to extremely low concentrations and the hypomethioninemia persisted for several days (unpublished observation). A possible explanation for this unexpected result lies in the fact that methionine metabolism is influenced, and in opposite directions, by both methylenetetrahydrofolate reductase and adenosylcobalamin, the former through the propionyl-CoA synthetase reaction which increases the serum methionine. The latter is a coenzyme in the methylenetetrahydrofolate reductase reaction, this is the last reaction in the propionyl-CoA synthetase pathway along which methionine is catalysed to the citric acid cycle. Adenosylcobalamin, therefore, aids the catabolism of methionine and depresses its concentration, and the above finding suggests that adenosylcobalamin alone is active and that the action of methylenetetrahydrofolate is not expressed, and the methionine synthetase reaction therefore not activated by these very minute doses of vitamin B12. They do, however, invariably produce a reticulocyte response, often a very brisk one, which again suggests that vitamin B12 has an erythropoietic action which is independent of the methionine synthetase reaction, and hence, from both the methyfollate trap and the formate starvation hypothesis, in the absence of the thymidylate synthetase action as well. Unfortunately by the time the possible interaction of the results was realised the work could not be repeated. If, however, the above interpretation is correct it would probably take several days for these very small doses of vitamin B12 to correct the dU suppression test. This contention could therefore readily be tested by relating this interval to the daily reticulocyte count following a daily dose of 2 μg of vitamin B12 in pernicious anaemia. A clear reticulocyte response not necessarily the peak, occurring before the dU test is corrected would lend it support.

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Risk of inhaling cyanide during necropsy examination

I read with interest the article by Forrest, Galloway, and Slater on the risk of inhaling cyanide during necropsy on cases of cyanide poisoning.1 There is, admittedly, a theoretical risk of inhaling a large amount of cyanide, as observed by Andrews et al.2

The recommendation that a respirator be worn during the necropsy or that the stomach should be opened in a fume cabinet is commendable but suffers from one draw-
back. In one of my cases the diagnosis of cyanide poisoning was made only after opening the stomach. This case presented a sudden natural death and my diagnosis of cyanide poisoning, based solely on the smell, was greeted with considerable disbelief by the investigating police officers. Subsequent examination of the stomach revealed a history of a suicide note and an empty container but the impression of everybody concerned, including myself, was that of sudden natural death. At least I am fortunate that I can smell cyanide (My colleague at that time has never been able to).

Theoretically, then, a pathologist who could not smell cyanide would inhale potentially dangerous amounts of cyanide during such a necropsy. Should pathologists routinely wear respirators when performing any necropsy where the circumstances of death are not clear? Or should they routinely open the stomach in a fume cabinet in all such cases? When we refer to the changing face of pathology is it because pathologists of the future will be wearing gas masks? Perhaps readers should be told.

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DrS Forrest, Galloway, and Slater comment:

We are confident that either common sense or natural selection would prevail in the situation Dr Fernando describes. Pathologists will make a sensible judgement of the risks in a particular case and the precautions that reasonably ought to be taken in the light of all the circumstances, including their knowledge of their own offlactory capabilities.

Glove puncture in the post mortem room

I cannot allow Drs Weston and Locker’s comments on my criticism of their paper to go unchallenged. They have not correctly cited the paper of Hall et al.3 This study involved 664 technicians (588 anatomical pathology technicians and 76 auxiliary (technicians), not 76 as they claim, plus 774 consultants. It also included a control group of Coroner’s officers. Two cases of hepatitis B were indeed reported as Drs Weston and Locker state. However, these was in Coroner’s officer and therefore unlikely to be due to unnoticed glove puncture! The incidence in the air and control groups was therefore equal. The reported case of tuberculosis is almost certainly unrelated to glove puncture. The discussion at the end of the paper concludes that apart from the expected high rates of respiratory disorders, the digestive and infectious disease excess noted in the technicians was similar to the findings of a large scale survey of medical laboratory workers. I would therefore reiterate my conclusion that unnoticed glove puncture is not in itself a health hazard. Laceration of the skin is undoubtedly a health hazard but is not likely to be affected by more frequent glove changes. I agree with the other correspondents that the efforts to minimise the risk of blood born infection in the post mortem room would be better directed towards reducing that hazard. I have found that the available chain mail protective overgloves for the left hand are of great value in this respect.

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The teaching of death certification

Death certificates are usually issued by pre-registration house officers, often badly, and sometimes with only a mode of death as opposed to the disease producing death.1

