facilitate synchronization with the mechanical ventilator. Clinical evidence supports the use of opiates in such a situation.[4 5]—I am, etc.,

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1 Earle, B. V., *Thorax*, 1953, **8**, 195.
2 Samter, M., and Beers, R. F., Jun. *Annals of Internal Medicine*, 1968, **68**, 975.
3 Marchand, P., and Hasselt, H., *Lancet*, 1966, **1**, 227.
4 Pontopiddan, H., Geffin, B., and Lowenstein, E., *New England Journal of Medicine*, 1972 **287**, 743.
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Corticosteroids in Neonatal Hepatitis

SIR,—In January 1972 we extended a study of aetiological, epidemiological, and prognostic factors in the neonatal hepatitis syndrome in the South-east Metropolitan Hospital Board area to include a controlled trial of the effect of corticosteroids in this disorder. This trial was undertaken because corticosteroids are recommended[1] and used by some authors although their value has been disputed and the hazards of their use are well known.

We elected to consider separately neonatal hepatitis in infants with genetic deficiency of serum α_1-antitrypsin since it had been reported that cirrhosis was the almost inevitable outcome,[2] and in three of five such infants seen in the first year of our study this had occurred by the age of 12 months.[3] A more recent report of neonatal cholestasis in five α_1-antitrypsin-deficient infants reinforces the poor prognosis in these subjects.[4]

The pathogenesis of the liver disease in this deficiency state is not understood but it has been postulated that the uninhibited action of proteases released from microorganisms, leucocytes, or parenchymal cells in response to intercurrent infection or toxins may be important in causing continuing tissue damage. If this is so, the anti-inflammatory effect of corticosteroids may be helpful, particularly if given early and for a prolonged period. A placebo group is considered ethically justified as some cases apparently do well without corticosteroids.

Since the incidence of neonatal hepatitis syndrome in α_1-antitrypsin-deficient subjects in the United Kingdom is estimated to be about 1 per 12,000-20,000 live births, we should like to extend the present trial to other parts of the country so that a significant conclusion can be reached in a reasonable period of time. May we therefore appeal to paediatricians, when they see such children, to contact us as soon as the diagnosis is considered so that the true effect of early and prolonged corticosteroid therapy can be assessed in this rare but often severe disorder?—We are, etc.,

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1 Kaye, R., in *Textbook of Pediatrics*, ed. W. E. Nelson, V. C. Vaughan, and R. J. McKay, 9th edn., p. 834. Philadelphia, Saunders, 1969.
2 Sharp, H. L., *Hospital Practice*, 1971, **6**, 83.
3 Porter, C. A., *et al.*, *British Medical Journal*, 1972, **3**, 435.
4 Aagenaes, O., Matlary, A., Elgjo, K., Munthe, E., and Fagerhol, M., *Acta Paediatrica Scandinavica*, 1972, **61**, 632.

The Old and the Cold

SIR,—In a recent article Dr. R. H. Fox and others (6 January, p. 21) reported an association between environmental temperature and body temperature in old people, which adds to similar findings reported previously by other authors. They conclude that low environmental temperature could place the elderly at risk of developing hypothermia. We consider that the number of people at risk could be very much greater than is implied by the number of deaths coded as due to hypothermia in the Registrar General's statistical review[1] and that there is an important link between this environmental/body temperature association and a recent paper by Stitt *et al.*[2]

These workers investigated differences in certain clinical and biochemical indicators of cardiovascular disease between men living in towns of high and low mortality from cardiovascular disease. The five measurements in which small significant differences were observed were heart rate, diastolic blood pressure, plasma cholesterol, skinfold thickness, and vital capacity Gardner *et al.*[3] have attributed the differences in cardiovascular mortality between towns to differences in hardness of drinking water supplies, and the inference in Stitt's paper is that the differences observed in the five measurements listed above could indicate a mechanism by which water hardness affects cardiovascular disease. However the two features (E.C.G. changes and angina/chest pain) most widely regarded as indicative of coronary heart diseases showed no significant differences between the two groups.

An alternative explanation which we find more attractive is that the intertown variations in ischaemic heart disease mortality are highly associated with climatic factors—temperatures and rainfall.[4] The same mechanism explains very satisfactorily the marked seasonal variation in death rate from ischaemic heart disease.[5] On our interpretation the findings of Stitt *et al.* would be that among men in colder, wetter towns there is a small but significant physiological adjustment to the climate—increased heart rate, increased diastolic pressure, and greater skinfold thickness with an accompanying higher plasma cholesterol. That Elwood *et al.*[6] found no significant differences in these respects in a study of 600 men from neighbouring areas of very different water hardness but similar climate lends support to our hypothesis. We consider that low body temperature places the elderly at risk not only to hypothermia but also to death from myocardial infarction. We await the results of Dr. Fox's study to see whether the geographical distribution of low body temperature in the elderly is similar to that of high mortality from ischaemic heart disease.—We are, etc.,

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1 *Registrar General's Statistical Review of England and Wales, 1970*, part 1, tables, medical. H.M.S.O. London (1972).
2 Stitt, F. W., Clayton, D. G., Crawford, M. D., and Morris J. N., *Lancet*, 1973, **1**, 122.
3 Gardner, M. J., Crawford, M. D., and Morris, J. N., *British Journal of Preventive and Social Medicine*, 1969, **23**, 133.
4 West, R. R., Lloyd, S., and Roberts, C. J., *British Journal of Preventive and Social Medicine* 1973, **27**, 36.
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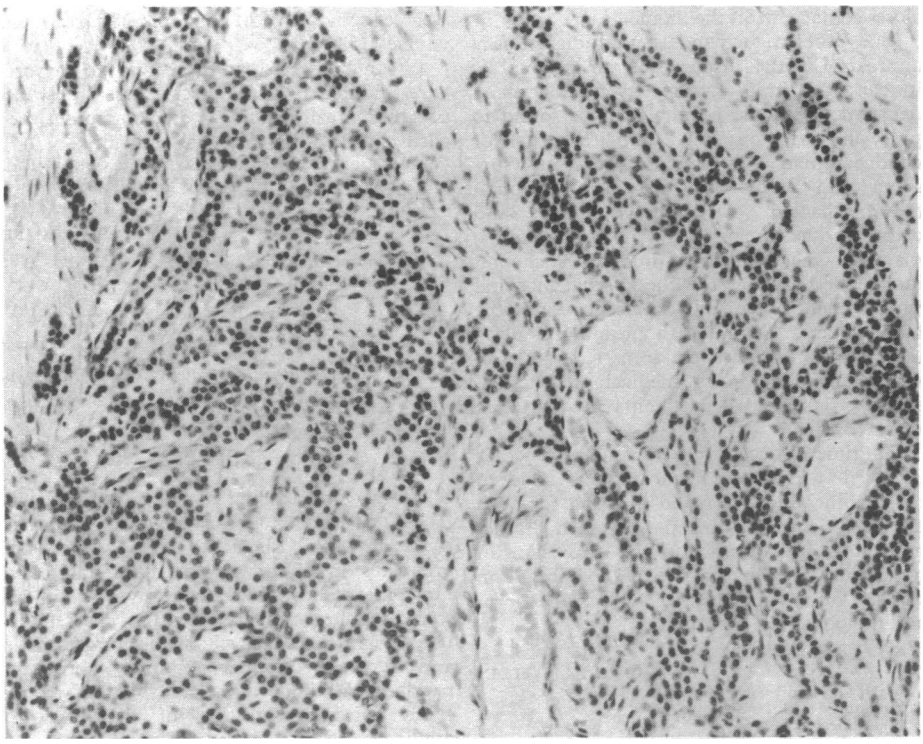
Glomus Tumours

SIR,—It is for me an ironical coincidence that your leading article on glomus tumours (10 March, p. 565), with its prognostic comment that these have "no malignant potentiality," should appear in the week when a colleague—in another country—died from what may reasonably be assumed to have been metastatic spread of a glomus tumour. He had himself diagnosed the nature of the original tumour on the grounds of its characteristic presentation with severe paroxysmal pain that he could locate precisely to one point in the skin, on the dorsum of a foot close to the ankle. The tumour, 0·4 cm in diameter, was found at that point. Histologically, it differed in no way that I could recognize from any other glomus tumour. There was no recurrence at its site, but a year later the patient noticed the lymph nodes in the groin of the same side to be enlarging and he had them excised. The nodes were extensively infiltrated by tumour cells that were indistinguishable from the glomus cells of the primary tumour. These cells might have been mistaken for those of a somewhat atypical lymphosarcoma but for their occasional arrangement in a manner that exactly reproduced the familiar histological pattern of the common form of glomus tumour, particularly in their relation to blood vessels. There was already *x*-ray evidence of secondary deposits in the lungs. The patient died a year and nine months later, after a short final illness that began with focal epilepsy and signs of rapidly rising intracranial pressure, presumably caused by metastatic growth of the tumour. There was no necropsy.

If the diagnosis in this case is thought not to be sufficiently substantiated by the findings in the lymph nodes, it may be argued that the deposits in the nodes, lungs, and brain could have originated in the coincidental presence of an unsuspected primary cancer elsewhere. That is as may be. But in rare other cases there has been unequivocal evidence that glomus tumours may metastasize. The accompanying photomicrograph is from such a case. The patient was a woman of 66 and the initial growth, in the skin of the thigh, had been present for at least five years before excision, the patient having previously rejected medical help. The tumour was solitary, some 7 cm in its longest dimension, and superficially ulcerated. It had invaded the deep fascia and underlying muscle over much of its extent. The patient died two years later in a cachectic state, with secondary deposits in the regional and abdominal lymph nodes and in the lungs, brain, liver, and vertebral bodies.

Professor Zilton A. Andrade, of the Federal University of Bahia, in Salvador, Brazil, showed me preparations last year from another unequivocally malignant glomus tumour. Again, an infiltrating glomus tumour, assuredly malignant, was reported in the *B.M.J.* last year,[1] and there are similar cases in the literature, including one referred to by Professor Willis on the page of his book cited in your leading article.[2] Such observations make it clear that there is cer-



Woman aged 66. Metastatic glomus tumour in hilum of a juxta-aortic lymph node. The tumour cells in infiltrating the tissue have oriented themselves to the blood vessels in a manner immediately reminiscent of the relation between glomus cells and vasculature in normal glomera and in glomus tumours (glomangiomas) as they are ordinarily seen. Although not well seen at this magnification, the tumour cells have the features of glomus cells, with rather small deeply stained nucleus and distinct but relatively narrow cytoplasmic rim. (Haematoxylin-eosin. × 160.)

tainly no justification for categorizing all glomus tumours as hamartomatous as, regrettably, I must admit to having done elsewhere.[3]

The interpretation of the histological picture in these cases is admittedly debatable, if not controversial. Nevertheless, it cannot be maintained that they are all misinterpreted instances of "haemangiopericytoma," Kaposi's sarcoma, or other accepted varieties of angiosarcoma, or of lymphosarcoma or secondary carcinoma, though I have heard such explanations put forward by specialists in diagnostic tumour pathology. On the other hand it may be remarked, without malice but echoing the timely admonition in your leading article, that the cause of those who would shed light on these problems is not helped by the runaway enthusiasts who support their contention of the occasional malignancy of glomus tumours by citing published references to malignant examples of chemodectomas, some of which arise from the glomus jugulare and other "glomera" that have nothing whatever to do with the totally different glomera in the skin and some other situations that are the origin of the tumours generally known as "glomus tumours." I would stress that these strictures have no direct bearing on your leading article; it is with its contention that a glomus tumour is necessarily a benign tumour that I take issue.—I am, etc.,

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1 Lumley, J. S. P., and Stansfeld, A. G., *British Medical Journal*, 1972, 1, 484.
2 Willis, R. A., *Pathology of Tumours*, 4th edn., p. 651. London, Butterworths, 1967.
3 Symmers, W. St. C., in *Systemic Pathology*, ed. G. Payling Wright and W. St. C. Symmers, vol. 2, p. 1561. London, Longmans, 1966.

Latent Morbidity after Abortion

SIR,—Dr. D. M. Potts (24 March, p. 739) states that the Karman catheter is becoming popular in many centres and quotes figures published in 1971 by Beric and Kupresanin[1] from Yugoslavia. Dr. Potts has overlooked the paper by these authors, together with Dr. J. F. Hulka, published in September 1972,[2] in which they describe their clinical trials of the Karman catheter. In this paper they say that to the best of their knowledge no previous results of clinical trials of this catheter had been published. The trials described covered 322 patients. After using the Karman catheter retained products of conception were found in 12% of all pregnancies of six weeks' gestation, 47% at seven weeks, 85% at eight weeks, and 100% of pregnancies of over eight weeks' gestation. The authors concluded: "We felt the catheter offered the advantage of no dilatation and anesthesia for most early pregnancies but was not suitable for terminating pregnancies beyond the sixth week of gestation."

We are grateful to Dr. Potts for drawing our attention to a recent Hungarian paper. May we draw his attention to the long series of papers on the sequelae of induced abortion in *Československá Gynekologie*, including many data on first- and second-trimester spontaneous abortions, prematurity, and sterility following previous terminations, and to the report in that journal[3] of a national conference on the sequelae of abortion? There is a similar series of papers in a journal of the Romanian Academy of Sciences, *Obstetrica şi Ginecologia*. It has never been explained clearly to English readers just why, after liberalizing abortion for some years, the Romanians decided to reimpose heavy legal restrictions. The assumption that this change of policy was made only on demographic grounds much underestimates the influence of the Romanian medical profession. It is now proposed in Romania that a previous "curettage" should be one of the main factors to be given a numerical value in computing a risk index for pregnancy to help antenatal care.[4] It was one of our recommendations to the Lane Committee that this substantial literature should be reviewed and we explained in our preface that we had covered only a fraction of what is available.

We disagree with Dr. Potts on one point. He expresses concern at the distress caused to women who have had an abortion by knowledge of the possible sequelae. If a woman who has had an abortion conceals this fact from her obstetrician in a subsequent pregnancy she reduces her chances of a successful outcome. She may best be persuaded to confide in him by being made aware of the risks. Knowledge of the less fortunate consequences of our actions is part of the substance of health education; it may be painful, like the knowledge of the morbid consequences of smoking to smokers. The spread of such knowledge is an essential part of primary prevention. We consider that knowledge about the latent morbidty that follows induced abortion is an important part of the case for the responsible use of contraception and of education for parenthood.—We are, etc.,

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1 Beric, B. M., and Kupresanin, M., *Lancet*, 1971, 2, 619.
2 Beric, B. M., Kupresanin, M., and Hullal, J. F., *American Journal of Obstetrics and Gynecology*, 1972, **114**, 273.
3 *Československá Gynekologie*, 1970, **35**, 325.
4 Sirbu, P., *Obstetrica şi Ginecologia*, 1972, **19**, 743.

SIR,—It may be worth pointing out that the Yugoslavian statistics cited by Dr. D. M. Potts (24 March, p. 739) do not in fact provide any unequivocal support for his conclusion that outpatient abortion under paracervical block does not increase the prematurity rate for subsequent pregnancies, in contrast with the situation in Hungary, where increased prematurity after abortions induced by dilatation and curettage under general anaesthesia is well attested.

What Dr. Potts shows is that legal abortions in the Novi Sad hospital increased from 4,580 in 1960 to 6,445 in 1970 and that there was no significant increase in the prematurity rate for deliveries over the same period. But the figures as they stand give no information about the induced abortion rates for mothers later delivered of live babies. Has Dr. Potts any evidence that these increased over the 10 years? If so, then his conclusions about prematurity and ectopic pregnancy rates may be justified —but otherwise not.—I am, etc.,

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Treatment of Spina Bifida Cystica

SIR,—In your recent leading article (10 March, p. 565) you state that "the problem of what treatment should be offered was pre-