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.,

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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.,

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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.,

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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.,

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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-

gations: haemoglobin 7.6 g/100 ml, white cell count 18,000/mm³, and a negative sickling test.

A diagnosis of late pyomyositis was made and after 24 hours treatment with penicillin and streptomycin the thigh was drained under general anaesthesia, and obviously necrotic skin was excised. Wound swabs yielded a mixed growth of coliforms, but acid-fast bacilli were not sought at this stage.

In spite of apparently free drainage from the leg, appropriate antibiotics, piecemeal excision of more necrotic skin, and transfusion of one unit of blood, the child remained very ill three weeks after admission. Re-examination under general anaesthesia showed that the ulcer on the thigh now extended over 3% of the body surface and the lateral edge only was adherent to underlying granulations. Elsewhere the ulcer margin was undermined 6 cm towards the groin and up to 12 cm distally, lifting the skin off the patella and linking the main ulcer in the thigh with smaller ulcers either side of the tibial tubercle. Thick plaques of gritty necrotic fat were excised from under the skin flaps and much cheesy debris was wiped out of the large cavity. A biopsy of skin and subcutaneous tissue was taken from the superior limit of the lesion. Even at this unsuitable site the histology was typical of Buruli ulcer, and clusters of acid-fast bacilli were seen in Ziehl-Neelsen stained sections. An attempt to culture *Mycobacterium ulcerans* from necrotic fat was not successful.

During the week after operation the child lost her fever and began to eat, and she was cheerful and pain free for the first time. The discharge from the ulcer lessened and the undermined edges began to stick down to healthy granulations. Skin grafting was performed four weeks after débridement, but was only partially successful. The need for more aggressive excision was not appreciated until the remaining ulcer showed signs of renewed activity, but further débridement and skin grafting eventually secured healing.

This child was admitted with extensive disease which probably corresponds to the "fulminating" lesion described by the Uganda Buruli Group.² Ulceration had only just started but severe secondary infection was already well established. She was in pain and very ill, features which are rare in uncomplicated *Mycobacterium ulcerans* infection, and this diagnosis was not considered until the ulcer continued to enlarge in spite of apparently adequate treatment. It is interesting to note that fever and malaise persisted until most of the necrotic debris had been removed from the leg, but thereafter the child was well and lively throughout the weeks of treatment needed to secure healing of her ulcer.

In Uganda cases of Buruli ulceration cluster round tributaries of the Nile flowing through marshy ground. One woman with a probable Buruli ulcer lived in the same village as the patient and used the same water supply, and a second woman with a suspicious ulcer lived near the mouth of this river. A third patient, a child with a large ulcer on her elbow, lived near a second river which, like the Densu, flows across the Accra plains within 50 miles (80 km) of the capital. It seems possible that a reservoir of infection exists in relation to these two rivers and that more cases will be recognized in this area.—I am, etc.,

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¹ MacCallum, P., Tolhurst, J. C., Buckle, G., and Sissons, H. A., *Journal of Pathology and Bacteriology*, 1948, **60**, 93.

² Uganda Buruli Group, *British Medical Journal*, 1970, **2**, 390.

Use and Abuse of Thyroidectomy

SIR,—We should like to comment on your recent leading article (20 March, p. 619) on the use and abuse of thyroidectomy for thyrotoxicosis, in which you deal principally with the sequelae of operation for thyroid cancer. While by and large we agree that the recurrent laryngeal nerves are at particular risk in operation on the undifferentiated anaplastic tumour, we would mention the advantages of surgery following deep x-ray therapy. The gland is greatly reduced in size and relatively avascular, and unless infiltrated by cancer the recurrent laryngeal nerve may be less at risk by using a special technique of pledget dissection.

But we cannot accept your statement that the morbidity of "hypoparathyroidism" is related to interference with the parathyroids or their blood supply. Postoperative hypocalcaemia seldom follows thyroidectomy for non-toxic goitre even where resection has been radical.^{1,2} In another context hypocalcaemia is unusual in patients (excluding those with overt bone disease) subjected to removal of a parathyroid adenoma and in whom biopsy of the remaining parathyroids has been carried out. In contrast postoperative hypocalcaemia is not uncommon following thyroidectomy for thyrotoxicosis particularly in the young, previously severely thyrotoxic patient who has been left with a small remnant. Here there is good evidence to link the hypocalcaemia with thyrotoxic osteodystrophy, which is generally manifest preoperatively by raised levels of serum alkaline phosphatase. Hypocalcaemia and related symptoms are generally transient and cease with calcium repletion within the hungry bones.

It is now doubtful if Fourman's³ thesis of partial parathyroid insufficiency has real validity and several reports and controlled trials⁴⁻⁷ have failed to substantiate his findings. Indeed, while accepting that hypocalcaemia, albeit as a rule of temporary nature, may follow ablative surgery for thyroid cancer, it is now considered that the term "hypoparathyroidism", when used for hypocalcaemia developing within one or two days of thyroidectomy for thyrotoxicosis, is misleading and should be discarded.—We are, etc.,

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¹ Michie, W., et al., *Lancet*, 1971, **1**, 508.

² Murley, R. S., and Peters, P. M., *Proceedings of the Royal Society of Medicine*, 1961, **54**, 487.

³ Davis, R. H., Fourman, P., and Smith, J. W. G., *Lancet*, 1961, **2**, 1432.

⁴ Michie, W., Stowers, J. M., Frazer, S. C., and Gunn, A., *British Journal of Surgery*, 1965, **52**, 503.

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⁶ Billis, A., Montgomery, D. A. D., and Clark, G. E., *Irish Journal of Medical Science*, 1967, **105**.

⁷ Stowers, J. M., Michie, W., and Frazer, S. C., *Lancet*, 1967, **1**, 124.

Ulcerative Colitis and Rectal Carcinoma

SIR,—In his interesting article (24 April, p. 203) Mr. S. Aylett claims that the risk of cancer arising in the retained rectal stump after colectomy and ileorectal anastomosis for ulcerative colitis is relatively slight because such growths developed in only 2% of the

350 surviving patients on whom he had performed this operation between 1952 and 1968 inclusive. To many people, however, an incidence of even one case in 50 may not seem insignificant. Moreover, this bald statement of frequency almost certainly underestimates the continued capacity of the diseased rectal stump in these cases to produce malignant tumours. As is well known, the predisposition to large bowel cancer in ulcerative colitis is a cumulative process, increasing year by year,¹ and it may not be for 10-15 years after colectomy and ileorectal anastomosis that the diseased stump of rectum begins to reveal its true malignant potential. Thus no less than four of the carcinomas in Mr. Aylett's cases arose in the 29 patients who had their operations performed during the years 1952-55, the incidence of rectal cancer in this group, who were all followed up at least 15 years after operation, being accordingly of the order of 14%. We would anticipate, therefore, that when all his patients have been followed up for a similar period of time, Mr. Aylett may have many more rectal carcinomas to report.

Operation for colitis is most often undertaken in adolescence or early adulthood, and these young patients may nowadays reasonably expect to enjoy several decades of life after successful surgical treatment. Mr. Aylett's long-term data unfortunately do little to dispel the nagging fear that, if their operation takes the form of a colectomy and ileorectal anastomosis instead of the more usual complete proctocolectomy and ileostomy, they may be exposed to a serious risk of developing rectal cancer. It seems to us, therefore, that whatever may be the immediate advantages of this operative technique—and we would not deny that in some cases they may be considerable—the surgeon who elects to use it in the treatment of ulcerative colitis assumes a grave responsibility in leaving his patients inadequately protected against the risk of subsequent malignant disease. True, there are some hopes that it may be possible by means of rectal biopsy to select patients specially liable to produce colitis carcinomas,² but no one could pretend that the reliability of this method is as yet anything like established.—We are, etc.,

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¹ de Dombal, F. T., Watts, J. McK., Watkinson, G., and Goligher, J. C., *British Medical Journal*, 1966, **1**, 1332.

² Morson, B. C., and Pang, L. S. C., *Gut*, 1967, **8**, 423.

Encouraging Recruits in Medicine

SIR,—I feel I must add my twopenceworth to your columns on the subject of medical students' remuneration (3 April, p. 51, and 17 April, p. 17). I have just finished working for three years for the Medical Research Council, where my salary was almost £2,000 p.a. I have now returned to medical school to complete the clinical course on a grant of £314 p.a. I certainly do not consider I am making a "sacrifice"—on the contrary, I think I am very lucky.

In my naïveté I am astonished at the attitudes evinced by some of your student correspondents, and in conversations I have had in my own medical school. A small pro-