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Acute, Exercise Dose-Dependent Impairment in Atrial Performance During an Endurance Race

2D Ultrasound Speckle-Tracking Strain Analysis

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ABSTRACT

OBJECTIVES This study sought to understand and characterize the acute atrial response to endurance exercise and the influence of the amount of exercise performed.

BACKGROUND Endurance exercise seems to be recognized as a risk factor for developing atrial arrhythmia. Atrial geometrical and functional remodeling may be the underlying substrate.

METHODS Echocardiography was performed in 55 healthy adults at baseline and after a 3-stage trail race: a short race (S) (14 km), n = 17; a medium race (M) (35 km), n = 21; and a long race (L) (56 km), n = 17. Analysis consisted of standard, speckle-tracking assessment of both the left ventricle (LV) and right ventricle (RV) and both the left atrium (LA) and the right atrium (RA): a-wave strain (Sa) and strain rate (Ra) as a surrogate for atrial contractile function and s-wave strain (St) and strain rate (SR) as reservoir function.

RESULTS After the race, RA reservoir function decreased in group M (Δ% SRs: −12.5) and further in group L (Δ% SRs: −15.4), with no changes in group S. RA contractile function decreased in group L (Δ% SRa: −9.3), showed no changes in group M (Δ% SRa: +0.7), and increased in group S (Δ% SRa: +14.8). A similar trend was documented in LA reservoir and contractile function but with less pronounced changes. The decrease in RA reservoir after the race correlated with the decrease in RV global longitudinal strain (GLS) (Δ% RVGLS vs. RASt and RASRs: +0.44; $p < 0.05$ and +0.41, respectively; $p < 0.05$).

CONCLUSIONS During a trail-running race, an acute exercise-dose dependent impairment in atrial function was observed, mostly in the RA, which was related to RV systolic dysfunction. The impact on atrial function of long-term endurance training might lead to atrial remodeling, favoring arrhythmia development. (J Am Coll Cardiol Img 2016;■:■-■) © 2016 by the American College of Cardiology Foundation.

Recent data have shown that endurance training is associated with an increased risk for atrial fibrillation (AF) and atrial flutter (1-3). A dose-response relationship between endurance training load and AF also has been demonstrated (4-6). The pathophysiology of potential exercise-related AF is still poorly understood, but exercise-induced atrial remodeling seems to be

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ABBREVIATIONS AND ACRONYMS

FWLS = free-wall longitudinal strain

GLS = global longitudinal strain

LVEDV = left ventricle end-diastolic volume

LVEF = left ventricle ejection fraction

RVEDA = right ventricle end-diastolic area

RVFAC = right ventricle fractional area change

Sa = strain during contractile atrial phase

SRa = strain rate during contractile atrial phase

SRs = strain rate during reservoir atrial phase

St = strain during reservoir atrial phase

one of the major contributing factors (1,7). Classically, atrial remodeling has been described in terms of biatrial dilation (8,9) and normal atrial function among athletes (8,10). However, recent studies have shown reduced atrial deformation in the contractile and reservoir phases in athletes at rest compared to those in sedentary controls (11,12). The high cardiac output required during endurance exercise training has been shown to impose a high degree of stress on all myocardial structures, which seems to be proportionate to the amount of exercise performed (13). Following Laplace's law, this increase in wall stress should be especially important for heart cavities with thin-wall structure: the right ventricle (RV) and both the left atrium (LA) and right atrium (RA). Indeed, various studies have shown an acute RV performance impairment after high-intensity endurance exercise (13–15). Impairment in LA function (16) and indirect evidence of myocardial edema after completing long-distance endurance races has been reported (17). However, limited data are available for the acute response of the atria (particularly the RA) to high-intensity endurance exercise and to the impact of different amounts of exercise.

In recent years, atrial strain and strain rate analysis by 2-dimensional (2D) speckle-tracking echocardiography has emerged as a novel method to evaluate LA and RA functions (18). The assessment of atrial function by strain and strain rate has been used as a predictor of AF recurrence in various clinical situations (19). Recently, the utility of speckle-tracking echocardiography also has been demonstrated in the evaluation of atrial function in athletes (8,12).

In this study, we aimed to comprehensively evaluate atrial adaptation and remodeling after intense endurance exercise. The study had 3 objectives: 1) to analyze atrial performance in response to different cumulative intensities of exercise; 2) to analyze the influence of ventricular function on atrial performance; and 3) to characterize the different atrial responses among individuals. The principal hypothesis of our study was that, after high-intensity endurance exercise, an impairment in RV function would be presented, as was suggested before, but potentially also in both atria, particularly the RA, directly influenced by RV performance. In addition, we presumed that RV and atrial responses to endurance exercise were going to be influenced by the amount of exercise performed, but we also expected high interindividual variability in relation with individual exercise-adaptive mechanisms.

METHODS

STUDY POPULATION. Volunteers were recruited 8 months before a trail-running event in Catalonia, "La Gran Volta a la Cerdanya." Of 1,484 runners registered for the event at the time of recruitment, 716 agreed to participate. The competition included 3 different races: a 14-km race with 500 m cumulative altitude, a 35-km race with 1,500 m of cumulative altitude, and a 56-km race with 3,400 m of cumulative altitude. Participants were assigned to each distance group according to their prior training, defined as total self-reported hours of endurance training per week: <3 h/week for the short-distance race (group S), 3 to 10 h/week for the medium distance (group M), and >10 h/week for the long-distance (group L). We randomly selected 20 healthy males from each distance group. The 60 selected participants underwent a 3-month standardized training protocol adjusted for each race distance and guided by a personal trainer. Cardiovascular disease was excluded by a complete cardiovascular screening that included a detailed medical history and physical examination, 12-lead surface electrocardiogram, a treadmill stress test, and transthoracic echocardiography at rest.

The study protocol complied with the Declaration of Helsinki and was approved by the Ethics Committee of our institution, and all participants provided written informed consent.

ECHOCARDIOGRAPHY. Echocardiography was performed 24 h before the race and within the first hour of arrival at the finish line. All echocardiographic images were acquired with a commercially available ultrasound system (Vivid Q, GE Medical, Milwaukee, Wisconsin). Images were acquired from the parasternal (long- and short-axis) and apical (4-, 3- and 2-chamber) views. Three cardiac cycles for each acquisition were digitally stored in a cinematic loop format for off-line analysis with commercially available software (version 113, EchoPac, GE Medical). The left ventricle (LV) and RV were assessed according to American Society of Echocardiography and European Association of Cardiovascular Imaging guidelines (20). Maximum and minimum volumes and atrial volume before the atrial contraction (Vpa) were calculated using the biplane summed discs method (20) for both LA and RA. Then, pump, reservoir, and conduction volumetric functions were calculated using previously described formulas (10). All dimensions were indexed for body surface area according to the DuBois formula. RV and LV diastolic