Correspondence

Letters to the Editor should not exceed 500 words.

Death from Exposure

SIR,—Many mountaineering deaths are put down as being due to "exposure," either alone or in conjunction with injuries. These unfortunate people are often diagnosed as being dead by lay people on the hill, and treated accordingly—that is, head covered, often by plastic or similar impervious material. Perhaps, therefore, it would be wise to emphasize the difficulties of diagnosing death in a patient suffering from hypothermia.

Mountain Rescue Figures in 1961-5 for Great Britain

	Accidents	Deaths	Reported Cases of Exposure
1961	106	23	Number not given; "several deaths"
1962	132	41	—
1963	159	19	16
1964	153	27	10
1965	187	35	23

In the body temperature range 75-90° F. (24-33° C.) shivering ceases, and muscular rigidity sets in; this can easily be confused with rigor mortis. Also the skin is pale and cold and subcutaneous oedema develops—again as in death. The pupils react poorly, if at all, to light, and the pulse and blood pressure are often unrecordable.¹ Respiration may be so shallow and slow as to be unnoticeable, especially on the hill, where one is not stripping the body to look for chest movement. One clue is that often, but *not* always, the pupils are constricted, whereas in death they are dilated. Thus if the pupils are constricted then the patient is alive; if dilated he may be either alive or dead. Therefore, unless the injuries are such that the victim cannot possibly be alive, in my opinion rescue teams should not abandon hope and should carry these people down with their mouth and nose uncovered.

One or two other points arise. It is well known that cold is often an added stress on the adrenals in a patient already subjected to other stresses, as in the case of mountaineering mishaps, such as fright, unaccustomed exercise, and injuries.² It has been noted that in hypothermia there is evidence of adrenal damage and depletion of cardiac and hepatic glycogen, which may lead to heart failure even at relatively high temperatures. Cortisone, glucose, and digitalis were recommended as treatment.³ Attempts are now being made to measure plasma cortisol in people suffering from exposure. There is evidence to show that corticosteroids produce a rise in body temperature when used in resuscitation.⁴ Glucose is already advised for exposure cases, and I can see no contra-indications for cortisone—preferably a quick-acting form such as Betnesol—either orally in a conscious victim or by injection. Lay people can do no harm with this and it would

be well worth a try in the apparently hopeless case. Patients should be suitably labelled, as is already done with morphine.

A working party on exposure⁵ recently stated categorically that when a patient has been suffering from exposure but has apparently recovered and insists that he is quite fit he must still be treated as a stretcher case. It is very difficult to maintain body temperature of a victim under conditions where exposure is likely to have arisen, and once completely immobilized on a stretcher the victim would almost certainly tend to lose heat. I quote from Cranston⁶: "For purposes of thermal regulation heat production may be increased rapidly by increased muscular activity, or shivering, in man; this appears to be the only mechanism whereby heat production can be increased rapidly . . ." I cannot therefore understand the rationale of denying a patient who feels fit and wishes to walk down the chance of getting himself back to normal by carefully supervised muscular activity. This would also constitute less of a danger to other members of the party, who themselves are liable to become exposure cases while awaiting a rescue team.—I am, etc.,

Glencoe, Argyll.

M. C. MUNDAY.

REFERENCES

- ¹ Duguid, H., Simpson, R. G., and Stowers, J. M., *Lancet*, 1961, 2, 1213.
- ² Selye H., *Physiology and Pathology of Exposure to Stress*, 1950. Montreal.
- ³ Rees, J. R., *Lancet*, 1958, 1, 556.
- ⁴ Kolpakov, M. G., *The Adrenals and Resuscitation*, 1965. New York.
- ⁵ Exposure, *The Climber*, October 1963.
- ⁶ Cranston, W. I., *Brit. med. J.*, 1966, 2, 69.

Multiple Sclerosis

SIR,—The clinical observations recorded by Professor Henry Miller and his colleagues (22 April, p. 210) and your leading article (p. 192) draw attention to some factors which may influence localization of the pathological process in multiple sclerosis, at least in the first instance. Since both clinical and experimental observations¹⁻³ have raised the possibility that the disease may represent an infection with prolonged incubation or latent period, some recent results on the way in which scrapie (a paradigm of "slow" virus infections) reaches the nervous system may be of interest.

When scrapie material is inoculated intraperitoneally into mice pathological changes are first found in the spinal cord; when inoculated into a forelimb the first lesions occur in the cervical cord at a time when the brain and lower cord are immune; when injected into the leg the first lesions are found in the lumbar cord, while the rest of the nervous system is yet clear. Moreover, if the animal is examined at the right moment, then

lesions after inoculation into a limb may be limited to the ipsilateral side of the cord in the corresponding segments. It would seem that the disease agent reaches the nervous system by the blood, and that its initial localization is conditioned, to some extent at least, by the site of inoculation. Presumably the stimulation associated with inoculation or peripheral trauma in some way facilitates access to the corresponding segments of the neuraxis. In a much neglected paper W. J. German and J. D. Trask⁴ clearly showed the influence which trauma might have in localizing blood-borne poliomyelitis virus to particular regions of the spinal cord.—I am, etc.,

M.R.C. Demyelinating
Diseases Research Unit,
University of Newcastle upon Tyne,
Newcastle upon Tyne.

E. J. FIELD.

REFERENCES

- ¹ Schapira, K., Poskanzer, D. C., and Miller, Henry, *Brain*, 1963, 86, 315.
- ² Pålsson, P. A., Pattison, I. H., and Field, E. J., in *Slow, Latent, and Temperate Virus Infections*, 1966, edited by D. C. Gajdusek, C. J. Gibbs, jun., and M. Alpers. U.S. Department of Health, Education and Welfare. NINDS Bethesda, Monograph No. 2, p. 49.
- ³ Field, E. J., *Brit. med. J.*, 1966, 2, 564.
- ⁴ German, W. J., and Trask, J. D., *J. exp. Med.*, 1938, 68, 125.

Propranolol-induced Depression

SIR,—We have noted with interest Dr. H. J. Waal's letter (1 April, p. 50) suggesting that the high incidence of depression among her hypertensive patients was associated with propranolol administration. This, however, does not agree with the experience of other workers. Published reports,¹⁻⁵ excluding Dr. Waal's paper,⁶ describing experience with propranolol in 154 patients with hypertension include only one case of transient depression.⁵

We have attempted to determine the incidence of depression in patients receiving propranolol. At present there are follow-up studies with propranolol in hypertension in progress both here and in other parts of the world in collaboration with this department. The trials involve some 167 patients, and so far only one case of overt depression during treatment with propranolol has been reported. This patient was known to be a depressive prior to propranolol treatment. No patients have had to stop their treatment because of depression. Thus the published and unpublished reports concern 321 patients who have been receiving doses of propranolol ranging from 70 to 1,000 mg. or more per day over a period of 6 to 39 months. Therefore there is at present no evidence from these studies to support Dr. Waal's suggestion that the depression she associates with propranolol is either time or dose dependent. With regard to general clinical experience, we estimate that approximately 60,000 patients are currently receiving propranolol in various indications. All reports of side-effects associated with propranolol administration are recorded and followed up by our adverse reactions section. Examination of these records to determine the incidence of depression shows that a