Thromboembolism. Four of these patients had normal pulmonary "wedged" pressures (indirect left atrial pressures) and in one there was a slight increase. In the remaining six cases the "wedged" and pressure record could not be analysed because of difficulty in identifying an appropriate waveform. This could have been due to technical imperfection, but an alternative explanation might be pulmonary venous hypertension without left atrial hypertension. The occurrence of pulmonary venous constriction following experimental pulmonary embolism was demonstrated by Daicoff et al.1 This mechanism could account for pulmonary oedema in the absence of left heart failure and might also account for the difficulty in obtaining an adequate pulmonary "wedged" record.

Although in man the mechanism is obscure, there is no doubt that pulmonary oedema can occur as a result of pulmonary thromboembolism.—We are, etc.,

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2 Smith, G. T., Dexter, L. and Dammin, G. J., in Pulmonary Embolic Disease, ed. A. A. Sazabas and M. Stein, p. 120. New York, Grune and Stratton, 1965.

Pathology of Malignant Hyperpyrexia

Stir.—There are two minor inaccuracies which should be corrected in your otherwise excellent leading article (3 February, p. 249).

Firstly, malignant hyperpyrexia was first recognized as a complication of general anaesthesia in the 1930s.1 At that time ether was probably the agent most frequently responsible. Secondly, Bradley and Murchison2 studied muscle biopsy specimens from six patients at risk in two families, not four as stated. An additional meeting of the Royal Society of Medicine on 26 January 1959, where the pathological and biochemical studies performed on malignant hyperpyrexia were discussed in greater detail.—I am, etc.,

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3 Proceedings of the Royal Society of Medicine, 1973, 66, 63.

Thiocyanate Metabolism in Human Vitamin B1 Deficiency

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