COMMUNICATIONS

REFERENCES


Exposure to Hard Metal

To the Editor:

Re the editorial in Chest 1984; 86:513-14, I agree there is much work to be done to delineate which people are sensitive, what the precise immunologic and cytotoxic mechanisms are, and the dose response data, I must differ strongly with the opinion that the causal agent has not been clearly identified.

The work of Schepers in 1955 presented a beautiful photomicrograph documenting the same very unusual "can nibalistic" giant cells seen in humans with so-called giant cell interstitial pneumonia. On reviewing a series of the rare cases of this pathologic entity a few years ago, I noted that nearly all (33 of 16) cases had either historic evidence of exposure to the tungsten carbide industry and/or documentation of this exposure by tissue microanalysis. We also were able to study bronchoalveolar lavage as a method for documentation of exposure. While most of the cases did not show detectable levels of cobalt in the biopsies, the exposure was there, and coupled with the animal data producing the same pathology (which is one of the few newly uniquely pathognomonic reactions) and provides good evidence that there is a distinct pathologic disease produced by cobalt.

Jerrold L. Abraham, M.D.,
Associate Professor and Director,
Environmental and Occupational Pathology,
State University of New York
Upstate Medical Center, Syracuse

To the Editor:

In my recent editorial, I offered the scientifically conservative view that the cause of the parenchymal lung disorders which have been associated with exposure to hard metal (tungsten carbide with binders including cobalt) remains uncertain. Dr. Abraham, citing some of his own work and the classic inhalation studies at Saranac Lake,1 argues that the pathogenic role of cobalt has been established. His basis for this is the similarity between histologically distinct giant cell reactions seen in some reported cases of hard metal disease and the pathologic response to cobalt metal dust reported by Schepers.1

While I agree that sufficient similarities have been demonstrated to sustain cobalt as the most likely cause of parenchymal lung disease associated with this complex dust exposure, I continue to maintain that caution remains important in further exploration of this problem for several reasons. First, as I cite in the editorial, some, but by no means all cases of so-called hard metal disease share the pathologic features of giant cell pneumonitis which he dubs pathognomonic for this condition. Furthermore, the spectrum of animal responses to cobalt and other constituents of hard metal have been very far from uniform. Neither Kerfoot et al2 in a miniature swine model, nor Kitamura et al3 in a mouse model produced lesions of a similar nature. Even the experiments of Schepers must be examined critically. Only with low repeated doses of cobalt were giant-cell reactions observed and these were apparently associated with only mild inflammatory reaction surrounding. Giant cells were also induced with minimal tissue reaction in experiments with tungsten carbide (without cobalt) alone. Perhaps most interesting, the responses to the combined inhalation of tungsten carbide and cobalt together produced a further reaction, with evidence of involvement of the lymphatics, seen in response to neither of the metals alone.4

None of this analysis, of course, exonerates the role of cobalt nor should it be inferred that cobalt remains anything other than the most worrisome suspect in the search. Rather, my point was and remains that as the domain of the investigation in this disorder moves from morphologic to immunologic analysis, the potential roles of "inert" constituents should not be entirely overlooked. This is especially important in view of the large spectrum of reported human responses and the likely role of idiosyncrasy in at least some of them suggested by epidemiologic patterns of disease manifestation. Furthermore, from a public health perspective, we must be very cautious about transferring our suspicion of a "single" cause of hard metal disease to settings in which exposure to the constituents may occur separately.

Mark R. Cullen, M.D.,
Assistant Professor of Medicine and Epidemiology; Director,
Yale-New Haven Occupational Medicine Program, New Haven

REFERENCES

1. Schepers GWH. The biologic action of particulate cobalt metal. AMA Arch Ind Health 1955; 12:127-33
5. Schepers GWH. The biologic action of tungsten carbide and cobalt. AMA Arch Ind Health 1955; 12:140-46

Fluid vs Liquid

To the Editor:

Regarding the excellent article by Wiedemann et al5 which included remarks on the hazards of balloon-tipped pulmonary artery catheters, the authors twice warn against the use of "fluids" to inflate the balloon. I feel confident that they meant to say "liquids," as the definition of fluid is and always has been "a liquid or a gas." That is, all liquids are fluids, but not all fluids are liquids. Treating "liquid" and "fluid" as synonyms unnecessarily wastes a useful distinction.

Robin M. Lake, M.D., F.C.C.P.
Monroe, LA

1. Wiedemann HP, Matthey MA, Matthey RA. Cardiovascular-pulmonary monitoring in the ICU, (3). Chest 1984; 85:666-68