report on aids for the disabled[1] said, "In theory wheel chairs are prescribed by consultants, but in practice few consultants have studied this problem adequately and the responsibility is often passed on to social workers or appliance clerks who have not been trained to do this work. Part of the fault stems from gaps in the doctor's education, particularly at a postgraduate level, but the situation could be improved by delegating responsibility to physiotherapists or occupational therapists specially trained to assess patients and their wheel-chair needs." The B.M.A. working party estimated that possibly as high as 30% of wheel chairs were unsuitable for the patient concerned. This may well be an underestimate.—We are, etc.,

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REFERENCE

1 British Medical Association Planning Unit. *Report of the Working Party on Aids for the Disabled.* London, British Medical Association, 1968.

Hyperpyrexia during Anaesthesia

SIR,—Malignant hyperpyrexia has now been classified into two groups: a rigid type and a non-rigid type.[1] Malignant hyperpyrexia with rigidity should be easy to identify by the rigidity itself, serum muscle enzyme studies (during and after the event), and muscle biopsy.[2 3]

On the other hand, malignant hyperpyrexia without rigidity is virtually a diagnosis of exclusion, since this group of patients have normal serum muscle enzyme levels and a normal muscle biopsy.[3] How then is non-rigid malignant hyperpyrexia to be distinguished from other pyrexial reactions during anaesthesia? The non-rigid hyperpyrexia-prone patients apparently fail to produce a neuromuscular block when given intra-arterial suxamethonium.[3] This is the only differentiating test and requires voluntary co-operation by patients, which may not always be forthcoming. Since no simple test is available, a more critical approach should be made before classifying a pyrexial reaction as non-rigid malignant hyperpyrexia, or this group will be in danger of becoming a dustbin for all types of pyrexial reactions that occur during anaesthesia. Where there is an alternative explanation for a pyrexia occurring during anaesthesia a diagnosis of malignant hyperpyrexia should not be made.

Drs. Sheila Kenny and H. Rolfe (21 November, p. 492) report a case of pyrexia during anaesthesia, which they classified as non-rigid hyperpyrexia when there might equally have been an alternative explanation. Their patient already had a preoperative pyrexia, with presumably some peritonitis. The temperature may have started to rise between when it was last recorded and the beginning of the operation, especially if atropine or scopolamine premedication were given. The temperature would have risen further when heat loss was reduced by covering the patient in surgical drapes. The situation reminds one of the so-called "ether convulsions" which occurred in preoperatively pyrexial children with peritonitis. How easy was it to cool the patient—how much iced fluid was given? The fulminating rise of temperature in malignant hyperpyrexia is not usually easy to reverse. Since a diagnosis is being made by exclusion, the results of blood culture and/or peritoneal swab culture should be taken into account. Did the patient receive any antibiotic therapy after surgery? Both antibiotics and closing of the perforation might have been sufficient to account for the return of temperature to normal on the next day. The metabolic acidosis could be a consequence of the rise in temperature (and metabolic rate) during anaesthesia. While this may yet be a case of non-rigid malignant hyperpyrexia, it is not conclusively proved if an alternative explanation cannot be excluded.

All anaesthetists are now aware of the danger of malignant hyperpyrexia, and should treat any pyrexia occurring during anaesthesia expectantly. However, because they do this, it does not mean that all pyrexias are due to malignant hyperpyrexia.—I am, etc.,

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1 Kalow, W., Britt, B. A., Terreau, M. E., and Haist, C., 1970, *Lancet*, **2**, 895.
2 Denborough, M. A., Forster, J. F. A., Hudson, M. C., Carter, N. G., and Zapf, P., *Lancet*, 1970, **1**, 1137.
3 Britt, B. A., Symposium on Metabolic Effects of Anaesthesia at Royal College of Surgeons, England, September, 1970.

Rational Oxygen Therapy

SIR,—Dr. J. M. Leigh makes the aceptable plea that the dosage of oxygen during oxygen therapy should be better controlled (5 December, p. 620). May I add that in addition to using high air flow with oxygen enrichment, using the Ventimask series, the use on the wards of a paramagnetic oxygen analyser provides a very practical method of monitoring the inspired oxygen during therapy with patients breathing spontaneously, and makes minute to minute adjustment easier.

The method is flexible as it can be used in most circumstances when controlled oxygen dosage is of importance. If sampled via a fine nasopharyngeal catheter breath-by-breath concentrations can be measured.

Oxygen analysers of the paramagnetic type are inexpensive compared with equipment required to measure the blood oxygen tension. They are suitable for use by nursing staff and the readings can be recorded with those of the usual parameters.—I am, etc.,

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Low Birth Weight

SIR,—Your leading article (19 September, p. 657) was of interest and in particular the reference[1] to survival and subsequent development of infants weighing between 850 and 1,250 g. at birth. Results from this hospital on infants of similar birth weight distribution may be of interest.

During the four-year period 1966-9 65 infants weighing between 850 and 1,250 g. at birth were liveborn, of whom 32 (49%) survived. Subsequent follow-up—to one year in every case—showed evidence of permanent brain damage in 6 (18%) and a further three were considered suspect. During the same four years there were 19,844 livebirths, of whom 1,288 (6·5%) weighed 2,500 g. or less at birth. Neonatal death in the first week occurred in 134 (10·5%).—We are, etc.,

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REFERENCE

1 Vapaavuori, E. K., and Räihä, N. C. R., *Acta Paediatrica Scandinavica*, 1970, **59**, 353.

Facial Paralysis after Local Dental Anaesthesia

SIR,—In their report on this very rare complication of local anaesthesia Squadron Leader I. B. Tiwari and Flight Lieutenant T. Keane (28 March, p. 798) list many causes of facial palsy. We report a further case, one of three seen this year in which facial paralysis followed local anaesthesia, because aetiological factors other than those they mention may have been responsible.

A woman aged 25 years gave a life-long history of eczema, hay fever, and asthma. Three weeks before the relevant dental treatment she was unwell and enlarged painless glands appeared in both sides of her neck which persisted for five days. One hour after injection of local anaesthetic around a left lower premolar tooth there was a rapid onset of partial bilateral ptosis, diplopia, and vertigo. The ptosis cleared within two days, but defective conjugate movement of the eyes in all directions with fine nystagmus on lateral gaze persisted. She lost her visual symptoms after four days, when a complete right lower motor neurone facial palsy, without ageusia, and severe rombergism supervened. These signs rapidly responded to treatment with prednisolone. Two weeks after the original injection she developed complete anaesthesia of the 2nd and 3rd divisions of the right trigeminal nerve. Again the response to prednisolone was swift, the sensory symptoms abating in two days. No haematological abnormality was found and a full neurological investigation, which included a lumbar puncture, was negative. Three weeks later she was completely cured.

Obscure neurological syndromes may be encountered in 1% of cases with infectious mononucleosis, and when an isolated cranial nerve is involved differential diagnosis may be difficult. In a recent report[1] a patient with infectious mononucleosis appeared to be developing a posterior fossa tumour because the 5th, 7th, and 8th nerves and later the cerebellum progressively became involved, but this neurological condition was cured with the administration of steroids.

Our patient developed after dental anaesthesia lesions within the mid-brain and later the pons which rapidly responded to steroid therapy. She had had some painless glandular enlargement in her neck earlier, and though we could not prove the diagnosis we suspected that this was infectious mononucleosis. However, in view of her long history of various forms of allergic response, flitting lesions in the brain stem of an angioneurotic oedematous type are a more likely explanation for her very dis-