to extremely low concentrations and the hypomethioninaemia persisted for several days (unpublished observation).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 synthetase reaction which increases the serum methionine. The latter is a coenzyme in the methylmalonyl-CoA mutase reaction; this is the last reaction in the propionyl-CoA-succinyl CoA pathway along which methionine is catabolised to the citric acid cycle. Adenosylcobalamin, therefore, aids the catabolism of methionine and depresses its concentration in serum. The above findings suggest that adenosylcobalamin alone is active and that the action of methylcobalamin 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 by inference, from both the methylfolate trap and the formate starvation hypothesis, in the absence of the thymidylate synthetase action as well.

Unfortunately by the time the possible importance of these 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.

T E PARRY
Awelon, Pen-y-Turnpike,
Dinas Powys,
South Glamorgan CF6 4HG

- 1 Chanarin I, Deacon R, Lumb M, Perry J. Cobalamin and Folate, recent developments. *J Clin Pathol* 1992;45:277-83.
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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.³ This case presented as 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 investigations led to the discovery 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.

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

- 1 Forrest ARW, Galloway JH, Slater DN. The cyanide poisoning necropsy: An appraisal of risk factors. *J Clin Pathol* 1992;45:544-5.
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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 olfactory 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.*¹ This study involved 664 technicians (588 anatomical pathology technicians and 76 mortuary 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, one of these was in a 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 one 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

- 1 Hall AJ, Aw TL, Harrington JM. Morbidity survey of post mortem room staff. *J Clin Pathol* 1991;44:433-5.

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,¹ while Wright *et al* reported overexpression in a single case.² We have so far examined 33 cases of male breast carcinoma for c-erb-B-2 expression using the monoclonal antibody NCL-CB11 (Novocastria) 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 al*³ 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
I O ELLIS
C W ELSTON
Department of Histopathology,
City Hospital, Hucknall Road,
Nottingham NG5 1PB

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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