capsid antigen. The IgM titre was screened routinely at 1/8 but no further dilutions were assayed. The discrepancy in the titres reported from Manchester and Colindale on roughly contemporaneous samples can only be attributed to methodological differences.

Antibodies to cytomegalovirus were detected in the laboratory of Professor Harold Stern, St George's Hospital Medical School, using complement fixation as stated in our article. Numerous attempts were made to isolate this virus from the patient's urine but without success.

Because of limitations of space, we could only summarise the serological data in our article and we could not fully discuss the diagnostic possibilities. We agree that one cannot be dogmatic about the nature of this patient's illness. It is our belief that the most likely explanation is that she had a primary infection with EB virus to which she was unable to make an adequate immunological response. Indeed, the antibody titres to cytomegalovirus may be attributable to cross-reaction of antibody between this virus and EB virus.[1] However, we agree that we cannot exclude the possibility that this patient had a double infection with both viruses and that the impaired response to EB virus reflects the immunosuppression induced by cytomegalovirus.

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[1] Horwitz, C A, *American Journal of Clinical Pathology*, 1977, **67**, 210.

1918 influenza epidemic

SIR,—Minerva (16 December, p 1722) says that there are few suriving medical witnesses of the 1918 influenza epidemic to remember any peculiarities of the disease.

I was serving in the RAMC at that time and was temporarily seconded to be medical officer to the First Reserve Brigade of the South African Infantry as two of its MOs had succumbed to the disease. I do not remember the "unique stench" mentioned in the American paper quoted, but I do recall vividly the curious violet cyanosis which often occurred quite soon after the onset of the attack. This was quite different from the bluish-grey of ordinary cyanosis and also from the pinkish hue of carbon monoxide poisoning. The phenomenon was so remarkable and widespread that a medical journal (I think the *BMJ*) sent an artist to make a watercolour sketch of one of our patients (colour-photography not having been invented). The picture was duly reproduced but the patient died the same evening and the artist a few days later.

The course of the disease could be extremely rapid. For example, it was quite common for two or three men to collapse during a morning route-march, to be carried back to barracks on stretchers, and to die before nightfall. I believe that every soldier who contracted influenza before he had been in Europe for three weeks died and that the South African expeditionary force lost more men from the disease than from wounds and gas poisoning combined. The tragedy was made worse by the fact that no available treatment appeared to have any beneficial effect, and also because most of the patients were of Boer descent and did not understand English. Indeed, by a cruel irony, their fathers may well have fought *against* the British in the Boer War some 18 years before.

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Postoperative deep vein thrombosis in Nigerians on high-fibre diets

SIR,—It was with great interest that I read Mr U Osime's article (9 December, p 1607) and I would like to make a few comments.

Several years ago I visited Equatorial Africa and India with Mr Denis Burkitt and cross-questioned several hundred surgeons and doctors, and it is quite obvious that pulmonary embolism in these areas is very rare. Impressed by this fact, over five years ago we commenced giving bran routinely to our patients in one surgical ward—mainly urological. It was given preoperatively and postoperatively if the patients were able to take it. Since then we have had no case of pulmonary embolism and only one case of clinical deep vein thrombosis. Three other surgeons in different parts of the UK have had similar experience.

We conducted tests on 50 patients—the majority having had open prostatectomies—using ^{125}I-labelled fibrinogen. We found positive calf signs in 22% but in the vast majority these were transient and reverted to normal in 48 hours. There was no case of clinical deep vein thrombosis. I would be interested to know how many of Mr Osime's were also transient in appearance.

Might it be that high-fibre diets alter fibrinolytic activity? If so, this could explain why the Nigerians rarely develop pulmonary embolism.

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Dealing with residual bile duct stones

SIR,—Mr R Mason (23-30 December, p 1788) rightly points out the difficulties in wire basket extraction of gallstones via the T-tube track when the track is narrow or tortuous, and recommends a 12 French gauge or larger T-tube brought out directly lateral to the common bile duct.

A large T-tube should certainly be used and there is usually no difficulty in placing such a tube in the dilated common bile duct which has contained stones. The track closes equally promptly after removal of the tube whether 14 or 16 French gauge or smaller T-tubes are used. The largest steerable catheter for stone extraction (Medi-Tech Company, Watertown, Massachusetts 02172) is 13 French gauge and Burhenne recommends a minimum T-tube size of 14 French gauge.[1] If an 8, 10, or 12 French gauge T-tube has been used and stones are subsequently found then the T-tube may be removed and a guide wire inserted, over which successively larger catheters are passed to dilate the track up to 14 French gauge. This may be done immediately before a first attempt at stone extraction six weeks after operation.

I would caution surgeons against bringing the T-tube out too directly. Many patients undergoing cholecystectomy are very obese and the T-tube exit wound may move downwards several inches when the patient stands up a few days after operation. If the T-tube takes too direct a route to the skin it can be pulled out of the common bile duct. I have heard of a case in California that had fatal results. The T-tube should therefore describe a smooth curve between the common duct and the skin wound.

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[1] Burhenne, H J, in *Advances in Surgery*, vol 10, ed W P Longmire, p 121. Chicago, Year Book Medical Publishers, 1976.

Breathing and control of heart rate

SIR,—Your leading article (16 December, p 1663) entitled "Breathing and control of heart rate" comments on the physiological good sense of swallowing water to relieve faintness. Although this manoeuvre may stimulate respiration and thus relieve bradycardia it may also directly cause slowing of the heart.

The majority of reported cases of "deglutition syncope" appear to be associated with oesophageal abnormalities,[1] but even in people without oesophageal disease syncope may be so severe and repeated as to require insertion of a demand pacemaker.[2] In susceptible individuals a drink of cold water might therefore be an appropriate treatment for supraventricular tachyarrhythmias rather than for bradycardiac syncope.

Your leading article also draws attention to the complex interaction between lung inflation and bradycardia initiated by face immersion. This interaction has recently been clarified.[3] Our results confirm that breath holding at inspiration leads to bradycardia, and show that face immersion has a marked effect only if performed at expiration. These findings explain why other works have not been able to demonstrate a consistent effect of lung volume on the diving response in man, since bradycardia is seen irrespective of face immersion at high lung volumes.

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[1] Tomlinson, I W, and Fox, K M, *British Medical Journal*, 1975, **2**, 315.
[2] Wik, B, and Hillestad, L, *British Medical Journal*, 1975, **3**, 747.
[3] Openshaw, P J M, and Woodroof, G M F, *Journal of Applied Physiology*, 1978, **45**, 783.

Polyunsaturated fatty acids and colchicine in multiple sclerosis

SIR,—We were interested to read the report by Dr D Bates and others (18 November, p 1390) on the use of polyunsaturated fatty acids (PUFA) supplementation in multiple sclerosis (MS). We think that it is important to point out that the evening primrose oil (Naudicelle) capsules used in this trial contained dyes including tartrazine and ponceau R. In unpublished studies we have shown that both these dyes block the conversion of PUFA to prostaglandins (PGs) and this effect of tartrazine has also been reported by others.[1] The importance of this lies in the fact that the major biological function of the PUFAs is to give rise to PGs.[2] The effect of PUFAs in suppressing experimental allergic encephalomyelitis in rats is completely abolished by inhibition of their conversion to PGs.[3]

In preparation for a double-blind placebo controlled trial we have for the past six months