McNamee also takes issue with some aspects of our interpretation of our results. We did not "imply that the highest possible value of Cohen's kappa is not always one." We merely pointed out that this value could not be attained when the marginal distributions are unequal. For each trial the marginal distributions as well as the kappas were presented, thereby providing complementary components of the inter-rater agreement.

The second point concerns the qualitative significance to be attached to various values of kappa. Labels such as "good," "excellent," "moderate" are arbitrary, as various authors have indicated, and depend to some extent on the subjective expectations of the investigators. Whereas we believe that in this context values of kappa in the order of 0.6 represent excellent agreement, the reader was given sufficient information to decide for herself.

The third, and most important point, concerns the connection between our results and relative risk estimates. The purpose of our case-control study was to generate hypotheses concerning past occupational exposure and cancer. There is misclassification in our exposure estimates and this misclassification leads to biased (toward the null) relative risks. It is not as important to estimate the true relative risk correctly as to determine whether or not there is excess risk. The key issue is whether the degree of misclassification is likely to be so great as to eliminate the chance of detecting excess risk. Statistical power for detecting excess risk is a function of several parameters, including the true relative risk, the degree of misclassification, and sample size. It would therefore be interesting to estimate the loss of power corresponding to levels of inter-rater agreement observed in our trials. This was beyond the scope of the paper, however. Failing such an analysis, we relied on a heuristic approach to interpreting the results. We were encouraged by finding Kappas in the order of 0.6. Furthermore, for various reasons that were outlined in the article we believe that the results of these trials provides a lower limit to the reliability of the exposure assessments carried out over a period of years in our case-control study.

We expect that the loss of statistical power corresponding to these levels of inter-rater agreement is not so great as to render the prospect of detecting excess risk a hopeless task. This expectation is premised on the unverifiable assumption that these "relatively high levels" of inter-rater agreement reflect similar levels of validity, and that such levels of validity combined with the sample sizes and appropriate values of other parameters yield adequate power for detecting excess risk.

Finally, it is important to recognise that an evaluation of our exposure assessment strategy should not

be made in isolation of the alternative methods. Previously, monitoring studies to discover occupational carcinogens have been based on subjects' job titles which were either obtained on death certificates or by more reliable means such as interviews. Although our approach entails misclassification, we are convinced that there is much more misclassification when a subject's exposure status is inferred simply from the job title than from the chemist-rater approach we have used.¹ It is in relation to the practical alternatives that we believe our approach provides reasonably accurate data. Had the results of the trials produced very low indices of agreement, we would probably have abandoned or greatly modified the project. The fact that they were not low encouraged us to carry on. We hope investigators will develop and evaluate methods of retrospective exposure assessment that would improve the prospects for case-control studies of occupational carcinogens.

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Pulmonary fibrosis in asbestos insulation workers with lung cancer

SIR,—Kipen and colleagues reported (1987;44:96-100) that all 138 cases of asbestos insulation workers with lung cancer studied histologically had asbestosis.

It is important to note that the histological definition of asbestosis used in this study was not one which would be generally accepted by United Kingdom pathologists. The subpleural connective tissue was considered part of the interstitium for the purposes of assessing interstitial fibrosis whereas in the United Kingdom only fibrosis in the interalveolar septa or around the respiratory bronchioles and alveolar ducts is usually considered.¹ In only 130 (94%) of the cases were one or more asbestos bodies seen. Asbestosis is never diagnosed in the absence of asbestos bodies using criteria conventional in the United Kingdom where, not infrequently, the diagnosis is rejected in favour of cryptogenic fibrosing alveolitis (idiopathic pulmonary fibrosis) on the grounds that "too few" asbestos bodies are present, even when the subject is known to have been an asbestos worker.

It appears that Kipen *et al* accepted any interstitial fibrosis as evidence of asbestosis in these workers known to have had heavy exposure to asbestos. I do not intend to suggest that the fibrosis seen in these cases was not caused by exposure to asbestos but to

draw attention to the different criteria for the histological diagnosis of asbestosis used by different workers.

This point is of considerable practical importance because in the United Kingdom compensation by the State for lung cancer is given to asbestos workers only if asbestosis or bilateral diffuse pleural thickening is also present. If United Kingdom pathologists used the criteria used by Kipen *et al* many more asbestos workers would be eligible for compensation for lung cancer.

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Metal polishing, stomach cancer, and clearing houses

SIR,—In May 1985 we were informed of a patient aged 37 with stomach cancer. He had worked as a metal polisher for a local firm for nine years from 1966 to 1975. The occupational history was taken by a conscientious medical student, who learnt that several of his former metal polishing colleagues had apparently died young from cancer.

The factory concerned was inspected by the Employment Medical Advisory Service (EMAS). Metal polishing was done by hand, applying polishing agent to a rotating brush. A venting system eliminated visible dust. The metals polished were primarily stainless steel and aluminium with some brass and chromium plated articles. The polish consisted of fatty acids, glycerides, mineral oil, waxes, and aluminium oxide abrasives.

A review of the employment records since 1956 established the names of 16 men who had worked in the polishing shop for one year or more. Of these, one man had collapsed and died at work at age 60, probably from a myocardial infarction. Four men, excluding the index case, had definitely died from cancer: one (age 65) from stomach cancer and three (ages 41, 46, and 64) from anaplastic cancer of unknown primary site. The number of cancers expected in this small group of workers, based on the 1982 age specific cancer registration rates for England and Wales, is 0.6, including 0.04 stomach cancers, although these figures are not adjusted for occupational class or for changes in cancer rates—for example, the decrease in incidence of stomach cancer since 1956.

Interestingly, there is some documentary evidence of an increased risk of stomach cancer in metal polishers. A Swedish report in 1983 noted an odds ratio of 9 for stomach cancer based on four deaths in a

cohort of 86 workers.¹ The ratio for all deaths from cancer was not significantly raised. Similarly, a paper from the United States in 1980 reported a ratio of 3.9 for gastric cancer based on five deaths in metal polishers working in the jewellery industry, using the expected distribution of deaths in all United States men for comparison.² The ratio for all cancers was not raised. Conversely, a study by Blair found no increased risk of cancer.³

In the United Kingdom occupational mortality reports from the Office of Population Censuses and Surveys are difficult to interpret since occupations are seldom sufficiently well defined to allow precise coding: this point is made specifically in relation to the definition of occupational unit 54 (which includes metal polishers) in the 1970-2 decennial report.⁴ For unit 54, however, a standardised mortality ratio of 1.3 (136/107) was reported for stomach cancer in men and 2.0 (36/18) for stomach cancer in women⁵ compared with the expected deaths based on social class specific rates, whereas the overall rates for deaths from cancer in men and women were not increased.

The reasons for presenting these data are twofold. Firstly, we think that further epidemiological investigation is desirable but we have not been able to assemble a sufficiently large local cohort of metal polishers, and we would welcome collaboration in other places. Secondly, we often become engaged in investigating occupational and other non-infectious diseases and, as with our work in infectious diseases, this usually entails assessing sporadic clusters of disease. In our experience this may only be done effectively if there is a central agency such as the Communicable Disease Surveillance Centre to act as a clearing house and reference point—not simply to collect but also to disseminate relevant information. In this investigation such a clearing house was sorely missed, and we would welcome suggestions as to how this need should be met.

Finally, we would like to thank Dr J Huckbody of EMAS for his help and advice.

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