Correspondence

British Journal of Industrial Medicine 1987;**44**:287–288

advocated by Aldridge and Connors⁶ and Lauwerys and Louis-Ferdinand.⁷

B NEMERY

*Industrial Toxicology and Occupational Medicine,
University of Louvain,
Clos Chapelle-aux-Champs 30.54,
1200 Bruxelles,
Belgium*

Bright red blood of Bhopal victims?

SIR,—In his letter (1984;**43**:502–4) Salmon elegantly discusses and refutes the theory of cyanide poisoning in Bhopal victims. Some limited animal work further denies that cyanide intoxication is involved in the intoxication with pure methyl isocyanate (MIC).¹ On the basis of the little information available, however, I am not even sure that the colour of the blood of the victims was really bright red. An early report about this issue is that of Ivan Fera “The day after” published in *The Illustrated Weekly of India* on 30 December 1984 and reprinted in a compilation of documents from Indian publications.² This article states: “By that afternoon (Dec 3rd 1984), the first autopsies had been performed. In many cases, the blood was purple red; the lungs ash coloured, and severely edematous” (p 49, my underlining). Only in the next paragraph is it said that “Heereshchandra, the head of the forensic department, felt that the evidence pointed strongly to cyanide. This was apparent in the bright red, almost pink colour of the blood and the unpleasant odour given off when the lungs were cut open—the oil of bitter almonds, the smell of cyanide” (p 50). Later sources, still quoting the same person, mention “cherry-red blood”³ or “dark, cherry-red blood,” with lungs and other organs also red.⁴

It may seem futile to argue over different shades of red as reported by journalists, but the contradictory nature of the information in itself does little to support the whole cyanide issue. If the first quotation (purple blood) had been emphasised then the problem which had serious therapeutic and even sociopolitical implications^{4,5} could have been at least partly resolved.

The origin of this MIC cyanide issue might well prove to have been heavily influenced by semantics and the confusion between cyanide and methyl isocyanate. This sort of confusion would be quite understandable, even among medical people, in the early hours of a disaster caused by a hitherto toxicologically unknown compound. Whatever the reasons for the cyanide theory, the MIC disaster underlines, besides many other important issues already discussed in the *Journal* (1985;**42**:477–8), the need for accurate documentation and rapid scientific publication of clinical and laboratory findings after chemical accidents. This could be facilitated by effective international cooperation and close collaboration with experimentalists, as

References

- 1 Nemery B, Dinsdale D, Sparrow S. Methyl isocyanate: thiosulfate does not protect. *Lancet* 1985;ii:1245–6.
- 2 “Bhopal: industrial genocide” Hong Kong: Arena Press, Asian Regional Exchange for New Alternatives, 1985.
- 3 Hamlyn M. Bhopal: was cyanide the culprit? *The Times* 1985 17 January.
- 4 Agarwal A. The cloud over Bhopal. *New Scientist* 1985; Nov 28: 38–41.
- 5 Anonymous. New Bhopal dispute: cyanide poisoning of victims claimed. *Chemical and Engineering News* 1985;**63**:6.
- 6 Aldridge WN, Connors TA. Chemical accidents and toxicology. *Hum Toxicol* 1985;**4**:477–9.
- 7 Lauwerys R, Louis-Ferdinand R. Human toxicology: the need for a closer collaboration between clinical and experimental toxicologists. *Hum Toxicol* 1984;**3**:61–2.

Chrysotile asbestos exposure and mesothelioma

SIR,—The recent paper by Gardner and colleagues (1986;**43**:726–32) highlights the continuing controversy regarding the carcinogenic potential of chrysotile asbestos. As the authors point out, although available data suggest that amphibole fibres are more potent for producing mesothelioma than chrysotile, this relation is not well defined for lung cancer.¹ The recent observation of a statistically significant excess mortality due to lung cancer among textile workers exposed exclusively to chrysotile is also of interest, suggesting that differences in airborne fibre characteristics may partially account for differences in mortality between chrysotile exposed occupational groups.²

This latter point may be important in that exposure to chrysotile fibres that have been physically “altered” due to industrial processing or through consumer use may prove hazardous. For example, to my knowledge no systematic investigation of brake repair workers, exposed solely to chrysotile, has been conducted, although some evidence exists which suggests that this occupational group may be at risk for developing mesothelioma.³

As part of an epidemiological study of diffuse pleural mesothelioma being conducted at the Canadian Tumor Reference Center, I have recently completed a preliminary review of 37 cases of this tumour associated with occupational asbestos exposure. (All cases were confirmed at necropsy.) Of these, three (all male) had job titles of “brake mechanic (auto),” “elevator