in one or more skeletal muscles, I asserted that multiple abscesses were a classical feature of the disease. A classical presentation is not necessarily the most commonly encountered but is one in which the diagnosis is most likely to be correct and generally acceptable.—I am, etc.,

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Malignant Carcinoid Tumour with Gangrene of the Small Intestine

SIR,—Dr. I. M. Murray-Lyon and others (29 December, p. 770) report four cases of malignant carcinoid tumour with gangrene of the small intestine and discuss the cause of the ischaemic change. Vascular change with elastic tissue proliferation due to the presence of tumour—elastic vascular sclerosis—is favoured by Anthony and Drury,[1] while Dr. Murray-Lyon and his colleagues suggest that a combination of restriction of blood flow due to the presence of tumour tissue and the characteristic fibrotic reaction in the root of the mesentery may lead to thrombosis of the mesenteric vessels. The vessels in their four cases also showed elastic tissue proliferation but as the lumen was not strikingly narrowed this was considered unimportant.

A case recently presented at this institute in an 83-year-old woman dying of peritonitis following gangrene of the small intestine due to thrombosis of the superior mesenteric artery as it passed through an isolated, well-defined (3 × 2 × 2 cm) secondary deposit of carcinoid tumour in the mesentery. The primary tumour, an ulcerated, raised area 7 cm in diameter, was in the gangrenous loop of the small bowel. A solitary metastasis, 2 cm in diameter, was present in the liver, but there had been no clinical evidence of carcinoid syndrome. Histological examination showed the wall of the superior mesenteric artery to be surrounded by tumour tissue with thrombus in the lumen. The wall itself showed proliferation of elastic tissue but the lumen was not narrowed. The thrombus was confined macroscopically to the area running through the tumour deposit. Mesenteric fibrosis was not seen macroscopically. In the primary tumour the vessels, both arteries and veins, also showed proliferation of the elastica without marked narrowing of the lumen. This is in contrast to the case reported by Anthony and Drury,[1] in which such vessels were reported as near normal. In the veins these changes were accompanied by tumour cell infiltration and thrombosis.

The present case thus stresses the direct association between tumour and thrombosis rather than an indirect association via elastic vascular sclerosis. Like Dr. Murray-Lyon and his colleagues we feel it is difficult to accept that the gangrene was due to elastic vascular sclerosis. In our case, as in theirs, elastic tissue changes were present, but the vessel lumen was not narrowed though it was obstructed by thrombus. This interpretation in no way detracts from the clinical value of recognition of patients with carcinoid syndrome as "at risk" for mesenteric thrombosis.—We are, etc.,

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[1] Anthony, P. P., and Drury, R. A. B., *Journal of Clinical Pathology*, 1970, 23, 110.

Radiation Protection in Dentistry

SIR,—With reference to your leading article on this subject (19 January, p. 86), I would like to point out that the lack of response to the National Radiological Protection Board's offer of radiation surveys of dental *x*-ray equipment was due almost entirely to a lack of knowledge on the part of the dental profession of the availability of this service.

The situation has recently been corrected. In 1973 the National Radiological Protection Board circulated all members of the British Dental Association with details of its monitoring and survey scheme for dental *x*-ray units. This was supported by articles in the dental press on all aspects of radiation protection, radiography, and radiology in dentistry. I would submit that this has led to an increased awareness throughout the dental profession of the need for routine dental *x*-ray equipment checks and also for all operators to make sure that their standards of both safety and efficiency are constantly under critical review in the interests of patient, staff, and operator.—I am, etc.,

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False Interpretation of Fetal Heart Monitoring

SIR,—Recent observations (29 September, p. 694; 17 November, p. 420; 5 January, p. 39) on the false interpretation of fetal heart rate patterns prompt us to suggest that all monitoring equipment should have the facility to permit display of the E.C.G. after initial amplification but prior to the application of any automatic gain control.

Many fetal heart monitors are equipped with such a signal output facility, but it is doubtful if it is widely used. Perhaps this failure of use could be attributed to a false sense of economy in the purchase of additional display equipment. The oscilloscopes which we use for this purpose cost less than £100 each and have been invaluable in detecting poorly applied electrodes, failure of leads, and other artefacts which may lead to false interpretation of the fetal heart rate. We should like to encourage others to make use of these simple display devices.—We are, etc.,

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Correction of Plasma Calcium Measurements

SIR,—Readers may have been surprised to find consecutive papers by Dr. E. M. Berry and others (15 December, p. 640) and by ourselves (p. 643) which recommended that plasma (or serum) calcium values should be adjusted by reference to plasma albumin concentration, but which advocated different equations.

Applications of the adjustment of Dr. Berry and his colleagues to the calcium values of the 200 specimens received for liver function tests which we examined gave a calcium distribution, with 95% limits, of 9·5—10·9 mg/100 ml. After applying our adjustment the 95% limits were 9·0—10·4 mg/100 ml, identical with the limits of our normal range.

What is the reason for the discrepancy? Dr. Berry and his colleagues did not quote their normal range for plasma albumin, but they stated that the mean was 5·0 g/100 ml, and inspection of their fig. 3 shows albumin values in 25 healthy persons before venous occlusion which ranged from 4·1 to 5·6 g/100 ml. The normal range for this laboratory, not taking account of small differences related to sex and age, is considerably lower, with 95% limits of 3·7—4·7 g/100 ml. Our performance over the past six months in the Wellcome Group Quality Control Programme shows that the means of our assay values for albumin and calcium were not significantly different from the overall means of more than 300 participating laboratories. We therefore believe that our adjustment—adjusted calcium = calcium − albumin + 4·0 (where calcium is in mg/100 ml and albumin in g/100 ml) or adjusted calcium = calcium − 0·25 albumin + 1·0 (where calcium is in mmol/l. and albumin in g/l.)—can be applied to data from the majority of laboratories in this country.—We are, etc.,

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Chemotherapy of Disseminated Malignant Tumours

SIR,—We were interested in the letter from Dr. D. A. Cook (12 January, p. 77) in which references was made to our article on the multiple drug therapy of disseminated malignant tumours.[1] Our paper was based on preliminary experience with a new multiple drug schedule for metastatic solid tumours, and the study was initiated in 1969. At that time combination chemotherapy for solid tumours was very much less used than at present, and there was widespread belief that such treatments could be carried out only with unacceptable levels of toxicity to normal tissues. The whole point of our paper was to show that such toxicity could be markedly reduced by the application of new concepts of the cellular basis of cancer chemotherapy. We are pleased to note that Dr. Cook has confirmed this aspect of our study.

With regard to the efficacy of the schedule against specific types of tumour, we were careful to avoid any dogmatic assertions as we did not have sufficient numbers of any type of tumour to constitute a statistically valid sample. We did infer that further studies of breast and bladder carcinomas might be warranted, but Dr. Cook's statement about optimistic results in lung cancer are his words and interpretation of the results, not ours. The response of any small number of lung cancer cases will be biased among other things by the proportion of the oat-cell type of disease. Therefore no conclusion regarding efficacy can be drawn from five cases and, in our paper, none was.

Our initial response rate in bronchogenic carcinoma (3/5) has not been sustained in