Sudden death is well documented as an unexpected occurrence in trained long distance runners, skiers, and swimmers. On post-mortem examination in such cases, the abnormal findings may include unsuspected congenital anomalies or unrecognized coronary atherosclerosis or cardiomyopathy. In many cases, no abnormality is found, and here, recourse for an explanation involves the prevalence of cardiac arrhythmias in endurance athletes. In all such cases, the issues are clear at the time of clinical presentation, although the etiology may be completely obscure.

But what about the athlete who collapses at or near the end of a race, and does not die or prove to have any lasting ill effects? The physician on the scene may think of some sort of cardiac attack, or perhaps a spontaneous pneumothorax as the cause of the collapse. Having ruled out pneumothorax by ordinary physical examination, the physician at the scene may call for an electrocardiogram, knowing full well that it is not completely reliable under the circumstances. On the other hand, he is likely to have complete confidence in the measurement of certain serum enzyme activities. At the moment, the serum enzyme activity generally held to be most specific in the diagnosis of myocardial infarction is the serum creatine kinase activity, especially heart specific isoenzyme. Actually, this measurement will merely heighten the confusion and uncertainty in the circumstances here discussed.

The measurement of serum creatine kinase activity and of the activity of the so-called "heart specific" or MB isoenzyme has by now been shown to give elevated values in endurance athletes after a race, ten studies of this subject having already been published with complete agreement. These positive findings have been correlated tentatively with the development of microscopic lesions in skeletal muscles. When the serum activity elevations were studied in healthy (but exhausted) athletes, the increases were found to be related to age, although this relation is not found in the subjects in whom the elevations were owing to myocardial infarction. The increases in the two groups were not significantly different as a whole, but the rate of clearance of the enzyme from the blood was perhaps more rapid in the case of the normal exhausted athletes. Actually, there is no proof in the cases reported that the enzyme did not originate in the myocardium of the healthy athletes in whom the rise occurred.

There seems to be no laboratory test that will distinguish the two groups except for the signs of tissue damage in the myocardial infarct group, i.e., fever, leukocytosis, elevated sedimentation rate. Clinical judgment at the time of clinical presentation is the main diagnostic recourse. If there is any lesson to be learned from all this it is that biochemical tests describe biologic phenomena, and do not have specificity required for clinical diagnosis. As an astute Nobel laureate in physiology once pointed out, "One has to be a good clinician to interpret laboratory data." The common comment that such data, even when useless in diagnosis, enhance our understanding of disease processes is not in accordance with available fact: what passes for understanding in such cases is usually nothing more than the harmonizing of test results or other observable data with some currently popular theory.

REFERENCES
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