Pulmonary Manifestations of Vinyl and Polyvinyl Chloride

To the Editor:

I would like to comment on the paper of E. M. Cordasco, et al, entitled "Pulmonary Manifestations of Vinyl and Polyvinyl Chloride (interstitial lung disease): Newer Aspects," (Chest 1981; 78:898-34) and caution against any premature conclusions reached from this paper.

While their article supplies interesting information, it does not substantiate their main contention that the three cases represent what they call vinyl chloride/polyvinyl chloride lung disease. Instead, each case is completely different and not really comparable.

The first case represents an alleged pulmonary disease from the inhalation of pyrolysis products of PVC meatwrapping film. The second case is an apparent exposure to vinyl chloride fumes; the third case represents one where the exposure is polyvinyl chloride resin dust. Each of these materials has different properties and characteristics and should not be considered a specific entity. Furthermore, the environmental information concerning exposure in each case is extremely scanty and limited. Therefore, having three individual cases of completely different exposures, and having little information on the specifics of the exposures does not constitute a designation of a disease entity. This fact should be made clear.

The first patient described was a meat wrapper exposed to PVC film thermal decomposition products as well as emissions from the adhesive heat label. The exact details of the latter exposure were not given. Blood tests for phthalic anhydride were said to be elevated. Phthalic anhydride was said to be a decomposition product of PVC. This, unfortunately, is not the case. The thermal decomposition of plain PVC has been studied extensively and emission products have been identified.1-6 PVC alone is different from PVC meat packaging film, which also contains a number of selected additives including plasticizers (di-2-ethylhexyl adipate) which can comprise up to 30 percent of film. Major emissions from hot-wire cutting of PVC meat packaging film are hydrogen chloride and plasticizer breakdown products including di-2-ethylhexyl adipate. PVC is not part of the emissions, nor is VC. Since phthalic anhydride and phthalates are generally not part of the PVC meat wrapping film, the elevated blood phthalic anhydride level is not due to decomposition of PVC meat wrapping film. Materials such as DEHP, MEHP, phthalic acid and phthalic anhydride measured in tissue or blood cannot be presumed to be the result of exposure to pyrolysis products of plasticized PVC, nor from the metabolism of phthalate plasticizers. Phthalic plasticizers and phthalic anhydride are present in a number of commercial products. Unless one has information on what are normal blood and tissue levels, then a measurement in the blood cannot be given any special significance. The information concerning normal blood levels of the phthalate compounds is not given in the manuscript. Phthalic anhydride is a component of many plastic materials and also a constituent of the thermal-activated price label "hot-melt" adhesives.1 Thermal activation of the price labels is performed by heating the labels from the printed side with possible emissions being phthalic anhydride. Our group has studied populations exposed to this chemical and have not identified cases of interstitial lung disease, but have noted cases of bronchial asthma.8 Furthermore, a number of recent studies of meat wrappers have not demonstrated significant lung disease.9,10 I seriously doubt, therefore, that the interstitial lung disease described only in one case was due to exposure to PVC meat wrapping film emissions.

The second case of the individual exposed to VC spray paint is perplexing. There is discussion on contents of a spray paint, indicating that vinyl chloride is one of the main components and not just a propellant. My information indicates that vinyl chloride is not used in this type of paint and therefore I question the accuracy of the information provided by Dr. Cordasco. Unfortunately, few environmental data are given and there are no details on other environmental exposures. VC is a potent carcinogen and for the past several years has been excluded as a propellant for commercial products.11

The third case is also confusing. This case involves a 55-year-old man exposed to PVC dust during a reactor cleaning operation. Apparently, the exposure was transient, over a 5-6 week period. The only information we have concerning environmental air concentrations is the statement that PVC in the immediate operational area varied between 200-300 parts per million. This level is not possible. Dust measurements are made in particles per cubic feet of air or weight (le mg or μg) per cubic meter of air. Cases are measured in parts per million. Prolonged exposure to PVC has been reported to cause a pneumoconiosis, as indicated by Dr. Lilis in her commentary. Prolonged exposure is necessary and a 5-6 week exposure is unlikely to result in significant lung disease.

In summary, therefore, I would again caution against reaching any definite conclusions from the findings in this paper. The environmental data are scanty and inconsistent and in many cases erroneous. Actual levels are lacking and all environmental contaminants have not been adequately identified. The authors have, unfortunately, fallen into the common trap befelling many practicing physicians of attributing an occupational disease to be present because a potentially toxic material is present in the work place, and without properly and accurately determining what the actual exposures are, in what concentrations, the duration of exposure, as well as properly excluding other agents and non-occupational causes for the disease.

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To the Editor:

We have a follow-up report from Dr. Kerkay, Professor of Biochemistry at Cleveland State University, who has been working with his group in conjunction with our clinical studies on polyvinyl chloride. He was co-author of the paper recently published in Chest. His comments follow:

“Dr. Stuart M. Brooks’ criticism of the first patient’s results and biochemical data is out of context. When he refers to published data (his references 2-6) of PVC breakdown products, he cites the thermal decomposition of bulk PVC rather than the plasticized final product. His reference 6 confirms our findings qualitatively (p 383 and 390) where they report the volatilization of phthalate plasticizers during thermal decomposition of certain (plastic E & G) PVC compounds. In his reference 1, Dr. Brooks cites an article by Vandevert and co-workers, which was published in 1977 and deals with the thermal decomposition of meat packaging films. There have been changes and improvements in the plasticization of meat wrapping films during the past 4 years. Our laboratory does have GC and HPLC tracings of the patient’s blood and urine indicating the presence of phthalate and not adipate plasticizers, as confirmed by mass spectroscopy. It is true that many commercial products contain phthalate plasticizers; however, these plasticizers are leached out of plastics only if the plastic comes in direct contact with blood (or lipid containing solutions) or when inhaled following volatilization. We have tested blood and urine specimens from over 100 normal individuals and not one of them showed the presence of phthalate or adipate plasticizers in greater amounts than the detection limit of our method of 40 ng/ml serum or urine. Therefore, we assume that the normal phthalate level is less than 40 ng/ml, which is undetectable.

“Our findings of DEHP, MEHP, phthalic acid, and phthalic anhydride in this patient resulted from the exposure to thermal degredation products of the plasticized PVC and from the metabolism of these phthalate plasticizers.

“The cytologic examination of lung tissues from the three cases presented in our paper are very similar to the changes observed in lung tissues of dogs following intravenous injection of chemically pure bis (2-ethylhexyl) phthalate (DEHP).”

Now in conjunction with the special vinyl chloride paint composition that one patient had been exposed to, this has been very difficult to follow-up because the dental equipment company, known as Weber of Canton, Ohio, has totally closed down, and I am in the process and have almost completed the process of finding additional information through the major dental equipment company in New Jersey. They have referred me to the dental school here and also through Dr. Carl Zenz of Milwaukee, Wisconsin. A special paint which is identical to the chemical B, is now being analyzed for total and specific contents.

The third case delineated the PVC occupational atmospheric level at 200-300 parts per million, which is what we had obtained from the industrial safety director of a specific plant. Our chemist co-author discussed this in detail with the Research Chemist Supervisor of B.F. Goodrich Company of Akron, Ohio (new plasticizer division), who stated that this pollutant can be reported in micrograms per cubic meter or in parts per million. We are fully aware of the fact that as a particulate, this is appropriately reported in micrograms or milligrams per cubic feet of air. Therefore, we were in error as the occupational atmospheric reading should have been reported as micrograms per cubic foot.

We have seen many patients, in consultation with two chemical plants in New York State, with vinyl chloride and polyvinyl chloride exposures. We believe that the patient reported represented a case of subacute interstitial pneumonitis and not de-facto pneuconiosis.

If one reads the article carefully, on page 833, paragraph 2, we alluded to the fact that the relationship is highly presumptive and not conclusive as to cause-effect relationship in the development of interstitial fibrosing pneumonitis. We cautioned in our conclusions that the total clinical significance of the factors referable to the biochemical changes were still unknown and will be pursued further.

Consequently, we have felt that the published paper has left some unknown answers and in no way have implicated these agents to be direct causative factors of pneumoniconis in any of these three patients. We do feel that these histopathologic changes are probably related to the excessive exposures of the VC/PVC group of agents. Therefore, we disagree with Dr. Brooks that the authors have “fallen into the common trap of attributing occupational disease to be present because of potential toxic material in the work place.”

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