron microscope magnification X 16,000.

Tentative diagnosis: Edema or dermatosclerosis of the upper extremities, of unclear etiology.

Job background: For the past ten years had been laying polyvinyl chloride (PVC) pipe. Sealing the pipe with thin fluids' (PVC glues containing e.g., butynol and tetrahydrofuran (Tangit glue made by Henkel). The glue was applied with a rag held in the left hand so that the glue flowed more intensively over the left hand and forearm than the right, and a layer of adhesive film accumulated. After work, this was removed either by rubbing or with butanol or methylene chloride.

Final diagnosis: Intoxication by vinyl chloride polymers and/or their additives.

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[2]Translator's Note: expansion unknown.

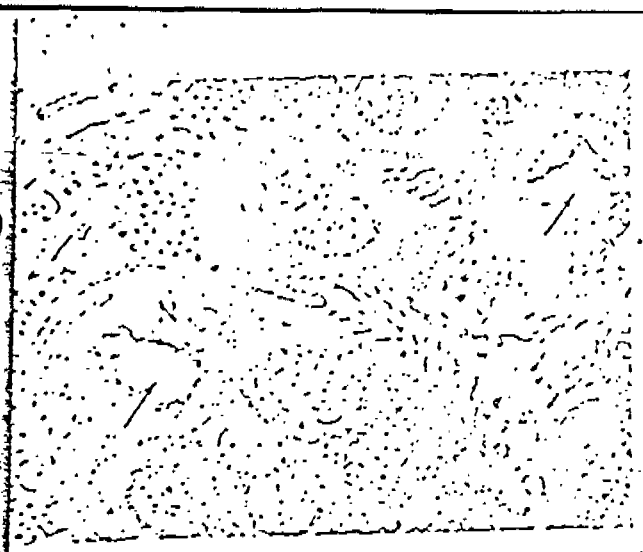

Figure 7b. Electron Microscope Magnification of a Cross Section of Non-medullated Nerves (N) of the Corium: Local Destruction (↑) in the Axon and Marginal Thickenings. Electron microscope magnification X 40,000.

Figure 7c. Electron Microscope Magnification of Section of Non-medullated (N) and Medullated (mN) Nerve from the Corium with Axon Edema (↑). Myelin sheath (My) intact. Electron microscope magnification X 26,400.

Epicrisis: The 50-year-old telephone installer fell sick about 4 years ago with swelling, paresthesia and weakness of the hands, especially the left hand; these changes became more pronounced in the course of time; painful sensations appeared in the hand-forearm region; tendency to edematous swelling leading to sclerosing; fingers turned whitish when scratched. For years, unsuccessful attempts at treatment with the disease of a rheumatoid disease, admitted for in-patient treatment with the tentative diagnosis of progressive scleroderma. When he was admitted to the hospital the skin changes showed signs of the edematous and sclerosing stage of progressive scleroderma, they also pointed to so-called pseudoscleroderma [5] with various basic conditions, e.g., a disease of the rheumatoid type. The laboratory diagnosis and morphological and functional findings, taken on the basis of suspected progressive scleroderma or rheumatoid disease, ruled out the tentative diagnosis and pointed to damage by vinyl chloride polymers and/or their additives on the basis of job history and pertinent literature.

Discussion

The course and symptoms of the disease of our patient fitted well into the variegated clinical spectrum of poisoning by PVC and/or additives, as may be gathered from the literature on the subject [1, 3, 6, 7, 8, 14, 15, 16]: with (probable) dependence on factors given by the individual type of work, changes appear with a specific set of subjective symptoms (Table 1), functional (Table 2), and morphological changes (Tables 3, 4, and 5), with differential accentuation and for overlapping of individual symptoms in accordance with rules which are not initially clear. Incorrect diagnosis of the changes, i.e., ascribing them to a rheumatoid disease or progressive (acro-)scleroderma is frequent; this is also found in the case history of our patient.

It should be pointed out that, in particular, the typical location and prominence of the bone lesions are pathognomonic in nature. The distinguishing characteristic of acroosteolysis ([11], see also discussion of nomenclature and nosology of disease groups) may be delimited by x-ray examination from the familiar acreosteolysis, the idiopathically unfamiliar acroosteolysis, and

familiar osteosclerosis with acroosteolysis [9, 16] and should — after precise analysis of the clinical symptoms and the course of the disease taken together with job history — in the future be classified as occupational poisoning.

TABLE 1. SPECTRUM OF SUBJECTIVE SYMPTOMS
(Almost exclusively in the arm and leg regions, and very little in the shoulder girdle)

Sensation of cold
Burning, tingling and feelings of numbness, weakness and tension
Arthralgia, pain caused by pressure, various pronounced sporadic painful sensations
Also:
Discomfort of the upper abdomen, hissing in the ears, dizziness

TABLE 2. SPECTRUM OF FUNCTIONAL CHANGES
(Almost exclusively in the upper extremities, not always bilateral)

Paling of one or more fingers (dead finger phenomenon)
After cold stimuli, when clenching the fist, or in particular positions of the arm;
Raynaud's syndrome
or Raynaud-like syndrome
Spontaneously or after cold stimuli
Movement impeded
Spasms of the hand arteries and/or brachial arteries
Also:
Visual impairment, liver function disturbances

TABLE 3. SPECTRUM OF MACROMORPHOLOGICAL SKIN CHANGES
(Almost exclusively in upper extremities)

Moderate bluish, pale-whitish, ivory-colored, dirty yellowish, and bright colors.
Nodular, knotty, plaque-like, undulated, more or less raised and xanthoma--like infiltrations.
Diffuse skin patches, pileation retained
Shortened and watch-glass-like nails
Swelling of hands or fingers and/or forearm
Extension of changes to forearm or upper arm region
Drumstick fingers
Impairment of skin's ability to slide over the supporting tissues
Tight edematous waxy sclerotic consistency of integument
Also:
Bloated mask-like facial features
Flattish indurations in the cheek region

In studying the contamination of the air in the vicinity of shaft-type driers, when drying polychlorovinyl resin, vinyl chloride was not found in 82.3% of the samples. In the remaining samples its content varied from 0.7 to 0.12 mg/l, while the air samples in which vinyl chloride was found were collected in the vicinity of the fresh resin, in areas not equipped with local exhaust fans.

In areas where driers using the V. I. Stroganov system are in operation, there is a significant contamination of the air by dust from the finished product. The dust escapes when the examination doors of the furnace are opened (in case the technological process is disturbed), during the crushing and sifting of the resin. The dust concentrations of polycholorovinyl resin at the oven varied from 20 to 567 mg/m^3, while the crushing and sifting processes as well as the bagging, produced from 25.5 to 62.5 mg/m^3.

In some cases, the vicinity of shaft-type driers was found to have a high air temperature (23-37.5° with an outside temperature of 11.5-23.5°).

In order to improve the sanitary working conditions for those engaged in the operation of shaft-type driers, it is necessary to equip all areas with raw materials to be dried with a local mechanical exhuast fan. It must also be provided in places where the finished product is likely to produce dust that could enter the air.

Drum-type driers are used for drying coarse, fine, granular and firable materials; brown and anthracite coal, sand, clay, phosphorites, grain, granulated sugar, sawdust and the like.

In the operation which we examined, the drum-type drier was used for drying crystalline salts of the sulfates of ammonium and sodium.

A drum-type drier (Figure 5) consists of a rotating drum, 2 m in diameter and 10 m long, connected at one end with a furnace and at the other end with a loading bunker for the dry product as well as a gas pipe for trapping the furnace gases which have been exhausted. There are metal shelves inside the drum. The drum is rotated by an electric motor, acting through a gearbox.

The ammonium and sodium sulfate salts to be dried are fed into the drier by a screw conveyor. Drying is carried out using furnace gases at temperatures up to 220°. The dried salt is transported by a screw conveyor and an elevator to the bunker for storing the finished product.

The operation of the apparatus consists of monitoring the instruments and adjusting the salt drying process.

In the course of drying salts in a drum-type drier, the air is not contaminated by toxic substances because a vacuum is produced in the drier.

Meteorological conditions in production areas near driers are unfavorable. In winter the air temperature was 18-32° and in summer from 21-35°. The highest temperatures were recorded at the window of a drum-type furnace through which the operator checks the feed of salts into the drier. An air shower should be provided at this point.

TABLE 4. SPECTRUM OF MICROMORPHOLOGICAL SKIN CHANGES

Flattening of rete function
Interstitial edema, deposition of metachromatic, fine-granular, and filamentary materials
Plate-like fibrosis of the corium near the epidermis, thickening of collagen fibers and edematous relaxation of the collagen fiber bundle
Fragmentation or circumscribed clumping of the tunic elastica
Thickening of dermal arteries, hypertrophy of the tunica media, fibrosis of the intima, endarteriitis or endarteriolitis
Dilation of lymph capillaries
Inflammatory and predominantly lymphocytic pervascular proliferation with few histiocytes, involvement of mast cells
Degeneration of finest nerve fibers

TABLE 5. SPECTRUM OF X-RAY CHANGES
(Almost exclusively phalanges of the hand — often excluding ringed fingers)

Cortical changes
Circumscribed rarification
Crescent-shaped defects
Periarticular erosion of interphalangeal joints
Striated translucence to the extent of osteolysis of the pseudo-fracture type
Central osteolysis
Shortening and/or swelling of the ends of the phalanges
Acroosteolysis
Circumscribed (reparative) structural thickening
Also:
Osteolysis of ulna, radius, clavicle, patella
Widening of iliosacral foramina

Our data and deductions on the etiology of the syndrome are extremely scanty. PVC was synthesized by Regnault in 1835 (see [12]), and large-scale manufacture began in 1927 [10] and the first publication reporting damage caused by handling PVC appeared in Romania in 1963 [13]. In 1957, the German literature still contained no mention to danger to the health in discussion of the syndrome [10]. Other patterns of damage are mentioned [2] especially the acutely toxic ones [4]. Weichardt [15] found in 1970 that, despite the fact that Germany produces no less PVC than other countries, no disease attributable to it has been found; in 1972 Juehe and Veltman [7] and Juehe and Lange [6] published their observations on the topic.

From the relevant publications [1, 3, 8, 10, 15, 16] it can be deduced that only long-term close skin contact with incomplete VC polymers or various medium-to-high molecular intermediates (resins) and/or their aggressive additives such as plasticizers, stabilizers, accelerators, inhibitors, etc., lead to chronic toxicity. The characteristic pattern of damage is thus far known only in workers in the PVC manufacturing industry who have to scrape off the residue, whose exact composition is unknown, from autoclave walls.

(polycleaner [16]). On the other hand, diseases of the kind described here never appeared in those working with completely depolymerized, inert, and physiologically harmless plastics [16].

It is found from the literature that the pattern of damage is to be interpreted as the result of factors which may be chemical, mechanical (continuous scratching and scraping with polycleaners) or individual (only 3.0 to 4.5% of people doing the same kind of work are involved [1, 16]).

On the basis of the job history of our patient, who for years had to spread liquid PVC in his work as a telephone installer, no decisive pathogenic roles can obviously be ascribed to the "mechanical" factor described above.

For the "individual" factor it should be noted that it is probably to be seen as individual contact of various intensities with the responsible agent. The degree of strictness with which the individual observes or supervises the work instructions can also explain the varying frequency with which the syndrome is to be observed, and the differing periods (months to years) before the symptoms became patent. The job history of our patient instructively supports this interpretation of the "individual" factor: he was injured earlier and more extensively because of his special job causing his extremities to be coated with the film-like "special" polymer.

The mechanism by which the "chemical" industrial toxicological factor comes into play can in the first instance only be sketched: the subjective symptoms and functional changes — supported by macromorphological and micromorphological findings — point to a (initial?) neurovascular disturbance.

More details will be given on the etiology and pathogenesis of the syndrome in a later publication [13].

For prognosis and therapy of the disease after discontinuance of harmful contact while on the job, we may say that the skin changes recover spontaneously [6, 8] and the latter can be promoted whenever possible by physical therapy, as observed in our patient. However the bone lesions can progress even after work is suspended, finally terminating and recovering.

Summary

Case histories of chronic intoxication caused by intensive, long-lasting skin contact with incomplete vinyl chloride polymerization products and resins and/or their additives are presented. Paresthesia, functional, and macromorphological and micromorphological changes build up to a variegated pattern of symptoms. A syndrome of scleroderma-like dermatosis, Raynaud's and dead finger syndromes and pathognomonic bone defects are characteristic. When these symptoms are taken together with the occupational circumstances, this easily misinterpretable disease can be diagnosed.

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