have taken them up by absorption through the skin. Furthermore, the compounds were deliberately placed near to the surface of the paper to make the process more efficient, accentuating a potential hazard.

The diazo process as described was in fairly widespread use up to the mid-1970s (I understand that there are photographic applications today, but although there seems little danger of skin contact in them it would be as well to reconsider methods of waste disposal). As bladder cancers can take up to 20 years to develop many would have turned up after some delay, and indeed, it this explanation is correct, some have yet to surface. I make no claim of proof on the basis of this one case, but I suggest that current patients with bladder cancer should be surveyed for possible correlations, and also that an analysis be performed on some exposed papers for the suspected chemicals.

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Objective testing for vasospasm in the hand-arm vibration syndrome

Sir,—Regarding the article by Allen, Doherty, and McGrann (1992;49:688–93), may I make the following comments: (1) We are unaware of any objective test that can distinguish hand-arm vibration syndrome of occupational origin from primary Raynaud’s syndrome or any other secondary cause. Such a test is eagerly awaited not only for medicolegal reasons but in the management of true hand-arm vibration syndrome/vibration white finger. (2) Tests for vascular dysfunction do just that and do not give any information that the cause of dysfunction is related to vibration. (3) The authors describe the use of an investigative technique that showed a good correlation between a clinical white finger and a valid demonstration of digital vasospasm. But to describe this as objective testing for vasospasm in the hand-arm vibration syndrome is a non-sequitur that does not assist in the search to diagnose hand-arm vibration syndrome/vibration white finger of occupational origin on sound medical grounds.

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The Authors’ reply

The investigative technique described was developed to detect the occurrence of digital vasospasm and grade its severity in an objective manner. This is helpful in the assessment of vasospastic disease whether it is primary or secondary in origin as diagnoses need no longer be based solely on the history given by the patient. Signs of cold induced vasospasm are often absent at the time of medical examination and so objective testing can help to clarify the situation.

The test procedure was first evaluated in patients with non-occupational vasospasm and it has never been suggested that it can differentiate between the various forms of Raynaud’s syndrome, which are distinguished on clinical grounds and not investigative findings. In subjects diagnosed to have vibration white finger on clinical assessment, an objective test detecting vasospasm supports the clinical diagnosis and helps grade its severity. Such testing does therefore assist diagnosis of vibration white finger.


Pulmonary effects of exposure to fine fibreglass: irregular opacities and small airways obstruction

Sir,—I read with interest the article by Kilburn et al (1992;49:714–20). I first became aware of the results of Kilburn’s investigation in 1989, when he reported his findings at the Second California thermal insulation conference. As a follow up to his report, Owens-Corning contacted General Electric, the operator of the plant. General Electric provided me with a copy of the industrial hygiene data that it had provided to Kilburn. From these data it seems that he has incorrectly reported the concentrations of fibreglass and asbestos that were present. For concentrations of fibreglass present in the plant, Kilburn states that: “when examined by light microscopy, they showed less than 0.1–0.4 fibres/ml with total dust concentrations of 0.13–0.47 mg/m³ measured gravimetrically.”

In fact, the highest concentration of glass fibres reported in the 29 samples that were provided to him was 0.04 fibres/cc. Five of the data points were below detectable limits, which was pointed out to Kilburn. Fourteen of the measurements were 0.01 fibres/cc. The remaining data points were either 0.02 or 0.03 fibres/cc. In short, the data provided to Kilburn were at least a full order of magnitude below that reported in his paper.

Kilburn reported the historical data, from 1975, that was provided. Those measurements were less than 0.1 mg/m³ of respirable dust. He failed to mention, however, that at that concentration they were well beyond the limit of detection of the sampling technique. He did not report at all the 1979 data, which also indicated that the glass fibres were below the limit of detection.

As to asbestos fibre concentrations, Kilburn states that: “no asbestos fibres were reported in the samples by phase contrast microscopy.”

That is true for the 1989 data with which he was provided. The data from 1974 and 1975 given to him, however, showed that there were low amounts of asbestos present in the plant. There were 14 measurements, 10 of which were below detectable levels. Three of the other four measurements of asbestos (0.2, 0.10, 0.25 fibres/ml) were an order of magnitude higher than the concentrations of fine glass reported to Kilburn. Although they were all well below the permissible exposure limits in effect at that time, they would seem to be relevant to the analysis of the hypothesis proposed by Kilburn et al.

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