On Mount Kenya this year I witnessed high-altitude oedema presenting as coma. The patient, a 25-year-old European, had walked from 6,500 feet (1,980m) to 15,500 feet (4,724 m) altitude in a day and a half—he had proved to be the fittest member of the party. On the next day he developed headache and nausea, causing him to rest. He was rational till night. During that night he had loud but not alarming breathing. Next morning he was unrousable, with temperature of 38·5°C, tachycardia, tachypnoea, and frothy saliva. Cyanosis was not present at any stage of the illness. After first-aid treatment (parenteral digoxin, frusemide, chlorpromazine, and pencillin, with oxygen) he was evacuated by stretcher and helicopter. Chest *x*-ray showed pulmonary oedema. His fundi later showed retinal haemorrhages. C.S.F. pressure was high. After a week of treatment including positive-pressure ventilation, diuretics, dexamethasone, and antibiotics he recovered consciousness. A spastic quadriplegia gradually resolved, leading to complete recovery after about two months.

The mechanism of the cerebral "oedema" is uncertain. It is clearly not just due to hypoxia. Cerebral arteriolar constriction (due to hypocarbia), with or without diffuse thrombosis or haemorrhage, must be a partial explanation.

My thanks go to Dr. Ponte of the Consolata Mission, Nyeri, and Dr. Henderson of the Nairobi Hospital for permission to describe the care with which they provided the definitive medical treatment; also to Mr. Woodley, Warden of Mount Kenya and Aberdare National Parks, for the rescue and much information.

—I am, etc.,

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1 Singh, I., Kapila, C. C., Khanna, P. K., Nanda, R. B., and Rao, B. D. J., *Lancet*, 1965, **1**, 229.
2 Steele, P. R., *Lancet*, 1971, **2**, 32.
3 *British Medical Journal*, 1972, **3** 65.
4 Steele, P. R., *British Medical Journal*, 1972, **3**, 231.
5 Foster, J. C., *British Medical Journal*, 1972, **3**, 232.
6 Pines, A., *British Medical Journal*, 1972, **3**, 232.

Medical Schools in Italy

SIR,—With reference to the report on "Medical Research in Europe" (24 March, page 733) I should like to amend two figures referring to Italian medical schools. The update, figure for the number of medical schools in Italy is 27, and, much more relevant to a correct picture of our current situation, the number of medical students has reached 120,000 in 1973. With only some 6,000 teachers (1,000 professors and 5,000 lecturers) it is easy to imagine how happy and efficient the teaching may be.—I am, etc.,

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Spontaneous Periodic Hypothermia: Diencephalic Epilepsy

SIR,—With reference to the article by Dr. R. H. Fox and others (23 June, p. 693), I would like to express my admiration for the extremely thorough investigation in this unusual condition.

It is a pity however, that, among all the investigations carried out, the most important one of all was not illustrated. I refer to the report of the abnormal electroencephalogram. Surely a short page of a typical discharge, as described, would have added a great deal more to the paper's value? All those who are involved in interpretation of EEGs realize the fallacies of relying on a verbal report. Furthermore, the paper raises the question of what is epilepsy, and though I searched diligently, I found nothing in the discussion to substantiate the basic concept as stated in the title.—I am, etc.,

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Sick Sinus Syndrome

SIR,—We agree with your leading article (23 June, p. 677) that the sick sinus syndrome is of clinical importance and that it is commoner than generally realized. However, we were surprised that the one clear recommendation for therapy was for prophylactic anticoagulants. No reference was given for the alleged high incidence of systemic embolism in patients with the sick sinus syndrome, though cerebral embolism was inferred by Samarasinghe and Senanyake[1] in two cases of the syndrome and Rubenstein *et al.*[7] recorded systemic embolism in eight out of 33 patients. Our own experience is different; indeed, no instances of embolism were seen in the 46 patients we described in 1971,[3] nor have any occurred during subsequent follow-up. We would advise against the wholesale adoption of anticoagulant therapy in patients with the sick sinus syndrome on the present data.

We were disappointed that though the basic pathology of the sick sinus syndrome was described authoritatively as "usually degenerative change in the sino-atrial node (and other specialized conduction tissue)," again no relevant source was quoted. We have had difficulty in finding detailed pathological descriptions of the sino-atrial node in the literature and would have welcomed further references.

We would agree that the time has come for a more anatomical title than "sick sinus syndrome" and, though we were guilty of adopting the term "lazy sinus syndrome" some years ago,[4 5] the term "sinu-atrial disorder" suggested by Oram *et al*[6] would seem to be the best available.—We are, etc.,

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1 Samarasinghe, H. H. R., and Senanayake, N., *British Heart Journal*, 1973, **35**, 503.
2 Rubenstein, J. J., Schulman, C. L., Yurchak, P. M., and DeSanctis, R. W., *Circulation*, 1972, **46**, 5.
3 Eraut, C. D., and Shaw, D. B. *British Heart Journal*, 1971, **33**, 742.
4 Shaw, D. B., and Eraut, C. D., *British Heart Journal*, 1970, **32**, 557.
5 Shaw, D. B., and Eraut, C. D., *Bristol Medico-Chirurgical Journal*, 1969, **84**, 213.
6 Lloyd-Mostyn, R. H. Kidner, P. H., and Oram, S., *Quarterly Journal of Medicine*, 1973, **42**, 41.

Grades of Hypothyroidism

SIR,—We have read the recent article by Dr. D. C. Evered and his colleagues (17 March, p. 657) with great interest. The key group of patients in the series described was that said by the authors to be suffering from "subclinical hypothyroidism." Most of these patients had circulating antibodies to thyroid tissue and were distinguished from other patients with antibodies solely on the basis of a raised serum thyroid-stimulating hormone (TSH) concentration. Nowhere in their paper do the authors give the absolute upper limit of serum TSH concentration measured by their assay in the normal population. Without this information patients with "subclinical hypothyroidism" cannot, by definition, be categorized. In the discussion reference is made to an earlier paper[1] in which "the range of serum TSH concentrations in normal subjects has been defined." In fact a range is not given in the paper to which reference is made but only the arithmetic mean values for two relatively small groups of men and women. The "95% confidence limits" (sic) for these means are given but they have been calculated on the incorrect assumption that the data are normally distributed. Inspection of fig. 6 of the present paper shows that the authors themselves have ignored the upper calculated confidence limit to which they refer (2·8 μU/ml) and have fixed on one nearer to 4 μU/ml. This latter figure approximates to the highest value (4·2 μU/ml) in a group of 29 normal patients described by the same workers in an earlier publication.[2] As the lower normal values are below the limit of sensitivity of the assay employed and the data are therefore truncated, it is very difficult to predict the true distribution without a very much larger sample. It is possible that if a large population were to be sampled values in excess of 4·2 μU/ml would be encountered in some normal subjects.

The authors have therefore not provided a sound basis for their division of patients with autoimmune thyroid disease into those with and those without "subclinical hypothyroidism." Furthermore this arbituary division into two groups may serve to conceal a determinant of TSH secretion which is active in some patients. It is apparent from the data in this paper, and from the work of others, that in some euthyroid patients with damage to the thyroid gland the TSH concentration may be abnormally elevated by any criteria. If the plasma concentrations and effects of thyroxine and triiodothyronine are indeed normal in such patients, then the raised TSH must be sustained by a factor operating independently of the physiological negative feedback of the thyroid hormones on the hypothalamus and the pituitary. This factor could be related either to a reduction in the mass of thyroid tissue capable of synthesizing hormone or to something released from the damaged gland.

The classification that Dr. Evered and his colleagues have proposed does not, therefore, seem to us to be either valid or useful. Indeed the use of the term "subclinical hypothyroidism" is inappropriate as the patients, on the evidence presented, are neither subclinical nor hypothyroid.—We are, etc.,

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1 Ormston, B. J., Garry, R., Cryer, R. J., Besser, G. M., and Hall, R., *Lancet*, 1971, **2**, 1.
2 Hall, R., Amos, J., and Ormston, B. J., *British Medical Journal*, 1971, **1**, 582.