solution). The ethanol-induced effect is probably mediated through release of histamine, followed by H1-receptor activation.43 Nifedipine provided the most evident and significant protective action.6

We hypothesize that, in the near future, this class of drugs will be used as a minor bronchodilator drug in the treatment of airway diseases, as well as in therapy for cardiopathic subjects with respiratory problems.

Furthermore, it's desirable to have new dihidropiridinide derivatives (we are now testing nitrendipine) with more relaxant action on bronchial smooth muscle and major selectivity on lung tissue, using nifedipine equimolecular doses.

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REFERENCES


To the Editor:

We appreciate the interest Spedini and Lombardi have shown in our article. Their preliminary findings are encouraging, and seem consistent with the hypothesis that calcium antagonists may have some role in the therapy of obstructive airway disease, especially in patients with associated angina and atherosclerotic heart disease. Certainly, information with newer calcium antagonists will be of much help.

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Cobalt and Hard Metal

To the Editor:

In the April, 1985 issue of Chest, I read with interest the discussion between Drs. Abraham and Cullen with reference to the editorial in Chest 1984; 86:513-14. It is not my intention to argue whether cobalt is the single pathogenetic factor in hard-metal disease, but I would like to draw attention to an exposure hazard to cobalt not allowed to tungsten carbide, causing a similar pathology as in hard-metal disease. In recent years, grinding tools with a cutting edge of microdiamonds cemented in a binding substance, over 99 percent of which consists of extra fine cobalt without tungsten carbide, have been marketed for diamond polishing, at least in Belgium.

We have observed that some diamond polishers, exposed to the powder abraded from these disks, developed fibrosing alveolitis, and we found multiple giant cells and sometimes cobalt in open lung biopsy tissue as well as bronchoalveolar lavage fluid (Demedts et al, Am Rev Respir Dis 1984; 130:130-35). In the lavage fluid, no cobalt was detected some weeks after interruption of the exposure, but giant cells, or at least "loaded" multinuclear macrophages, could still be found even several years later in affected patients (in one, even after lung function and chest x-ray film normalized again).

Other exposed diamond polishers developed occupational asthma and presented a positive inhalation challenge test to cobalt, with a temporarily increased response to histamine (Cheyssens et al, Chest, accepted for publication). In the bronchoalveolar lavage fluid of exposed workers with occupational asthma, or in those without disease, no giant cells or cobalt was detected.

Finally, I want to draw attention to the fact that these diamond-cobalt disks are now used also for other occupational duties (e.g., building construction and tile-pavement) and that, in affected workers, the exposure hazard may be overlooked, especially because the manufacturers' misleading campaign stress that these tools contain no hard-metals.

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To the Editor:

Dr. Demedts makes an important point which I had included in my previous letter, but which had been edited out due to space limitations. I specifically pointed out that the recent work by Dr. Demedts and his colleagues showed the distinctive pathologic reaction and clinical picture of interstitial pneumonitis with peculiar giant cells (giant cell interstitial pneumonia) in workers exposed only to cobalt (not to other hard metals). The occupational asthma related to cobalt exposure in the diamond polishers is further support of the role of cobalt in so-called "hard metal" disease. Dr. Demedts and his colleagues have, in fact, kindly allowed me to review and analyze tissue from three of the patients they reported in 1984. In contrast to the results of my electron probe analyses of lung tissues from patients with exposure in the tungsten carbide industry, the cases from Dr. Demedts group of diamond polishers showed very little inorganic particulate dust in the lungs. Cobalt was detected in one of the biopsies, but not in the others (cases one and four). Similarly, cobalt was only rarely detected in the lung tissues from patients with exposure in the tungsten carbide industry.

The use of cobalt in potentially respirable form in numerous other industries is an important point also. A recent document from the United States Department of the Interior (Kirk, William S. Cobalt. In: Mineral facts and problems, 1985, Superintendent of Documents, Washington, D.C.) points out that the United States is the largest consumer of cobalt, accounting for about one-third of the total world consumption (U.S. consumption was 15.7 million pounds in 1983). Major uses of cobalt include transportation, electrical parts, machinery, paints, chemicals, and others.

The major point of all this continuing correspondence is that the type of uncertainty presented in Dr. Cullen's editorial may lead to unnecessary delay in cleaning up the work place to prevent the development of disease in the future. Appropriate engineering controls have been installed in most European countries once the problems have been recognized, and subsequent development of disease has either been eliminated or greatly reduced (personal communications from Dr. Demedts and from Dr. G. Hillerdal, who reported a similar problem in 1980 [Thorax 1980; 35:633-59]).

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