thromboembolic complications: six patients died of pulmonary embolism, seven had deep vein thrombosis as a presenting symptom, and three had evidence of amyloidosis. We report here a case of multiple myeloma presenting as venous gangrene of feet.

A 71-year-old woman presented with bilateral deep venous thrombosis, gangrene of her right foot, and a pre-gangrenous state of her left foot. She was drowsy, dehydrated, and hypothermic. Her peripheral arterial pulsations were normal, but she had swelling of both calves, which were warm and tender, and all five toes of the right foot were black and gangrenous.

The urine contained 2.8 g/l (280 mg/100 ml) protein and more than 100 pus cells per field. It was positive for Bence Jones protein. Her blood urea on admission was 86 mmol/l (518 mg/100 ml), haemoglobin 14.8 g/dl, and leucocyte count $54.9 \times 10^9/l$ ($54\,900/mm^3$), with 91% neutrophils, 6% lymphocytes, and 3% monocytes, but no immature cells. Blood film showed red cell rouleaux formation. The platelet count was $315 \times 10^9/l$ ($315\,000/mm^3$) and E.S.R. 63 mm in 1 h. The total plasma protein concentration was 97 g/l (9.7 g/100 ml), albumin 22 g/l (2.2 g/100 ml), and globulin 75 g/l (7.5 g/100 ml). Serum protein electrophoresis showed grossly increased γ -globulins with a sharply defined band of abnormal protein in the β position on starch gel electrophoresis. Serum immunoelectrophoresis showed the paraprotein to be an IgG and transferrin was reduced. On immunodiffusion IgG was shown to be increased to 68 g/l (6 800 mg/100 ml) (normal 8-16 g/l), IgA 1.1 g/l (110 mg/100 ml) (normal 0.9-4.5 g/l), and IgM 0.34 g/l (34 mg/100 ml) (normal 0.6-2.8 g/l). The Sia test for macroglobulins was negative and no cryoglobulins were detected. Bone marrow examination showed a marked increase in mature plasma cells with depressed erythropoiesis, and the haemoglobin level later dropped to 7.9 g/dl. Serum calcium was normal at 2.45 mmol/l (9.8 mg/100 ml) but serum phosphate was increased to 2.5 mmol/l (7.8 mg/100 ml) owing to renal failure. Serum urate was 79.7 mmol/l (13.4 mg/100 ml). Skeletal survey, which included x-rays of chest, pelvis, upper femora, skull, mandible, humeri, and dorsolumbar spine, showed no evidence of myeloma.

On intravenous fluids her blood urea began to fall and with further treatment it dropped to 8.9 mmol/l (54 mg/100 ml). She was treated with intermittent courses of melphalan and prednisolone with complete disappearance from her urine of Bence Jones protein. The total plasma protein dropped to 73 g/l (7.3 g/100 ml), albumin 32 g/l (3.2 g/100 ml), and globulin 41 g/l (4.1 g/100 ml) and the electrophoretic band due to abnormal protein became faint. IgG fell to 36 g/l (3 600 mg/100 ml). There was a remarkable improvement in her gangrene.

The incidence of venous gangrene in multiple myeloma is rare and it is interesting to note the remarkable improvement of the gangrene in this elderly woman with the treatment of multiple myeloma alone and without any surgical intervention. In view of the good arterial supply to the feet the superficial gangrene was attributable to occlusion of venules associated with the high level of IgG and hypothermia.—We are, etc.,

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¹ Catovsky, D., et al., *British Medical Journal*, 1970, 3, 438.

Illness in the Clouds

SIR,—Your leading article (8 February, p. 295) has caused me some disquiet. I travel not infrequently by air and am therefore one of the doctors who may be expected on statistical grounds to be available in an emergency. As a microbiologist engaged for the past 10 years in administration, this prospect fills me with no enthusiasm. Can anyone advise me on how to prepare for it? Do I need to carry an emergency kit, go

on a course, and carry extra international insurance? On reflexion, I shall avoid air travel where possible—and also reading the *B.M.J.*!—I am, etc.,

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New Causes of Malignant Hyperpyrexia

SIR,—Dr. J. A. Lack (4 January, p. 36) raised the possibility of inadvertent administration of halothane to a patient who developed malignant hyperpyrexia with nitrous oxide.¹ He pointed out that halothane would be present in the gas delivered from most anaesthetic machines due to halothane liberated from rubber breathing tubes, etc. Other sources of contamination have been found such as elastomeric seals and certain rigid plastics within anaesthetic apparatus.²

As we were aware of this danger we took the following precautions: (1) the vaporizer was removed from the machine (a conventional B.O.C. Mk II Boyle's machine); (2) all rubber tubing, rubber connexions, and mask were brand new; and (3) the machine was purged with a nitrous oxide/oxygen gas mixture at a flow rate of 8 l/min for about 30 minutes before induction of anaesthesia. It could be argued that even with these precautions enough halothane could still be present to induce malignant hyperpyrexia. However, during the third anaesthetic given to the patient, which consisted of thiopentone without nitrous oxide, oxygen was administered from the same machine and no pyrexia occurred. A second patient, the above patient's homozygous twin, received a thiopentone/oxygen anaesthetic similarly with no rise in temperature.

There is a potential danger to patients highly susceptible to halothane in leaving the vaporizer on the machine even though it is in the "off" position. On a hot morning in June 1973 we anaesthetized a third patient susceptible to malignant hyperpyrexia using an anaesthetic machine with the halothane vaporizer (Fluotec Mk II) in the "off" position. The patient developed a low-grade pyrexia towards the end of the procedure, which had lasted 1½ hours, and on investigation a faint smell of halothane vapour was detected in the gas mixture. A Hook and Tucker ultraviolet halothane meter indicated that a low but measurable concentration of halothane was present (less than 0.25% v/v). Professor J. S. Robinson and his colleagues in the University of Birmingham investigated our suspect vaporizer in their laboratory under similar conditions and found that there was a constant leak of halothane vapour of between 4 and 9 p.p.m. v/v. However, when the gas flow was first turned on comparatively high levels of halothane vapour were delivered; initial concentrations were well in excess of 123 p.p.m. v/v and fell rapidly to 10 p.p.m. v/v after 15 minutes. The effect of a rising ambient temperature (as we had experienced during the anaesthetic) was to almost double the constant leak. Professor Robinson and his colleagues have described their analytical technique.³

In the light of more recent experience it is impossible to know whether the low grade pyrexia in the third patient was due to nitrous oxide, low concentrations of halothane, both these drugs acting together,

or other cause. When anaesthetizing a patient thought to be highly susceptible to halothane for any reason it would be sensible to take the precautions outlined above. We now use Cyprane equipment comprising a Quantiflex gas mixer, single vaporizer, Selectatec back bar, and a conventional Magill attachment. No vaporizer has ever been fitted to this equipment and we feel that the inadvertent administration of halothane or other anaesthetic vapours is virtually impossible.—We are, etc.,

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¹ Ellis, F. R., et al., *British Medical Journal*, 1974, 4, 270.

² Robinson, J. S., Thompson, J. M., and Barratt, R. S. Personal communications.

³ Robinson, J. S., Thompson, J. M., and Barratt, R. S., *British Medical Journal*, 1974, 3, 515.

Changing from a High- to Low-dose Oral Contraceptive

SIR,—The trend towards reducing the steroid content of combined-type oral contraceptives continues, and products containing 20-40 µg/day ethynylestradiol are now available or on trials in many countries.¹ In the hope of reducing adverse side effects of oral contraceptives many practitioners will transfer patients to these new products. An important problem for such women is to determine whether the sudden reduction in dose is associated with "escape" ovulations during the first cycle of treatment with a low-dose preparation.

We have investigated this problem in 10 healthy young women who were switched from high-dose products (Gynovlar (3 mg norethisterone acetate + 50 µg ethynylestradiol) or Nordiol (250 µg D-norgestrel + 50 µg ethynylestradiol)) to a new low-dose preparation, Nordette (150 µg D-norgestrel + 30 µg ethynylestradiol). Venous blood specimens were collected daily during the first treatment cycle following the change in medication. Plasma progesterone and luteinizing hormone (LH) were measured by specific radioimmunoassays. Progesterone concentration did not exceed 0.9 µg/l in any specimen, while LH concentration remained below 21 IU/l.

We conclude that transferring patients from a high-dose oral contraceptive to one containing only 30 µg ethynylestradiol per day is not likely to allow escape ovulations during the first cycle with the new product. It would therefore seem unnecessary to advise the use of additional contraceptive precautions to patients who make this change in oral contraceptive preparations.—We are, etc.,

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¹ Woutersz, T. B., *Current Medical Research and Opinion*, 1974, 2, 95.

² Schneider, W., Spona, J., and Matt, K., *Contraception*, 1974, 9, 81.