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## Letters to the Editor

# Exercise-Induced Troponin Elevation

## Not Necessarily a Benign Phenomenon

In their comprehensive review of exercise-induced cardiac troponin elevation, Shave et al. (1) come to the conclusion that this is a benign phenomena most likely related to leakage of troponin from the cardiac myocyte membrane rather than myocyte necrosis—the usual cause of troponin elevation. In the absence of long-term follow-up of elite ultra-endurance athletes, this conclusion should be viewed with caution. There is increasing evidence that, in some athletes, participation in multiple extreme endurance events over a long period of years can lead to abnormal right ventricular (RV) enlargement, dysfunction, and—more ominously—potentially lethal arrhythmias (2–4). Such athletes are clinically and genetically distinct from those suffering from familial arrhythmogenic RV dysplasia/cardiomyopathy (5). An appropriate terminology for such individuals is "exercise-induced right ventricular dysplasia" (2–5). A plausible hypothesis for this syndrome is that extreme endurance exercise places a strain on the RV that on occasions leads to myocardial necrosis, albeit small, as reflected in post-exercise elevated troponin levels. The cumulative effect of repeated episodes of necrosis can eventually lead to sufficient fibrosis to result in RV dysfunction and to act as a substrate for potentially lethal arrhythmias. How prevalent exercise-induced RV dysplasia is and whether there is a genetic predisposition to the condition remains to be determined.

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## Reply

We appreciate Dr. Harper's interest in our recent report (1). Dr. Harper suggests that without reassuring evidence from longitudinal studies it is possible that ultra-endurance exercise can produce a variant of right ventricular cardiomyopathy and suggests that the elevated cardiac troponin (cTn) observed after exercise are not benign. From a historical viewpoint, ultra-endurance events are not new. At the turn of the last century walking and running contests of several days duration were popular spectator events and raised concerns similar to those of Dr. Harper, but concerns about bicyclists', runners', and rowers' hearts were never documented (2). Right ventricular enlargement from endurance activity is not unusual but is rather one of the expected adaptations from exercise training. Indeed, all 4 cardiac chambers enlarge with exercise training, but there is little evidence of right ventricular dysfunction, except for reversible, transient changes after endurance events (3)—which might be related to a number of factors, such as elevations in heart rate, plasma volume alterations, or desensitization of beta-adrenoceptors. Furthermore, the observations that cTn is elevated after low-intensity exercise such as walking (4), early during a treadmill marathon (5), and after 30 min of high-intensity running (6) suggest that cTn elevations are common with exercise and not necessarily a product of prolonged effort. Recent studies have also shown no relationship between exercise-induced cTn release and late gadolinium enhancement with cardiac magnetic resonance imaging (7) and that, unlike acute coronary syndromes, cTn rapidly returns to baseline after exercise. In combination, such observations suggest a benign event. Finally, we would caution Dr. Harper as he has us: in the absence of longitudinal studies, it is provocative and premature to suggest that right ventricular problems observed in a case series of endurance athletes are produced by their athletic participation.