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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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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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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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-