medical, educational, nursing, social work, and (note it is placed last) parental aspects of child health. This is to be done by tossing the fate of the unfortunate child into the arena of a multiprofessional team and, to quote some of the intentions:

"Child health visitors to have the right to apply for legally enforceable medical examinations."

"A doctor undertaking developmental surveillance should also be able to treat or refer to a specialist."

"A school doctor to have the right to examine a child if necessary without parental consent."

"Head teachers should be able to request examinations."

"Children under 16 could have advice and treatment, if necessary, without parental knowledge."

"Health records should be available to the Educational Authority."

It seems that not only does this strike at the very roots of the relationship of the family doctor with his patient but also at the concept of family unity. Furthermore, no one suggests that the general practitioner should cease to be held responsible for his patients' health and consequently be available for any litigation directed at him, but at the same time any Tom, Dick, or Harry wearing a welfare hat should have the right to examine, treat, or refer his patient.

One can only hope that this unwieldy child will be a stillborn casualty of the inevitable strictures that the NHS is about to face.

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¹ Committee on Child Health Services, *Fit for the Future*. London, HMSO, 1976.

Pentazocine addiction: a warning

SIR,—Pentazocine (Fortral) is generally regarded as a potent, non-addictive analgesic, and is exempt from international narcotics legislation.¹ Nevertheless, drug dependence has been described; by 1971 130 reports of alleged dependence to oral as well as parenteral pentazocine had been received from world-wide sources.^{2,3}

The following case history illustrates the development of physical drug dependence and serious withdrawal syndrome.

A male physician aged 50 years suffered severe recurrent abdominal pain of undetermined aetiology. Pentazocine was prescribed in 1971, orally in milder episodes and parenterally in severe attacks, accompanied by persistent vomiting. A slight euphoric effect and increased tolerance was noted. In November 1976 endoscopy and endoscopic retrograde cholangiopancreatography revealed duodenal ulceration only; truncal vagotomy and pyloroplasty were performed.

During the patient's stay in hospital pentazocine was administered parenterally in a dosage of 240–300 mg/24 h. The drug was stopped abruptly two weeks after operation; nausea, vomiting, abdominal cramps, drenching sweats, low-grade fever, feeling cold, yawning, depression, nocturnal confusion, tremors, motor inco-ordination, and hypertension developed immediately. These symptoms and signs were relieved by further pentazocine given parenterally until it was realised that the syndrome was one of physical dependence and withdrawal. Amitriptyline and sulphuride were prescribed and the dose of pentazocine was gradually reduced over two weeks; as the injection sites became bruised and indurated⁴ oral pentazocine (75 mg/24 h) was substituted for one more week. During the withdrawal period the patient had marked anorexia and lost 10 kg in weight. The feeling of cold, wretchedness, and abdominal cramps were the most lasting symptoms⁵; the hypertension settled after a further fortnight.

We would like to emphasise the necessity for caution by physicians in prescribing pentazocine for chronic pain, especially as so much emphasis has been placed on the non-addictive character of the drug.

Thanks are due to Sterling Drug International (HK) for immediately supplying us with information on pentazocine.

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¹ WHO Expert Committee on Drug Dependence, *Seventeenth Report*, WHO Technical Report Series No 437, p 24. WHO, Geneva, 1970.

² Alarcon, R D, Gelfond, S D, and Alarcon, G S, *Johns Hopkins Medical Journal*, 1971, **129**, 311.

³ Hart, R H, *Lancet*, 1969, **2**, 690.

⁴ Parks, D L, Perry, H O, and Muller, S A, *Archives of Dermatology*, 1971, **104**, 235.

⁵ Schoolar, J C, Idänpää-Heikkilä, P, and Keats, A S, *Lancet*, 1969, **1**, 1263.

Surgery for Menière's disease

SIR,—Your leading article (15 January, p 124) emphasises, we believe correctly, the low proportion of patients with this disease who have severe symptoms and sounds a note of caution regarding the place and effectiveness of the various forms of surgery. In a situation in which the symptoms are episodic, fluctuate in severity, and can resolve spontaneously, however, it is imperative if surgery is being proposed that controlled trials be performed. None has yet been reported; indeed, even prospective series with reliable follow-up are extremely rare. The scientific evidence supporting surgery is thus tenuous and your article might benefit from additional comments which are made under three headings.

(1) *Placebo operations*—The statement "the grommet drain works" is scientifically unproved. Even if this were subsequently shown to be true the metabolic effects of anaesthesia and otological trauma would have to be considered. It is more likely that the improvements that have been reported are due to the placebo effect of surgery.

(2) *Curative operations*—Under this heading are endolymphatic sac decompression and shunt operations, which are presumably performed in the belief that the symptoms are caused by increased pressure in the endolymphatic system. There is no direct evidence to support this, and, although histologically there is bowing of Reissner's membrane,¹ there are no reports of dilatation of the endolymphatic duct or sac. There has been a suggestion from biopsies that there is increased fibrosis of the sac,² but a preliminary study of temporal bones with endolymphatic hydrops has shown no gross histological difference between these and normal bones.³ In addition, as it is impossible to create a fistula or shunt in the endolymphatic system in animals because of regeneration,⁴ it is unlikely that in man microshunts function for any length of time. Though histology does not necessarily reflect function, the rationale for surgery of the endolymphatic sac must at present be considered unfounded and reported improvements are just as likely to be due to the placebo effect of surgery.

(3) *Destructive operations*—Labyrinthine vertigo can be eliminated by destroying the end organ, and labyrinthectomy, though not mentioned in your article, is the operation of choice if the hearing loss is minimal as it can be performed with minimum morbidity under local anaesthesia via the oval window. When

some hearing remains there is a natural desire to maintain this, especially in a disease that has a bilateral tendency, and it becomes a decision whether the risks of a neurosurgical procedure such as division of the vestibular nerve are justified. Whether the hearing is actually preserved is not fully documented, and, although the mean bone conduction in Smyth *et al*'s⁵ 18 patients was unchanged after surgery, of the three patients whose hearing was within the normal range before, only in one did it remain so after surgery.

Considering then the likely natural course of Menière's disease, the medical profession should insist that proponents of surgical operations perform randomised trials with unoperated controls before their efficacy is considered and even then, because of the high degree of surgical skill necessary for this type of surgery, their widespread adoption could not be recommended.

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¹ Schuknecht, H F, *Pathology of the Ear*. Cambridge, Harvard University Press, 1974.

² Plantenga, J F, and Browning, G G, in preparation.

³ Arenberg, K I, Marovitz, W F, and Shambaugh, G E, *Acta Otolaryngologica*, 1970, suppl 275.

⁴ Kimura, R S, and Schuknecht, H F, *Annals of Otolaryngology and Rhinology*, 1975, **84**, 1.

⁵ Smyth, G D L, Kerr, A G, and Gordon, D S, *Journal of Laryngology and Otolaryngology*, 1976, **90**, 823.

Child health and environmental lead

SIR,—Your leading article on this subject (29 January, p 255) referred to the paper by Beattie *et al*,¹ of which we were co-authors, and stated that "the severity of mental handicap seemed to be out of proportion to the measured blood lead levels." We wish to make it quite clear that no such relationship was or, indeed, could be claimed by us. The principal point of our study was that an excess of cases of mental retardation was found where lead concentrations of drinking water greater than 800 µg/l at first flush occurred in homes occupied by children in the first year of their lives and in the houses occupied by their mothers during pregnancy. This hypothesis is not unreasonable since drinking water of such high lead concentrations has been found to be associated with evidence of frank lead poisoning or toxicity in adults.^{2–5} All but three of the children investigated had been given dried milk in infancy, which means that they were drinking a volume of tap water much greater in proportion to their size than would occur in the adult. Furthermore, there is ample evidence that a significantly greater absorption of lead occurs in the infant and young child than in the adult. The blood lead concentrations in these children were measured several years after the critical period of exposure—namely, before and during the first year of life—and thus could not possibly be correlated with the severity of mental handicap.

We agree with you that there is insufficient evidence to incriminate air pollution of lead with disease in childhood, but the levels of water lead to which our study referred—namely, 800 µg/l—are greatly in excess of the WHO requirements (300 µg/l for first flush). The danger of the conclusion of your article is that it may discourage the laudable efforts to eradicate such high levels of lead in drinking water which are present in some parts of this country. We feel that such high levels are unacceptable, particularly when there are