To the Editor: Post-exercise release of cardiac troponin (cTnT) and cTnI has been previously reported after prolonged bouts of exercise (1). However, this release has only been observed in a limited number of subjects (2). It is unclear whether post-exercise release of cTn represents necrosis of cardiac myocytes, and thus irreversible damage, or a transient and reversible change in membrane permeability of the myocyte (2). Similarly, whether the substrate for post-exercise release of cTn is physiologic or pathologic has not been determined.

We examined the kinetics of cTnT release during and after completion of a marathon using repeated blood draws to investigate the time course of exercise-related elevations in cTnT and to differentiate post-exercise kinetics of cTnT from cTnT release subsequent to acute coronary syndrome (ACS). Nine well-trained men completed a marathon (42.2 km) on a motorized treadmill. Venous blood samples (5 ml) were drawn through a cannula at rest and at 30-min intervals during, immediately after, and at 1, 3, 6, 12, and 24 h after exercise. Serum was analyzed for cTnT using the third-generation immunoassay (Roche Diagnostics, Lewes, United Kingdom). During the marathon, between 60 and 120 min, cTnT increased in all participants. At race completion, or within 1 h of completing the marathon, cTnT had returned to baseline values in all subjects. All but 1 subject showed a further release of cTnT within the 24-h recovery period, with 5 of these subjects having an elevated cTnT 24 h after exercise (Fig. 1). Importantly, these data are the first to demonstrate that cTnT is released in all persons in response to exercise. Previous studies have reported between 0% and 78% of participants having a cTnT positive blood sample after bouts of prolonged exercise (1,3). The time course and biphasic release of cTnT shown presently suggest that the discrepancy between previous findings may be related to the timing of blood sampling and the simple pre–post-exercise design that most authors have adopted (1,3). The release of cTnT within the first 60 min of exercise indicates that exercise-induced cTnT release is not necessarily limited to prolonged endurance exercise. We suggest that the consistent elevation and pattern of cTnT observed in all participants during and after exercise likely reflect a physiologic as opposed to pathologic substrate.

Recently, using a highly sensitive assay for cTnI capable of identifying single troponin molecules, Wu et al. (4) have shown that there is a very low (≤7 ng/l) background level of cTnI in the healthy population. It is possible, therefore, that there is a continual turnover of cTn in the healthy heart and that exercise, and the associated increased myocardial demand, is a stimulus for an increased turnover. It has been previously demonstrated that no relationship exists between post-exercise cTn release and magnetic resonance imaging evidence of cardiac damage (5). Further, the serum concentrations of cTnT observed in our study were relatively small in comparison with those observed after a myocardial infarction (6). Together, this provides further support of a physiologic substrate responsible for post-exercise cTn release. Within skeletal muscle, physiologic adaptation is preceded by protein breakdown; perhaps exercise-induced cardiac hypertrophy follows a similar model.

Despite the early release and low absolute values, the biphasic release of cTnT observed during and after marathon running and the persistent elevation at 24 h in some subjects mean that 24-h sequenced blood draws cannot be used to differentiate exercise-induced cTnT release from the early onset of ACS. Accordingly, using cTnT diagnostically for patients admitted with suspected ACS subsequent to bouts of endurance exercise is problematic; these patients, therefore, require further work-up.

Early immunoassays for cTn exhibited cross-reactivity with skeletal muscle damage. However, the cardiospecificity of the third-generation cTnT assay has been validated in the presence of significant skeletal muscle damage (7). Accordingly, it is unlikely that the results of the present study are due to interference from skeletal muscle damage. To further understand exercise-induced cTn release, future work should use the newly available, highly sensitive troponin assays.

Until now, release of cTnT has been proposed as pathognomonic of cardiac necrosis (8). The present data should prompt reconsideration of this belief. It appears unlikely that minor elevations in cTnT subsequent to endurance exercise are due to myocardial necrosis. Rather, it is possible that post-exercise cTn release represents reversible cardiomyocyte membrane damage that may reflect part of a remodeling process. Although cTnT is diagnostic of ACS in the clinical setting, in a healthy exercising population, cTnT is routinely released in all persons after periods of increased myocardial demand.

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Figure 1 Individual cTnT Release During and After Completion of a Marathon

Individual cardiac troponin T (cTnT) release during (min) and after (h, post-exercise) completion of a marathon.