Correspondence

each subject. It enabled the subjects' heart rate in the final load to be brought within two to five beats of 80% of \( \bar{x} \) maximum heart rate for age, a level which in our view conformed with recommended maxima for exercise testing (ref 18) but also was high enough to ensure the minimum extrapolation for estimation of \( \dot{V}O_2 \) max.

Cotes rightly draws attention to the imperfection of weight related \( \dot{V}O_2 \) max as a fitness indicator. Our main reason for using \( \dot{V}O_2 \) max per kg was its near universal use in other studies (such as those in table 5). In interpreting Van der Walt and Wyndham's prediction of the energy cost of walking and running we have used their formulas for an individual weighing 76 kg, the mean weight for our population, recognising that there is not a direct proportionality.

We would prefer to use a more sensitive index when we come to the stage of reporting on morbidity in relation to fitness but it is not clear what that index should be. The gross value of \( \dot{V}O_2 \) max would appear to be much less meaningful, and relating \( \dot{V}O_2 \) to lean body mass or other body dimensions also can be less than helpful in clarifying the relation between \( \dot{V}O_2 \) and relative functional capacity. Other indices such as W150—used by some authors across entire age ranges—seem to give no helpful basis for comparison. We would welcome suggestions from other readers and in the meantime will certainly consider that of Cotes—but what are the "truly independent variables?"

The 2 x 2 histograms were not used in any analytical process but only to present mean results for different categories of one dimension in relation to another dimension. We acknowledge that individuals may poorly estimate their participation in activity. Such large numbers cannot be individually monitored—even if they could this would then affect their behaviour. We did, however, attempt to make the questionnaire as dependable as possible by carefully piloting it and using trained interviewers.

We hope that some interest will have been aroused by the paper despite the imperfections of our measures and procedures, most of which arose from compromises which have to be made in field work between ideality and practicality.

Benzene exposure in chemical workers

Sir,—In the 8th revision of the International Classification of Diseases (ICD) other neoplasms of lymphoid tissue is coded 202 and polycythemia vera is coded 208. There is an error in table 4 of the paper by Bond et al (1986;43:685–91) in which other lymphatic tissue was reported as ICD 208. Since there is interest in the association between lymphomas and benzene exposure, it would be helpful if the authors could give the results obtained for ICD 202 specifically in order to derive maximum value from this interesting study.

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Dr Bond and Dr Cook reply:
We are grateful to Dr Johnson for calling attention to a small error which appeared in table 4 of our paper. The category “Other lymphatic tissue” as used in our mortality analysis program is not restricted to ICD code 208 as is implied, but also includes codes 202 and 203. This was an oversight on our part in preparing the manuscript.

The one death coded to this category was the multiple myeloma (ICDA = 203) which is discussed on page 690. No deaths were attributable to code 202. Our mortality analysis program does not have rates specific for ICDA code 202, which would allow for the calculation of an expected value. In 1975, however, 46% of all deaths coded to ICDA 202, 203, or 208 among United States white men were attributable to ICDA 202. Therefore, a rough estimate of the expected number of deaths in the cohort due to code 202 is (0.46 x 1.3) = 0.6.

References
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Lung function in coalworkers' pneumoconiosis

Sir,—In a recent report (1986;43:644–5) Zhicheng considers the issue of lung function in coal workers’ pneumoconiosis. Although this is an important topic, the study design used in this report limits the inferences which may be drawn from the data, particularly with regard to the aetiology of the deficits in pulmonary function.

I think that the most serious flaw in this study is the lack of quantitative information regarding smoking history—for example, in pack-years—in the four groups. Cigarette smoking is associated with both restrictive and obstructive lung defects and...
although Zhicheng states that 38%-45% of the subjects in all four groups were smokers, the analysis presented does not control for this important variable. It is therefore impossible to evaluate the contribution of coal dust to the pulmonary function deficits shown. (Matching cases and controls on smoking history would probably have been more appropriate since smoking is such a strong confounding factor.)

Further support for this contention is the fact that the current data relating the levels of respirable dust to radiographic progression of coal workers’ pneumoconiosis are equivocal. Bearing this in mind, it is difficult confidently to assume that a clear gradient in exposure level exists among groups B, C, and D. The statement in the last paragraph of the paper, “Cigarette smoking may have played a part in the deterioration in lung function but the impairment was also inversely related to coal dust” must therefore be questioned.

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References