Response by Bohm et al to Letter Regarding Article, “Right and Left Ventricular Function and Mass in Male Elite Master Athletes: A Controlled Contrast-Enhanced Cardiovascular Magnetic Resonance Study”

In Response:
We agree with Heidbuchel and colleagues that our findings do not clearly refute the concept of exercise-induced right ventricular (RV) damage because no prospective longitudinal study has been performed so far. However, our finding of marked RV remodeling without functional RV impairment or scarring challenges the notion of an exercise-induced arrhythmogenic right ventricular cardiomyopathy.

First, we studied an exclusive cohort of world-class master endurance athletes with a training history of 29±8 years. Such athletes are not as easily available as ambitious recreational athletes, who would not demonstrate an extensive cardiac remodeling as our studied athletes. Our athletes demonstrated clear structural exercise-induced cardiac remodeling with a mild RV chamber size predominance of about 6% (left ventricular end-diastolic volume/right ventricular end-diastolic volume ratio <1, mean left ventricular end-diastolic volume 203±26 mL, and right ventricular end-diastolic volume 215±43 mL; control subjects: mean left ventricular end-diastolic volume 139±42 mL and right ventricular end-diastolic volume 133±37 mL), which means that the RV seems more susceptible to the hemodynamic load of endurance exercise.1 However, we agree with the editorialists Wasfy and Baggish that, in the absence of concomitant scarring or functional impairment, RV injury seems an unlikely interpretation.2 Supporting evidence comes from an early invasive study in elite athletes by Kindermann et al,3 which demonstrated that measurements of pulmonary-arterial pressures with Swan-Ganz catheterization at rest and during maximal bicycle ergometry in endurance athletes with athlete’s heart remained in the normal range even under exercise stress.

In our study, 5 athletes had right ventricular ejection fraction <45% (none ≤40%), and it is known that resting cardiac function in athletes with athlete’s heart, at times, lies in the low normal to mildly abnormal range. However, complementary echocardiographic functional metrics only showed a significant lower RV basal longitudinal strain in athletes compared with controls, a finding that has been confirmed in the literature.4 Those athletes with a right ventricular ejection fraction <45% showed an exceptional good exercise capacity and did not present any pathological signs at cardiovascular magnetic resonance. We agree that exercise stress testing with cardiac imaging represents a tool to document intact contractile reserve. However, to the best of our knowledge, no objective parameters or definitions exist that would allow us to differentiate between physiological RV remodeling in the athlete’s heart and early arrhythmogenic right ventricular cardiomyopathy by cardiac stress test imaging.

Some years ago, LaGerche et al5 presented a study in which 5 out of 40 endurance athletes (12.8%) were found to have delayed gadolinium enhancement. Interestingly, athletes with evidence of myocardial fibrosis demonstrated significantly larger RVs with lower indices of RV systolic function, and the authors deduced that cardiac scarring in endurance athletes results from repeti-
tive myocardial injury caused by long-term endurance exercise. Therefore, we wonder why this noninvasive tool, capable of assessing the extent of fibrosis in presumably healthy athletes, is now considered an inaccurate method. In our study, only 1 of 33 athletes demonstrated pathological late enhancement, and the region was limited to the posteroinferior epicardium, corresponding most likely to a previous asymptomatic pericarditis.

Regarding cardiac biomarkers, we determined high-sensitive troponin T and N-terminal pro-brain natriuretic peptide at rest to detect acute myocardial damage and maladaptation. Based on the fact that the cardiopulmonary exercise test until volitional exhaustion lasted ≈20 minutes only, a second determination of the biomarkers would not have added any further information in all likelihood.

Finally, we would like to emphasize that previous studies only demonstrated RV cardiac fatigue with a normalization after several days. Thus, the hypothesis of an exercise-induced arrhythmogenic right ventricular cardiomyopathy has not been proven so far. Furthermore, there is no epidemiological evidence that long-term high-volume endurance exercise at an elite level leads to premature death. In conclusion, as long as no longitudinal data on RV morphology and function exist in endurance athletes with athlete’s heart, care should be taken not to irritate athletes, coaches, and physicians with the terminus exercise-induced arrhythmogenic right ventricular cardiomyopathy. Instead, we suggest using the expression exercise-induced RV cardiac fatigue.

We thank Leischik and colleagues for their interest in our study, and we agree with them that healthy endurance athletes seem to show an overall benign pattern of cardiac remodeling in response to long-term high-volume training and competition.

DISCLOSURES
None.

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REFERENCES
Response by Bohm et al to Letter Regarding Article, "Right and Left Ventricular Function and Mass in Male Elite Master Athletes: A Controlled Contrast-Enhanced Cardiovascular Magnetic Resonance Study"
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_Circulation_. 2016;134:e364-e365
doi: 10.1161/CIRCULATIONAHA.116.024362

_Circulation_ is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
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