The "Rape" group you mention has been set up to explore the best ways of helping rape victims. Any doctor or health worker who has dealt with a rape victim is invited to write for a short questionnaire to 26 Telford Road, London S.E.15.—I am, etc.,

JUDY GILLEY

London N.W.11

¹ Amir, M., *Patterns in Forcible Rape*. Chicago, University of Chicago Press, 1971.

The Short-statured Child

SIR,—The study of causes of short stature by Lacy and Parkin,¹ which was discussed in your recent leading article (9 November, p. 308), indicated that of the children studied few with heights below the third percentile had organic disease. This investigation, undertaken in Newcastle, touches on the wider problem of the significance of short stature in children in relation to their health pattern on reaching adulthood. In a western society, to what extent does short stature in youth, due in part to failure to attain maximum potential of growth, affect the enjoyment of good health in later life?

In this connexion it would be very informative, as stressed elsewhere,² to have long-term information on the two series of children, in Boston⁴ and Iowa,⁵ who were studied several decades ago and whose anthropometric data are used as growth standards. Of these former pupils—now at middle age—among those who were of short stature compared with those who were of tall stature what are now the respective prevalences of obesity, hypertension, diabetes, and ischaemic heart disease? What have been their respective morbidity experiences since youth? Until information of this type is available is it legitimate, as is often done, to emphasize the desirability of children attaining their maximum potential of growth?

What is needed, of course, is follow-up studies of the Framingham type on series of children such that the numbers in the respective percentiles of height are sufficiently large to allow meaningful conclusions to be drawn. It is earnestly hoped that the longitudinal investigation being made at Newcastle will be continued long enough to furnish answers to some of the questions raised.—I am, etc.,

A. R. P. WALKER

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¹ Lacy, K. R., and Parkin, J. M., *Lancet*, 1974, 1, 42.

² *British Medical Journal*, 1974, 4, 308.

³ Walker, A. R. P., and Richardson, B. D., *American Journal of Clinical Nutrition*, 1973, 26, 897.

⁴ Vickers, V. S., and Stuart, H. C., *Journal of Pediatrics*, 1943, 22, 155.

⁵ Jackson, R. L., and Kelly, H. G., *Journal of Pediatrics*, 1945, 27, 215.

Deaths in the Dental Chair

SIR,—Your balanced and admirable leading article (8 February, p. 293) needs one small correction: since October 1973 there have been not five but six deaths. You have omitted the case of the 10-year-old boy in Lancashire (27 April 1974, p. 224) who had

cardiac arrest during recovery from brief inhalational anaesthesia administered by a consultant anaesthetist—a case exactly similar to the Croydon one you report on another page (8 February, p. 342).

You might also have drawn attention to the useful suggestion of Professor P. Bramley and his colleagues (4 May 1974, p. 270) that a list should be initiated of approved dental anaesthetists (doctors and dentists), which would be of immediate help to the dentist who at present gives his own anaesthetics. This use of doctors or dentists, however, would be costly, and both are in short supply. The ultimate solution to the operator-anaesthetist problem, I have suggested,¹ might be to train paramedical anaesthetists for this work.—I am, etc.,

J. G. BOURNE

Salisbury, Wilts

¹ Bourne, J. G., *Lancet*, 1970, 1, 38.

SIR,—I read with interest and approval your leading article (8 February, p. 293). It is gratifying to see you confirming points I have been stressing for many years in my lectures to postgraduates attending courses organized by the Society for the Advancement of Anaesthesia in Dentistry—namely, (1) the need for correct equipment and facilities for treating emergency situations; (2) training of chairside assistants in the care of the unconscious patient; (3) continuous monitoring of (a) the cardiovascular system by means of a pulsemeter and (b) the respiratory system by observations of movements of the reservoir bag on a nitrous oxide/oxygen apparatus; and (4) the avoidance of prolonged surgical anaesthesia by the lone operator/administrator, who should confine his efforts to sedation, tranquillization, and analgesia.

Your plea for the abandonment of general anaesthesia in dentistry except in properly staffed and equipped centres, while admirable, is, I am afraid, a "pipe dream" until the requisite medical and nursing personnel are available. It is a pity that you do not give encouragement to those doctors who are attempting to organize training and certification of dental anaesthetists by apprenticeship.¹

With reference to your medicolegal report on two dental deaths (8 February, p. 341), in the case of the late Mr. Crowther it is impossible to state at which point in the proceedings fatally irreversible damage occurred to cerebral and cardiac tissue or whether it was due to a cumulative effect. But in your report no reference is made to the fact that the ambulancemen while abandoning pulmonary inflation with oxygen (or anything else) and any other form of resuscitation for the slow journey from dental surgery to ambulance along a twisting corridor and down a steep staircase of 23 steps with a U-turn at the top and an S-turn at the bottom, also carried the collapsed patient seated upright in a carrying-chair. The reduction in cerebral oxygenation which would occur during this manoeuvre is obvious.—I am, etc.,

DONALD BLATCHLEY

London W.4

¹ Bramley, P., et al., *British Medical Journal*, 1974, 2, 270.

SIR,—In a short study of anaesthesia in general dental practice in 1970¹ I reached three conclusions:

(1) "That there is an important economic factor in the safety of general anaesthesia in that modern techniques take longer and are more expensive."

(2) "That dentists are at present having to subsidise general anaesthesia in general practice out of other earnings."

(3) "That the present system of payment is a disincentive on the dentist to improve his facilities and apparatus, and gives no encouragement to the employment of skilled or experienced anaesthetists."

These conclusions are still valid today, so here at least is one area where action could be taken. The British Dental Association has dragged its feet in this important matter for too long.—I am, etc.,

E. O. EVANS

Stratford-upon-Avon

¹ Evans, E. O., *Journal of the Royal College of General Practitioners*, 1970, 20, 72.

Malignant Hyperpyrexia

SIR,—The implication in your leading article (30 November, p. 488) that nitrous oxide is yet another anaesthetic drug which may trigger the malignant hyperpyrexia (M.H.) response and the apparently successful treatment of one such case with dexamethasone¹ neither advance the understanding of the pathogenesis of this syndrome nor provide a rational basis for treatment. It is, moreover, unfortunate that undue emphasis is laid upon the temperature rise when defining the syndrome. This is a symptom not necessarily directly proportional to the rapidly developing acidosis, hypercarbia, hyperkalaemia, and tachycardia which are characteristic of M.H. and which can be readily detected.

Investigations into porcine M.H. have provided the only information from controlled and systematically conducted studies,^{2,3} which are not usually possible in man for ethical reasons and also because of the sporadic and quite unexpected occurrence of cases. It is certainly justifiable to exercise caution when transposing data from one species to another, but to dismiss studies on pigs on the basis of Britt et al.'s⁴ somewhat tenuous evidence is neither reasonable nor valid. Defects of the calcium-accumulating ability of the sarcoplasmic reticulum or even of mitochondria suggest attractive explanations for the aetiology of M.H. in both people and pigs. However, if sufficient care is taken in the preparation of these subcellular organelles to prevent the extensive protein denaturation that occurs when muscle is allowed to develop rigor at temperatures greater than 30°C, or if these effects are taken into consideration, the apparent loss of functional capacity of the sarcoplasmic reticulum and the mitochondria can be explained.⁵⁻⁷ In M.H.-susceptible pigs the stimulation of any biopsy technique produces intense muscle contraction with a rapid acceleration of glycolysis and a fall in pH of the sample to that of muscle in rigor. Moulds and Denborough⁸ have also reported the extreme sensitivity of susceptible human muscle to a wide variety of unrelated stimuli, presumably with similar biochemical effects. On these grounds it is likely that