communications to the editor

Communications for this section will be published as space and priorities permit. The comments should not exceed 350 words in length, with a maximum of five references; one figure or table can be printed. Exceptions may occur under particular circumstances. Contributions may include comments on articles published in this periodical, or they may be reports of unique educational character. Specific permission to publish should be cited in a covering letter or appended as a postscript.

UFFI Dust

Nonspecific Irritant Only?

To the Editor:

Because of the controversy about the possible health hazard of urea formaldehyde foam insulation (UFFI), I think it important to reassess the strength of the evidence suggesting that disease might be related to this material. The paper by Frigas, Filley and Reed, "Asthma induced by dust from urea-formaldehyde foam insulating material," (*Chest* 1981; 79:706-07) is a case in point. This paper has several serious deficiencies which in my opinion make it questionable whether one can draw the conclusions stated by the authors, for the following reasons:

1. The study could certainly have been carried out single-blind and this should have been done, particularly in a situation such as this where on the patient's part it is likely that deterioration of asthma will be attributed to the UFFI because possible hazards from this insulating material have been much publicized in newspapers and on television. A double-blind study would have been preferable and certainly far from impossible.

2. The authors should have measured nonspecific reactivity and exposed asthmatic not living in UFFI homes but shown to have a similar level of nonspecific reactivity, to the foam dust to be sure that the response was not a nonspecific one resulting from dust.

3. The use of aluminum oxide dust as a nonspecific dust stimulus may be invalidated by the fact that this material has a density approximately four times that of water and many times that of UFFI. This would result in inhalation into the lower respiratory tract of very much less of the aluminum oxide dust depending on the particle size to which the dust was milled. It is unlikely that the two exposures were comparable.

4. At the time of these studies, this patient was doubtless very hyperreactive, because she required systemic steroids in addition to her usual bronchodilators. It is well known that such hyperreactivity may vary (for example a viral infection within weeks or months might increase hyperreactivity which may subsequently gradually decrease) so that the fact that she got better after she left her home and required somewhat less therapy, while at the same time the life-threatening episodes ceased, may merely have been a coincidence. As a minimum, she should have been challenged with the UFFI dust again after being out of her home for several weeks or months to confirm that she was indeed sensitive to the material rather than responding in a nonspecific way.

5. The fact that it took three successive challenges over a 30-minute period to evoke any response, followed by an unusual pattern of response—slowly progressive over one hour, is unusual and should certainly have been confirmed with a second test.

6. For the study to have been convincing, an occupational work exposure type test should have been performed in a double-blind fashion using as a control a similar dust not containing UFFI and in a research setting where the nonspecific airway hyperreactivity had been well established previously. Only by demonstrating that the UFFI specifically caused bronchoconstriction in this patient while another asthmatic with a very similar level of nonspecific hyperreactivity did not respond, could be considered convincing evidence that UFFI indeed caused her asthma.

As it stands, this paper is not convincing and does not constitute evidence that UFFI dust is asthmagenic except in a nonspecific way.

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To the Editor:

We share your implied concern that recent publicity and governmental actions banning urea-formaldehyde foam insulation may have unjustly harmed the very homeowners the regulations were designed to protect. At the least, the risk of formaldehyde has been exaggerated. As we stated in our case report, formaldehyde itself did not provoke asthma in our patient. We have since rechallenged this patient and challenged 37 additional subjects who had chest symptoms on exposure to formaldehyde for 20 minutes with formaldehyde gas delivered with a face mask by a Dynacalibrator through a Teflon tubing at concentrations up to and including 3.5 ppm. No one had a greater drop in FEV_1 after exposure to formaldehyde than after exposure to room air. Because of the characteristic odor of formaldehyde, these challenges were not truly blinded, but in the absence of a response, blinding becomes unnecessary to identify the cause of the response. Thus, we conclude that if formaldehyde does provoke asthma, it does so only rarely.

Concerning the patient in our report, we should describe several additional controls that were not included in the publication.

We have not repeated the challenge with the UF foam dust. After the patient moved to a different house, her condition improved. However, we did a challenge with formaldehyde gas approximately one year later using concentrations up to 3.5 ppm. As on the first occasion, the patient did not react. On the other hand, whenever she revisited her original residence, the asthma worsened, even after a stay of only a few hours.

We did not determine if the particle size of the UF foam dust and the aluminum oxide dust were comparable. The aluminum oxide dust was obtained from the work site by a patient with asthma associated with grinding capstans manufactured for tape recorders. This second patient had a 30 percent drop in FEV_1 on three occasions in the laboratory after exposure to this aluminum oxide dust and did not react to the UF foam dust.

Other controls were three subjects with asthma living in homes insulated with UF foam, and one asthmatic patient and two normal subjects not exposed to UF foam. None reacted to the challenge with the UF foam dust in question.

Undoubtedly, a double-blind study would have been preferable, but was impossible because of the characteristic appearance of the dusts involved.

In summary, we believe that the UF dust was responsible for the positive bronchial challenge in our patient, but we do not know the mechanism of the reaction.

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Tetracycline Pleurodesis for Refractory Pneumothorax among Inoperable Elderly Surgical Candidates

To the Editor:

The recent articles by Macoviak et al[1] and Goldszer et al[2] prompted a review of my own cases of recalcitrant or recurrent pneumothorax that underwent intrapleural tetracycline instillation. The review encompasses four men who, for preexisting severe cardiopulmonary and other debilitating conditions, were not surgical candidates. These individuals were initially managed by intercostal drainage for seven to ten days. They were addressed to the potential benefit derived with tetracycline (TCN) when used for malignant pleural effusions, and the use of the Heimlich valve attached to the shortened chest tube. All patients chose tetracycline even though it was clearly explained that: 1) its use is occasionally associated with severe, transient pleuritic pain, and mild temperature elevation, and 2) its success or failure rate remains untested by any known human studies.

The patients defined as inoperable were exemplified by: 1) a 78-year-old man with bilateral bullous emphysema, congestive heart failure, gastric bleeding ulcer, postoperative pneumonia, cystitis, fungemia, and septicemia; 2) a 73-year-old emphysematous man with an undifferentiated carcinoma of the left mainstem bronchus (almost completely obstructed), and a left pleural effusion (600 ml); 3) a 69-year-old man with severe emphysema and generalized pulmonary congestion; and 4) a 72-year-old man with a right upper lobe mass confluent with the right mediastinum and midtrachea, acute tracheal obstruction (5 mm lumen), bilateral wheezings, right vocal cord paralysis, and past history of inferior myocardial infarct, coronary bypass and abdominal aortic aneurysmectomy. All presented between June, 1981 and March, 1982 with symptomatic and refractory pneumothorax (>40 percent) unresponsive to intercostal drainage of seven to ten days' duration. All except patient 4 had resolution of their pneumothoraces after intrapleural tetracycline; one patient needed repeated instillation with one day interval for complete control. With experience obtained among patients with malignant pleural effusions, powdered tetracycline (500 mg) is dissolved in normal saline solution (30 ml) and 1 percent lidocaine (5 ml); the mixed solution is instilled in the chest tube, and allowed to gravitate to the chest cavity. Concomitantly, the patient is sedated with intravenous nubain or morphine (2-5 mg) for better tolerance of the procedure and the positional changes needed for the subsequent two-four hours. Within 24 hours of administration, the patients (except one) displayed no air leaks of pneumothoraces, and the chest tubes were removed.

Tetracycline pleurodesis for active, recurrent pneumothorax clearly falls in the armamentarium of therapeutic measures available, and affords minimal side effects and risks when compared with other sclerosing agents (talc,[3] quinacrine,[4,5] kaolin[6]), surgical pleurodesis, or the Heimlich valve. Moreover, because of its success among inoperable elderly candidates, the intrapleural use of tetracycline should be projected among the youth appearing with recurrent spontaneous pneumothorax. Failing such a course, the surgeon may then opt for surgical abrasion or pleurectomy.

What Dr. Macoviak and his group have demonstrated experimentally has been clinically produced in the human arena among a selected group of patients known to be inoperable. Yet, I must still caution that in patients with massive ruptured bleb(s) or bulla(e), single or multiple tetracycline instillations may not effectively control such active air leak. One should resort to continuous intrapleural drainage or to a Heimlich valve as in patient 4.

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