Correspondence

Letters to the Editor should not exceed 500 words.

Trypanosomiasis in Britain

SIR,—Two cases have recently come to my notice of failure to diagnose trypanosomiasis in an African in this country. In one, with the lapse of nearly three years since the patient was last in Africa, the possibility of a tropical cause of the illness may have been dismissed. In the other the presenting symptoms were personality changes, and trypanosomiasis was apparently never considered as a possible cause.

The chronic, Gambian form of trypanosomiasis may continue with virtually no symptoms for three, or even more, years before involvement of the central nervous system affects the patient noticeably. It may then present with complaint of persistent headache, with other meningeal symptoms, or with more apparently psychological signs such as increasing depression or with marked personality change, not infrequently with impaired responsibility.

I wish to make an urgent plea for the consideration of a diagnosis of trypano-

somiasis in any person from Africa, whether African or not, now under medical care, perhaps even in a mental hospital, in this country with undiagnosed symptoms which could be due to trypanosomiasis. The finding of a C.S.F. protein of under 30 mg./100 ml. and a normal cell count would be strong evidence against the diagnosis of trypanosomiasis, but if the C.S.F. protein is near or above 40 mg./100 ml., or if an abnormal cell count, particularly a lymphocytosis, in the C.S.F. is found, the possibility of trypanosomiasis should not be discarded until some other unquestionable cause has been found. If further opinion or advice is needed there are schools of tropical medicine with associated hospitals at London, Liverpool, and Edinburgh.—I am, etc.,

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Serum Creatine Kinase and Exercise

SIR,—In the paper of Drs. A. Vejjajiva and G. M. Teasdale (26 June, p. 1653) there are several points which merit discussion. The group of patients are all either "rugby players" or "oarsmen" and cannot be taken to be representative of the normal population. This is apparent (as the authors observe) in the fact that no less than one-quarter (3 out of 12) of the subjects exhibited pre-exercise serum-creatine-kinase (C.K.) values which are well outside the normal range for the method used.

One of the factors which makes serum-C.K. estimations so useful is the consistency of values obtained in non-exercising but ambulant subjects. Using almost identical methods, the normal range has been shown to be remarkably constant by the estimations of Hughes in 73 subjects,¹ Griffiths in 282 subjects,² and by Pearce, Pennington, and Walton in 105 estimations in 87 subjects.³ It is clear that the definite elevations (two to fourfold) of serum-C.K. activity in a quarter of the subjects reported by Drs. Vejjajiva and Teasdale are not representative of a random sample of healthy adults, and these high values implicate the operation of some other factor.

It is undoubtedly difficult to standardize physical exercise and to compare the results of one published series with another. Despite these inherent limitations, there has been a notable failure to demonstrate an increase in serum-C.K. levels after exercise in the studies reported.³⁻⁵ A further point of importance is that the assumption that exercise does not alter serum-C.K. levels in patients suspected

of having a myopathy does not appear to invalidate the value or the reliability of serum-C.K. estimations as a diagnostic measure.⁶⁻⁹ If exercise was even an occasional factor in the elevation of serum-C.K. levels, it would seem likely that a significant proportion of normal subjects and patients with neurogenic muscular atrophy would have an elevated C.K. value, which being recognized would pose a problem in the biochemical differentiation of such patients from those suffering from a primary myopathy. This has not arisen in the published reports.

All the subjects studied by Drs. Vejjajiva and Teasdale were of athletic inclination, and although the type and severity of the exercise performed for 80 minutes are not specified it seems reasonable to assume that the physiology of muscle contraction in the athlete may differ quantitatively from that of the more sedentary populace. Such subjects may have performed much more strenuous muscular exercise, and this could explain both the high pre-exercise C.K. levels and the increase which occurred after exercise. A good analogy is seen in patients suffering from tetanus, in whom moderate increases in serum-C.K. levels occur^{10,11}; this is perhaps the best example of repeated muscular contraction of great severity.

It is therefore probable that only muscular exercise of considerable severity can increase the serum-C.K. activity. Such a degree of exertion is not common amongst members of the population carrying out their everyday tasks, and for this reason it is not necessary to impose a period of physical inactivity

before taking blood for serum-C.K. estimation unless the patient has indulged in an unusual degree of exercise.—I am, etc.,

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SIR,—Drs. A. Vejjajiva and G. M. Teasdale (26 June, p. 1653) have given a timely reminder of the need for caution in interpretation of serum-creatine-kinase levels in view of the effect of prolonged physical exercise on the serum activity of this enzyme.^{1,2} The average serum-creatine-kinase levels in a group of medical students taking part in the 1963 London to Brighton "stroll" increased sevenfold by the time they reached Gatwick (25 miles) and twentyfourfold by the end of the journey (53 miles).³ Prolongation rather than severity of muscular activity appears to be the important factor, as short-term severe exercise appears to be ineffectual.³⁻⁵ It is of interest perhaps that prolonged tachycardia also is associated with a marked increase in serum-creatine-kinase levels.⁶

The second important point is that some subjects may betray serum activities greater than the accepted upper limit of normal when carrying on their normal daily life—this occurs particularly in children and young adults,¹⁻³ and is not necessarily related to the degree of physical labour involved. Such subjects might be termed "enzyme-labile."

Drs. Vejjajiva and Teasdale also point out that their normal range was based on a limited number of subjects. My studies based on 808 subjects forced me to adopt different normal ranges for adult ambulant and non-ambulant subjects, as the upper limit of normal for the former was roughly twice that of non-ambulant subjects. For children, of course, only an ambulant range is necessary. Females tended to have a slightly lower range of serum-enzyme activities than did males.

These findings are of particular importance when creatine kinase is used for detection of biochemical carriers of the muscular-dystrophy gene, as the ranges for carriers and normal ambulant subjects overlap,⁶ and unless attention is paid to the circumstances under which the specimen is drawn an incorrect