mentioned extracerebral sources, especially under the heavy strain of a marathon race, seem to us a more probable source for CK-BB activity in marathon runners after a race. This also tallies with the observation of Phillips et al. that none of the runners demonstrated any neurological abnormalities after the race. Furthermore, most of the runners also revealed increased heart-type isoenzyme (CK-MB) activities and crossreactivity of CK-MB in radioimmunoassays. It cannot be excluded as a possible explanation for the observations made by Phillips et al., as they note.

We however, are more concerned about the occurrence of increased CK-MB in most marathon runners after a race. CK-MB has established its value as a highly heart-specific enzyme. The presence of increased CK-MB together with electrocardiographic abnormalities and increased serum CK-MB levels during marathon competition. Stansbie et al. seem to share our concern about the risks when skeletal muscle can release excess creatine kinase in serum, but this cannot be excluded as an alternative source of increased CK-MB levels during marathon competition. It has been suggested that increased catecholamines might cause myocardial damage in stroke and acute head injury. Similar humoral mechanisms could exist during a marathon race and could explain abnormal CK-MB activities after a marathon. Normal skeletal muscle can release excess creatine kinase in serum, but this is muscle-type (CK-MM) not heart-type. The hypothesis that endurance exercise increases heart-type/muscle-type isoenzyme ratio in skeletal muscle, the situation peculiar to diseased muscle, remains to be proved before transient rhodobymolysis from skeletal muscle can be regarded as an alternative source of increased CK-MB activities in collapsed runners complaining of chest pain are not taken seriously.

HORMONES AND FLUID RETENTION IN CIRRHOSIS

Sir,-Your June 12 editorial notes that the extent of the contribution of the renin-aldosterone system to the pathogenesis of ascites in cirrhosis remains undefined. Analysis of the data of P. W. Wong and colleagues (Gastroenterology 1979; 77: 1171) may help to clarify the issue. These workers studied the response of plasma renin (PRA) and aldosterone to the infusion of saline or albumin in cirrhosis with ascites in a stable nutritional and metabolic state. Calculations based on their published data show that PRA and aldosterone (ALD) correlate well:

\[ \text{Albumin infusion} - \text{Cirrhotic} \]: ALD = 20.6 + PRA + 154.5 \times (r = -0.96). \]

\[ \text{Normal} \]: ALD = 2.21 + PRA + 28.5 \times (r = 0.98). \]

The slopes of these regression lines were steeper in the cirrhotics than in the controls—i.e., if the steady state in the cirrhotic is disturbed by the infusion of albumin or saline, then, for any change in PRA, the corresponding change in aldosterone is several times greater than normal.

When, however, natriuresis is induced by immersion in water, the slopes of the lines linking PRA and aldosterone are similar in cirrhotics and in controls (Epstein M, et al. Circ Res 1977; 41: 818—29). Immersion increases the central blood volume, the product of the circulation time and the cardiac output. When these factors are measured separately, the circulation time does not appear to change upon immersion while there is an increase in cardiac output (Arborelius MJR, et al. Aerospace Med 1972; 43: 256-59). The natriuretic effect of immersion in water is therefore presumably attributable to an increased cardiac output. These data suggest that the renin-aldosterone system in patients with cirrhosis responds differently from normal to a sodium challenge or to modification of the colloid osmotic pressure of the plasma, but similarly to normal patients to modifications in cardiac output. The extent to which these contribute to fluid accumulation in cirrhosis is likely to vary in different patients.