been substantiated. We were, however, somewhat disappointed after their statement that there was "a need for short-term and long-term controlled clinical trials to assess the benefits and risks involved in administering hormone therapy to postmenopausal women" to find that no assessment had been made of the benefits to the subjects they studied, and while documenting a substantial occurrence of "venous thromboembolic disease" in these women, they omitted to investigate the natural incidence of thromboembolic events (not requiring admission to hospital) in untreated late middle-aged oophorectomized Aberdonian women.

In this respect we feel that it is pertinent to mention our own experience of thromboembolic events complicating mestranol therapy for "menopausal osteoporosis." Since January 1969 we have been conducting a double-blind controlled trial of mestranol in a dose of 20-40 µg/day in oophorectomized women. At the present time we have just over 100 women aged 37-58 (mean 49.5 years) who have been taking either mestranol or placebo for between 1-28 months (mean 12 months), and to date we have had only one thromboembolic complication; in a woman of 47 whom we discovered on breaking the code had been taking the placebo. Thus, in the 50 women given 20-40 µg mestranol per day for an average of 12 treatment months we have recorded no thromboembolic episodes. Though the mean age of the women we studied (50) was less than the group studied by Dr. Gow and Professor MacGillivray (54), and the mean times since the menopause were four and nine years respectively, we feel that their dogmatic statement in the absence of adequate controls that "The gravity of the adverse effects far outweighs any beneficial ones and precludes the use of mestranol alone for long-term hormone replacement therapy in postmenopausal women" to be of dubious authenticity.—We are, etc.,

J. M. AITKEN
D. McKAY HART

University Department of Medicine,
Western Infirmary,
Glasgow W.1

Salmonella cubana and Carmine Dye

SIR,—In 1966 *Salmonella cubana* was isolated from cases of human salmonellosis in hospitals in the U.S.A.^{1,2} and U.K.³ Carmine dye used in studies of gastrointestinal function was shown to be the probable source of infection. Carmine dye is produced from the cochineal insect, which is obtained principally from South America and the Canary Islands.

Investigations in the British Isles confirmed the American findings, and as a result the *British Pharmaceutical Codex 1968* stipulated that batches of carmine dye should conform to a test for freedom from salmonellae.

A child aged 2 years had been in hospital since birth. When he was 1 day old four-fifths of his small bowel had been resected and consequently his stools were usually loose. He had received carmine markers on several occasions but until the present incident salmonellae were never isolated from stool cultures. On 1 February 1971 he again received a carmine marker and five days later his stools became green and

he was pyrexial. He had severe vomiting for several days and required parenteral fluids. *S. cubana* was isolated from the faeces on 8 February. Stool specimens were examined from ward contacts and a 9-year-old boy, who had never received carmine, was found to be a symptomless excretor of *S. cubana*. This serotype was isolated from carmine dye capsules in use in the hospital and from a stock bottle of carmine which had been used to fill the capsules. Inquiries showed that the dye in use was old stock received by the hospital pharmacy in May 1967 and therefore manufactured before the change in the requirements of the *British Pharmaceutical Codex 1968*.

S. cubana is rarely isolated from humans in the United Kingdom and the records of the Salmonella Reference Laboratory, Colindale, show fewer than 10 isolations per annum over the last 10 years. When this serotype is isolated from hospital patients the association with carmine dye should be remembered as old stocks of carmine dye may remain a potential source of infection.—We are, etc.,

B. ROWE
M. L. M. HALL

Salmonella Reference Laboratory,
Central Public Health Laboratory,
Colindale

R. L. NEWMAN
G. REHN

Queen Mary's Hospital for Children,
Carshalton, Surrey

- Lang, D. J., Kunz, L. J., Martin, A. R., Schroeder, S. A., and Thomson, L. A., *New England Journal of Medicine*, 1967, **276**, 829.
- Komarmy, L. E., Oxley, M. E., and Brecher, G., *New England Journal of Medicine*, 1967, **276**, 850.
- Lang, D. J., Kunz, L. J., and Moore, B., *Communicable Disease Centre, Salmonella Surveillance Reports*, 1966, No. 53, p. 2.

Cadmium Fume Poisoning

SIR,—As death from the inhalation of cadmium fumes is rare and preventable it seems important to report another fatality from this cause. Cadmium poisoning has been reviewed^{1,2} and the last death in the United Kingdom from inhaling the fumes was reported in 1966.³

A plumber twice brazed a valve into a copper pipe, twice because the joint failed under test, using silver solder and an oxyacetylene torch. There was no ventilation in the room where he worked. The total brazing time was 20 minutes, and his mouth was 30 cm from the valve. Severe symptoms and signs began to develop an hour or so later and were diagnosed as influenza with bronchitis, amended later to pneumonia from a welding accident. He died on the fifth day after brazing the valve.

At necropsy both lungs were completely and uniformly consolidated as in early grey hepatization and many vessels were thrombosed. Lung cultures were sterile and cadmium concentrations for 100 g wet weight were lung 0.15 mg, liver 0.1 mg, and kidney 0.5 mg. The lung histology was typical of cadmium pneumonitis,^{3,4} the prominent features being a single layer of P.A.S.-negative cells (type I pneumocytes perhaps)—some mitosing—lining the air spaces; hyaline membrane; arterial thrombosis; oedema; and congestion. Other organs showed no significant abnormality.

The diagnosis of cadmium fume poisoning was made from the lung histology and con-

firmed by using Browne's⁵ list of the hazards of oxyacetylene torch welding and by the movements of two men who were with the deceased around the time of the accident and who remained well. The cadmium source was the silver solder, an alloy containing 24% to 26% cadmium.

Cadmium fume is produced by overheating the metal, its alloys, and its compounds. Adequate ventilation prevents poisoning.

Mr. A. Hoole of the North-western Forensic Science Laboratory estimated the cadmium concentrations.

I am, etc.,

R. M. WINSTON

Hope Hospital,
Salford, Lancs

- Turner, F. M., *Cadmium Poisoning* (A.E.R.E. Med/M17), London, H.M.S.O., 1957.
- Kendrey, G., and Roe, F. J. C., *Lancet*, 1969, **1**, 1206.
- Beton, D. C., Andrews, G. S., Davies, H. J., Howells, L., and Smith, G. F., *British Journal of Industrial Medicine*, 1966, **23**, 292.
- Smith, J. P., Smith, J. C., and McCall, A. J., *Journal of Pathology and Bacteriology*, 1960, **80**, 287.
- Browne, R. C., *The Chemistry and Therapy of Industrial Pulmonary Disease*. Springfield, C. C. Thomas, 1966.

Homoeopathy

SIR,—On the radio (29 April) publicity was given to the system of homoeopathic medicine and to a claim that the common cold might be cured in a few hours.

Our profession generally does not appear much concerned about homoeopathy these days. Perhaps it believes that, unlike too many of our more scientifically developed remedies, Hahnemann's system at least does no harm. But does it do any good? Ought there to be controlled trials to establish, say, whether the Department of Health might beneficially establish homoeopathic cold-cure units? In short, what is the truth?

Because there was so much public concern over the tragic case of an attractive young athlete, the Medical Research Council investigated the claims of the Ringberg Clinic. Should we not be equally concerned about the efficacy of a system of medicine which has enjoyed the confidence of our most recent Sovereigns?—I am, etc.,

JOHN M. POTTER

Department of Neurological Surgery,
Radcliffe Infirmary,
Oxford

Buruli Ulcer in Ghana

SIR,—MacCallum and his colleagues¹ described chronic skin ulcers with undermined edges caused by an acid-fast bacillus which was later named *Mycobacterium ulcerans*. These first cases were seen in Australia but since 1958 similar lesions have been recognized in Uganda where the term Buruli ulcer was first used. I wish to report a case of Buruli ulcer seen in Ghana.

A 7-year-old girl was admitted to the Korle Bu Teaching Hospital in Accra in October 1969 with painful swelling of her left leg. On examination the child was in pain and ill, temperature 103°F (39.4°C) and pulse rate 120. There was cellulitis of the left leg, patches of necrotic skin on the lateral side of the thigh, and pustules and a small ulcer on the medial aspect of the calf. Pitting oedema was found below enlarged left inguinal lymph nodes, and fluctuation was elicited in the thigh. Investi-