to extremely low concentrations and the hypomethioninaemia persisted for several days (unpublished observation).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 methylcobalamin and by adenosylcobalamin, the former through the methionine synthase reaction which increases the serum methionine. The latter is a coenzyme in the methylenyl-CoA mutase reaction, this is the last reaction in the proprionyl-CoA 3-oxoacid degradation pathway which methylcobalamin is catabolised to the citric acid cycle. Adenosylcobalamin, therefore, aids the catabolism of methionine and depresses its concentration. The above findings suggest that adenosylcobalamin alone is not necropsy, and that the action of methylcobalamin is not expressed, and the methionine synthase 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 mitotic feedback reaction, and a difference, from both the methylfolate trap and the formate starvation hypothesis, in the absence of the thymidilate synthase action as well. Unfortunately by the time the possible interactions became evident the results were ready and 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 2μg of vitamin B12 in pernicious anaemia. A clear reticulocyte response is not necessarily the peak, occurring before the dU test is corrected would lend it support.

T E PARRY
Ascot, Pen-y-Torpin, Dinas Powys,
South Glamorgan CF4 4HG


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 drawback. 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 analysis of the small quantity of cyanide in the stomach confirmed this diagnosis, and it is a regrettable oversight not to have had 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 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.

G C A FERNANDO
Central Pathology Laboratory, Histopathology Department, North Staffordshire Hospital Centre, Hanterhill, Stoke on Trent ST4 7PH


Drs Forrest, Galloway, and Slater comments:

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 olfactory capabilities.

Glove puncture in the post mortem room

I cannot allow Drs Weston and Lockyer’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, 76 hospital pathology 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 Lockyer state. However, in one of these it was the Coroner’s officer and therefore unlikely to be due to unnoticed glove puncture! The incidence in the at risk 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.

P J DUNN
Department of Pathology, Royal Infirmary, Castle Street, Worcester WR1 3AS


c-erb-B-2 expression in male breast carcinoma

Fox et al recently reported a complete lack of c-erb-B-2 expression in 21 cases of male breast carcinoma,4 while Wright et al reported overexpression in a single case.5 We have so far examined 33 cases of male breast carcinoma for c-erb-B-2 expression using the monoclonal antibody NCL-CB11 (Novocast- tra) and a standard immunoperoxidase technique. Omission of the primary antibody and a known positive case of female breast carcinoma were used as negative and positive controls, respectively. Membrane staining was completely absent in 20 cases, but positive membrane staining was present focally within the tumour in 12 cases and throughout the tumour in one case. Thus 39% of our cases show evidence of c-erb-B-2 overexpression. This figure is similar to the 35% reported by Gattuso et al6 in their series of 26 cases.

Our results show that a proportion of male breast carcinomas are associated with c-erb-B-2 overexpression, which is usually related to gene amplification. However, it remains to be seen whether this has the same prognostic importance as that seen in female breast carcinomas.

I H LEACH
O ELLIS
C W ELSTON
Department of Histopathology, City Hospital, Hucknall Road, Nottingham NG5 1PB


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.7