several authors. The new findings in our study were the relation between body mass index and antibody response, and the interactions between this index and injection site and sex. We do, as suggested by Yassi *et al*, recommend checking antibody concentrations in staff in contamination incidents; this is part of a comprehensive programme of prevention and management of such incidents in our Health Authority.

Yassi *et al* make a good point regarding the need for more information about the rate of fall of antibody concentrations over time. Particularly, what is required is data to allow prediction of duration of persistence of protective antibody concentrations in subjects after immunisation. The studies reporting mean antibody concentrations at intervals after immunisation do not help much in this regard. We are presently analysing data from our immunisation programme, which includes repeat antibody testing at intervals, with the aim of devising a method of predicting the rate of decline of antibody concentrations. Unfortunately, the data presented by Yassi *et al* are inadequate for this purpose; only 60 people were studied and antibody concentrations were measured at varying and unspecified intervals up to six years from primary immunisation. No attempt was made to relate the measured concentrations to the concentrations immediately post immunisation, which were apparently known only in some subjects. The lower proportion with "protective" antibody compared with that in our larger study of concentrations immediately post immunisation is not surprising.

It is a cause of concern to note the apparently low coverage among staff reporting contamination incidents, in the hepatitis B immunisation programme reported by Yassi *et al*. This is despite the fact that 10% of the reported incidents were from known HBsAg positive sources. Achieving a high coverage with hepatitis B immunisation is at least as important as considerations of timing of booster doses.

Role of manmade mineral fibres in the causation of cancer

Sir,—We agree with almost all of the recent editorial by Enterline (1990;**47**: 145–6). Indeed, the latest update of the American epidemiological studies, of which Enterline is a co-author, make the conclusion of little or no risk in the glass and rock wool production industries even stronger (G Marsh, personal communication). As Enterline points out, any excess of lung cancer is virtually limited to the slag wool production industry more than 20 years ago. Much of the excess mortality in this group occurs among very short term workers (J Hernan, personal communication). Furthermore, the known exposures to asbestos and to other co-exposures in this branch of the production industry are now recognised as being more likely causes of any excess mortality among longer term employees.

Enterline draws the following conclusions:

It is doubtful whether much meaning can be attached to the small excesses among glass wool workers.

There are no excesses in workers in rock wool plants.

Probably fibres do not play the major part in the excess of cancer among slag wool workers so that fibre potency should not be estimated from epidemiological data on workers who produce these fibres.

Given these conclusions, it is surprising to note his final sentence which can be read to imply that exposure to manmade mineral fibres, whether made from rock, slag, or glass, can cause lung cancer. We wonder if certain key words were omitted and suggest that the sentence should read: "There is undoubtedly some evidence of a small cancer hazard attached to the manufacturing process in slag wool plants 20 to 50 years ago, when asbestos was used in some products. Even if glass, rock, or slag wool pose a fibre hazard, it is much less than that of asbestos and perhaps some other kinds of manmade mineral fibres."

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Author's reply:

Rossiter and Douglas suggest that there may be an inconsistency in my editorial. Whereas it is true that in two large epidemiological studies 20 years from first exposure, respiratory cancer excesses in glass wool plants are small and are non-existent in rock wool plants, I think it is important that the question of whether manmade mineral fibres are capable of causing cancer in man is not dealt with solely on the basis of the existing epidemiological evidence. The usefulness of the data is severely limited because of the extremely low fibre exposures. Because of the likelihood that it is the physical properties of asbestos fibres that cause respiratory cancer, similarities in the physical properties of manmade mineral fibres and asbestos, evidence that manmade mineral fibres are capable of producing cancer in animals, and clear evidence that non-asbestos fibres are capable of producing cancer in man, it would be irresponsible to conclude that ordinary manmade mineral fibres, whether made from rock, slag, or glass, do not carry some risk of cancer.

Rossiter and Douglas also raise the issue as to whether the excess seen in the slag wool plants may have been due to the use of asbestos many years ago. Probably because of the litigation problem in the United States, this has proved to be a difficult area for investigation. Statements from industry have come very slowly and are sometimes contradictory. Recently, however, we have obtained direct evidence of the use of asbestos in one slag wool plant where asbestos was apparently used in a cement mixing operation. In a case-referent study with lung tissue collected from decedents in the United States study we found four of six workers with amosite asbestos in lung tissue and only one in six matched referents.[1] We were also able to obtain lung tissue for five other rock wool or slag wool workers but for only two matched referents. Of the five, three were from a plant in which we have been told asbestos was used and which was originally a rock wool plant. Of these three, two had amosite asbestos present. In only one of the other five specimens (a referent) was any type of asbestos found. In our latest report for the slag wool plant where four of six specimens contained amosite asbestos the respiratory cancer standardised mortality ratio (SMR) 20 years from first exposure was 164·3 (23 deaths) whereas for the rock wool plant where two of three specimens contained amosite asbestos the respiratory cancer SMR was 79·2 (13 deaths).[2] Asbestos may, of course, have contributed to

the excess in respiratory cancer in the slag wool plant but I do not believe it played the major part in the overall respiratory cancer excess in the slag wool plants in our study. In two slag wool plants for which we are fairly certain asbestos was never used the respiratory cancer SMR was 258·5 (13 deaths).

1 McDonald JC, Case BW, Enterline PE, *et al.* Lung dust analysis in the assessment of past exposure of man-made mineral fiber exposure. *Ann Occup Hyg* (in press).
2 Marsh GM, Enterline PE, Stone RA, *et al.* Mortality among a cohort of U.S. man-made mineral fiber workers: 1985 follow-up. *J Occup Med* (in press).

Evaluation of a system recording non-pneumoconiotic abnormalities as part of coal workers' *x* ray surveillance programme

Sir,—Section 203 of the Federal Coal Mine Health and Safety Act of 1969 mandated the establishment of a programme of radiographic examinations to enable early detection of pneumoconiosis in underground coal miners. The Federal Mine Safety and Health Act of 1977 continued the congressional mandate for these radiographic examinations. The coal workers' *x* ray surveillance programme (CWXSP) is administered by the National Institute for Occupational Safety and Health (NIOSH).[1] Examinations and initial interpretation (from an "A" or "B" reader) are arranged and paid for by the coal mine operator. The NIOSH approves each operator's plan for examination of miners, approves *x* ray facilities, and certifies physicians to interpret radiographs for the programme according to the International Labour Office (ILO) classification system.[2] For each film, NIOSH also obtains a second interpretation (from a "B" reader) before a final determination regarding the presence of pneumoconiosis is made.

As well as classifying pneumoconiosis, the readers are required to note other radiographic abnormalities using symbols specified in the ILO guidelines. The standardised form for recording radiograph interpretations for the NIOSH programme includes an obligatory "other symbols" section comprised of labelled boxes that the reader marks to indicate the presence of particular suspected abnormalities.[3]

Although CWXSP data have been reviewed to assess the prevalence of pneumoconiosis,[4,5] to date no studies have described non-pneumoconiotic abnormalities noted on these films. We reviewed readings of the 51 374 posteroanterior radiographs taken from 1 June 1981 to 27 November 1989, and tabulated results for the 21 specific symbols in the "other symbols" section of the form.

The table shows the relative frequencies with which each symbol was marked. The symbol "co" (abnormality of cardiac size or shape) was the most often cited overall and by second readers, and "em" (emphysema) was the most often used symbol by first readers and the second most frequent abnormality overall. By contrast, "rp" (rheumatoid pneumoconiosis) was marked only once and "px" (pneumothorax) was noted only twice. First and second reader agreement on the presence of "other symbol" abnormalities was generally poor.

These findings highlight the need for further evaluation of the use and interpretation of the "other symbols" section of the ILO classification.

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1 Code of Federal Regulations. *Specifications for medical examinations of underground coal miners.* Washington: Government Printing Office, 1989. (Title 42, chapt 1, part 37.)
2 International Labour Office. *Guidelines for the use of ILO international classification of radiographs of pneumoconioses.* Geneva: International Labour Office, 1980. (Occupational safety and health series No 22 rev.)

Frequency of specific "other symbols" abnormalities reported by first reader only, by second reader only, and by both readers for chest radiographs taken in coal workers' x ray surveillance programme between 1 June 1987 and 27 November 1989 (n = 51 374)

Symbol	*Description*	*No (first reader only)*	*No (second reader only)*	*No (both readers)*
ax	Coalescence of small pneumoconiotic opacities	58	26	7
bu	Bulla(e)	35	172	14
ca	Cancer of lung or pleura	78	125	16
cn	Calcification in small pneumoconiotic opacities	165	20	0
co	Abnormality of cardiac size or shape	185	456	58
cp	Cor pulmonale	9	0	0
cv	Cavity	14	9	0
di	Marked distortion of intrathoracic organs	18	23	1
ef	Effusion	2	20	1
em	Definite emphysema	373	224	53
es	Eggshell calcification of hilar or mediastinal lymph nodes	13	0	1
fr	Fractured rib(s)	103	226	20
hi	Enlargement of hilar or mediastinal lymph nodes	62	238	13
ho	Honeycomb lung	4	11	0
id	Ill defined diaphragm	19	20	0
ih	Ill defined heart outline	20	13	0
kl	Septal (Kerley) lines	28	20	0
pi	Pleural thickening in interlobar fissure or mediastinum	140	34	6
px	Pneumothorax	0	2	0
rp	Rheumatoid pneumoconiosis	1	0	0
tb	Tuberculosis	151	187	15