Letters


Aldehyde disinfectants and health in endoscopy units

EDITOR.—I read with interest this report and would accept that it-forms the main outline of an employer's responsibilities. However, I would, however, question the statement that where an employee develops occupational asthma after exposure to glutaraldehyde and continues to work, that exposure cannot be avoided, that the employee must be made aware of the risks of continued exposure. The implication is that the employer is the worker's choice as to whether they can continue being exposed to glutaraldehyde. An employer has a responsibility to advise on fitness for work. In a case where asthma has been shown to be caused by glutaraldehyde it is not reasonable to leave the decision about continuing exposure to the employee, however well informed. The employer has the responsibility for protecting an employee's health. In these circumstances redeployment and retraining may be the best outcome that an employer can expect. The report drew attention to the need for pre-employment health assessment suggesting enquiry about asthma and other conditions. The authors did not comment on whether subjects with pre-existing asthma should be employed in jobs where exposure to glutaraldehyde may occur. This is a difficult issue that seems to have been avoided.

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Reply

EDITOR.—Dr Stevens makes two main points in his letter. Firstly—what is the management of a member of the endoscopy staff who develops glutaraldehyde related asthma? Secondly—what is the recommendation of the working party regarding employment of people with pre-existing asthma who will be required to work with glutaraldehyde?

We would like to point out that it was not within the remit of the working party to produce a manual detailing how health and safety policies might be implemented. It was assumed that individual hospitals will take steps to comply with COSHH regulations.

With regard to the first point, Dr Stevens has interpreted the report as leaving it up to the subject to decide whether he or she should continue to work with glutaraldehyde. We feel this is an extreme interpretation of what has been said. The report states quite clearly that employee choice should be left to the individual, but recognises that there may be circumstances when this is not possible or desirable. We have to assume that the development of occupational asthma in a member of staff working in an endoscopy unit will inevitably involve the local occupational health department, which will make an appropriate risk assessment and also inform the hospital management. This would take away the decision from the affected subject and give the responsibility to management.

It is reasonable to assume that any such decision will not be made in a vacuum. If the diagnosis of occupational asthma resulting from glutaraldehyde is not made, the employee will continue to work with glutaraldehyde. The employee should be removed from the working environment. If exposure can be reduced, however, and it is the considered opinion of the medical team responsible for looking to work in the environment is not a significant risk to the employee, and there is no suitable alternative workplace such that the subject wishes to continue to work, the hospital management, having been made aware of the pros and cons of doing so, we believe this is a suitable plan of action. The final decision would have to rest, however, with the manager of that department.

With respect to the issue of employing people with pre-existing asthma in jobs where exposure to glutaraldehyde may occur, we believe it would have been inappropriate for the working party to report on this specific issue as it has been, consistent with its stated intention to formulate recommendations rather than instructions. Once again a risk assessment would have to be carried out to take account of the severity of the disease, having been made aware of the pros and cons of doing so. The final judgement about fitness to work is the responsibility of an occupational physician and we did not think it was the role of the working party to preempt this.

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Correlation of PCNA with bromodeoxyuridine

EDITOR.—We noted with interest the paper by Weigangher et al [Gut 1993; 34: 1587–92] on proliferation cell nuclear antigen (PCNA) and its correlation with bromodeoxyuridine (BrdU). We would like to express our reservations about their conclusions. The most important one is their decision to only count the ‘strongly stained nuclei’ in the PCNA assay. This necessarily makes the assay highly subjective because of the difficulty of maintaining an identical standard for every nucleus that is assessed. The assay could only become reproducible in large numbers if either all stained nuclei were counted as positive, or if some form of extremely sophisticated imaging system that could differentiate stained nuclei on the basis of intensity and colour of their staining was used.

While a significant correlation between the two markers assessed has been shown, we believe that analysis has been performed on only 17 values. The correlation coefficient of 0·6 and Figure 1 shows the rather vague interrelation between PCNA and BrdU in this context. This observation is most important than the significant difference seen between the mean proliferation indices for two reasons. Firstly, there is ongoing debate about which fraction of the replicating population of cells PCNA measures—that is, the growth fraction, as in Ki67 labelling, or the S phase fraction as in tritiated thymidine or BrdU labelling. It may be dependent on the form of tissue fixation. 1–3

This study cannot identify which proliferating component has been labelled with any particular assay. Secondly, if there was a strong numerical relation between the two measured values for a given sample, then some form of paired statistical test should be appro- priate. No evidence is given that this sort of test has been performed in this study.

We do not feel that this paper does show a close relation between the PCNA and BrdU assays. We believe it is necessary to assess a greater number of biopsy specimens taken from a much greater number of subjects; to score all PCNA stained nuclei; and place more emphasis on the correlation between the assays rather than the actual numerical values measured.

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Reply

EDITOR.—We appreciate the critical evaluation of our paper by Dr Wilson and Dr Schofield. We agree with most of their comments, some of which have already been considered in the discussion of our article.

In an attempt at trying to mimic an S phase marker only strongly stained nuclei in the PCNA assay, the evaluation procedure certainly has to be highly standardised and the best way is by use of image analysis. If this standardisation cannot (for whatever reason) be achieved, it may be necessary to include all labelled cells in the PCNA analysis. The inclusion of all labelled cells in the PCNA assay, however, does not result in a significant correlation between the two markers in our study. We could possibly show such a correlation with a greater number of subjects, as suggested by Wilson and Schofield, and we agree that this correlation is