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# Different Effects of Prolonged Exercise on the Right and Left Ventricles

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**To examine the functional consequences of the greater increase in right ventricular work with exercise, the effects of prolonged exercise on the right and left heart chambers were compared in 41 athletes before, at the finish (13 min) and after recovery (28 h) from the Hawaii Ironman Triathlon (3.9 km swim, 180.2 km bike ride, 42.2 km run). Two-dimensional and Doppler echocardiograms were analyzed for left and right atrial and ventricular areas at end-diastole and end-systole, right and left ventricular inflow velocities and mitral and tricuspid regurgitation.**

**After exercise, left ventricular and left and right atrial sizes were reduced, whereas right ventricular size increased (diastole: 21.4 to 24.2 $cm^2$; systole: 15.8 to 18.2 $cm^2$; $p < 0.01$). The emptying fraction of all chambers was unchanged. Left but not right ventricular inflow showed an increase in peak velocity of rapid filling, whereas both atrial systolic velocities increased (26 to 38 cm/s tricuspid; 38 to 54 cm/s mitral; both $p < 0.01$). Overall, the right ventricular early to atrial velocity ratio was reduced after exercise (1.56 to 1.17; $p < 0.05$) and the left ventricular pattern was unchanged. The prevalence of tricuspid regurgitation was statistically unchanged (86% to 52%), although that of mitral regurgitation was greatly reduced (76% to 0%). Changes in all variables returned toward prerace values during recovery.**

**Thus, in highly trained athletes, prolonged exercise causes differing responses of the right and left ventricles. These differences may be due to changes in right ventricular function, shape or compliance.**

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The left ventricular response to exercise has been extensively characterized (1,2). Recently, the effects of prolonged exercise were investigated (3–7), and evidence for altered systolic and diastolic performance was found. Although the mechanism of such "cardiac fatigue" is unclear, it is likely related to the increased stresses imposed by exercise.

The effects of exercise are less well studied (8–12) on the right than on the left side of the heart, and the right heart response to prolonged exercise is unknown. Comparison of the work load placed on the right and left ventricles during brief exercise reveals several important differences. Although cardiac output and heart rate must be similar over the long term in both circulations, the reduction in pulmonary vascular resistance is less than the reduction in peripheral resistance, and the increase in pulmonary artery pressure is greater than that in systemic arterial pressure. Thus, in response to a given amount of exercise, right ventricular stroke work increases substantially more than does left ventricular work (8,9). However, the impact of such inequality, if any, is unknown.

The present study was performed to examine the hypothesis that this difference in work load results in differing right and left ventricular responses to exercise. Two-dimensional echocardiography and Doppler velocimetry were performed before, immediately on finishing and during recovery from the Hawaii Ironman Triathlon, an ultraendurance race consisting of a 3.9 km swim, 180.2 km bike ride and 42.2 km run.

## Methods

**Study subjects.** The study group consisted of 41 ultraendurance athletes with a mean age of 38 ± 10 years (range 24 to 63), including 22 men and 19 women. Environmental conditions during the race included humidity ranging from 40% to 85%, ambient temperature ranging from 24° to 42°C and water temperature of 26°C. During the race, the athletes chose their own speed and rest periods and had liberal access to fluids and food. Mean nude and dry body weight of

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30 subjects immediately before the race was 69.2 ± 11.7 kg (range 51.8 to 89.1), falling to 67.6 ± 10.5 kg (range 52.3 to 90.0) at race finish and 66.7 ± 11.5 kg (range 51.4 to 85.5) after recovery.

**Two-dimensional echocardiography.** All subjects underwent three echocardiographic studies: a baseline study 2 to 4 days before the race, a second study immediately on finishing the race (average time from finish to recording 13 ± 6 min; range 3 to 23) and a third study after 1 to 2 days of recovery (28 ± 9 h). We did not find any significant differences between those imaged early after the race (first 5 min) and those imaged later (last 5 min). For technical reasons, echocardiographic studies were not completed in two athletes.

Two-dimensional echocardiograms of all four cardiac chambers were recorded from the apical four chamber view using a commercially available ultrasonograph. Imaging location and gain settings were adjusted to yield on-axis views including the cardiac apex with optimal definition of endocardial and epicardial borders, and were recorded for use during subsequent imaging. Right ventricular images were recorded separately from left ventricular images when optimal definition of the apex and tricuspid orifice and maximal right ventricular short-axis diameter were obtained according to the method of Bommer et al. (13). Patient and transducer position was recorded at initial study to ensure that identical portions of all four cardiac chambers were imaged in a reproducible manner. All studies were performed by a single technician and subsequently digitized by a single observer.

*The cavity area of each of the four cardiac chambers was digitized at end-diastole and end-systole on three to five consecutive beats* using a computerized light pen system (Digisonics), and emptying fraction was calculated for each chamber. Results were thus obtained during all phases of the respiratory cycle during quiet respiration and were averaged to eliminate possible respiratory effects. In addition, total cardiac diastolic and systolic cavity areas (both atria and ventricles) were calculated (14).

*The apical four chamber view was used rather than short-axis views for several reasons.* First, the time available for data collection was extremely limited, and this view allowed imaging of all cardiac chambers and atrioventricular (AV) valve flow. Second, this view also allowed assessment of both right and left ventricular systolic function. Right ventricular planar emptying fraction obtained in this manner correlates closely with both contrast and nuclear angiographic ejection fractions (15,16). Third, high quality subcostal views were difficult to obtain immediately after race finish.

**Doppler recordings.** To assess right and left ventricular diastolic performance, 18 subjects underwent two-dimensionally guided pulsed Doppler ultrasound recordings of left and right ventricular inflow, and a different subgroup of 21 subjects underwent Doppler color flow mapping to assess the presence of mitral and tricuspid regurgitation. Studies were performed at two identical races; a color Doppler ultrasonograph was available at only one event and a pulsed Doppler ultrasonograph was available at the other. Because the study design required obtaining immediate postrace data, echocardiographic studies were kept quite brief so that recordings would not be obtained many minutes into the recovery phase. For this reason, we chose not to perform time-consuming pulsed Doppler mapping for detection of regurgitation when Doppler color flow mapping was not available; neither did we record left and right ventricular inflow velocities in those athletes undergoing color Doppler examinations. Importantly, the subjects undergoing pulsed and color Doppler examinations did not differ in any respect, either demographically or with regard to race performance. All Doppler studies were performed at baseline before the race, at finish and during recovery. The subjects finished the race over an extended time period (10 to 15 h); each individual postrace study averaged approximately 20 min and was completed before the next athlete crossed the finish line.

*Flow velocities were recorded at the midpoint of the mitral and tricuspid anuli* using the apical four chamber view parallel to presumed flow, with optimal definition of the spectral envelope. Data from three to five consecutive cardiac cycles were digitized to yield peak velocities of right and left ventricular inflow in early and late diastole and the ratio of early to late diastolic flow velocities. Validations of Doppler assessment of left ventricular filling have been performed by comparison with angiographic and nuclear data (17,18). Although we attempted to estimate right ventricular systolic pressure by determining the peak velocity of tricuspid regurgitation, reliable recordings at race finish were obtainable in only a few subjects and are, therefore, not reported.

*Doppler color flow examinations of tricuspid and mitral valvular regurgitation were performed in all possible views.* Using frame by frame review, we sought to identify turbulence arising proximal to each closed cardiac valve and persisting throughout the appropriate phase of the cardiac cycle. When present, the holosystolic duration of such turbulence and its high velocity (aliasing or velocity >1.5 m/s) were confirmed by pulsed Doppler studies. Color studies in all subjects were made using the same ultrasonograph with similar settings for gain, pulse repetition frequency and coding of turbulent flow.

*Quantitation of the severity of mitral and tricuspid regurgitation* was performed by digitizing the area of the maximal detectable flow disturbance or jet area (JA) and dividing it by the planar area (right or left atrial) of the receiving chamber (JA/LA or JA/RA), according to the method of Helmcke et al. (19).

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**Reproducibility measurements.** Reliability of echocardiographic and Doppler variables in the subjects under study has been examined previously by us (3) and shown to be quite acceptable. The relations between data obtained on different days are close, with correlation coefficients ranging from 0.980 to 0.997 and slopes from 1.01 to 1.03. Unfortunately, it is not possible to duplicate race finish conditions because repeated postrace studies would acquire data obtained under different physiologic conditions. Therefore, we could not obtain two data sets to be used in the evaluation of reproducibility of this case. Instead, a subset of race finish studies was digitized twice and yielded similar results; correlation coefficients for chamber areas ranged from 0.94 to 0.99 and slopes from 0.93 to 0.98.

**Statistical analysis.** Comparison of prerace, finish and recovery values was performed by one way repeated measures analysis of variance, with posthoc testing performed by the Newman-Keuls test (20,21). The relations among changes in cavity size, area shortening and inflow velocities were assessed by linear regression analysis. Data in text and tables are presented as mean values ±SD.

## Results

**Atrial and ventricular size, area and ejection fraction (Table 1).** Mean heart rate and systolic blood pressure for all data collections are listed in Table 1. After exercise, both right and left atrial sizes were reduced at end-systole but returned to normal at recovery. The percent change in area, or emptying fraction, tended to decrease at finish in both atria, but did not reach statistical significance in either. The right ventricle increased in size at race finish at both end-diastole and end-systole. During recovery, the right ventricle returned toward normal but remained significantly larger than at baseline. The emptying fraction, or percent change in area, did not change with exercise or recovery. In contrast to the right ventricle, the left ventricle decreased in size at end-diastole at race finish and was larger than at baseline during recovery. The percent change in left ventricular area, or emptying fraction, did not vary. The total area of all four cardiac chambers at end-diastole was unaffected by exercise or recovery, although at end-systole, it was slightly reduced at race finish and exceeded baseline value during recovery.

**Inflow patterns (Table 2).** The pattern of right ventricular inflow varied significantly after exercise and during recovery. Although the peak velocity of rapid filling (E) did not vary, atrial systolic flow velocity (A) increased significantly at race finish and returned to baseline value at recovery. As a result, there was a significant decrease in the tricuspid E/A ratio at race finish that returned to baseline value at recovery. The pattern of left ventricular filling also varied with exercise, although in a slightly different pattern. The peak velocity of rapid filling (E) was increased over baseline at both race finish and recovery. Similar to tricuspid inflow, the peak velocity of atrial systolic mitral flow was increased at race finish and returned to normal at recovery. In contrast to findings in the right side of the heart, the mitral E/A ratio was not significantly reduced at race finish, although it was augmented above baseline value at recovery.

**Mitral and tricuspid regurgitation (Table 3).** To assess the possible effects of valvular dysfunction as a contributor to the differences between right and left ventricular areas after

**Table 1.** Cavity Areas at Baseline, Race Finish and Recovery in 41 Athletes

| | Baseline ($cm^2$) | Race Finish ($cm^2$) | Recovery ($cm^2$) |
|---|---|---|---|
| Heart rate (beats/min) | 54 ± 7 | 84 ± 12* | 58 ± 7‡ |
| Systolic blood pressure (mm Hg) | 120 ± 15 | 117 ± 16 | 118 ± 16 |
| Right atrium | | | |
| Diastolic | 12.7 ± 3.0 | 12.5 ± 2.2 | 12.7 ± 2.9 |
| Systolic | 19.5 ± 3.3 | 18.2 ± 3.6† | 19.9 ± 4.4‡ |
| % emptying | 35 ± 12 | 29 ± 14 | 35 ± 12 |
| Right ventricle | | | |
| Diastolic | 21.4 ± 5.3 | 24.2 ± 5.7* | 22.1 ± 4.9†‡ |
| Systolic | 15.8 ± 4.4 | 18.2 ± 6.2* | 16.8 ± 5.0†‡ |
| % emptying | 25 ± 14 | 26 ± 13 | 24 ± 14 |
| Left atrium | | | |
| Diastolic | 12.0 ± 3.3 | 11.1 ± 2.4 | 11.6 ± 2.4 |
| Systolic | 19.4 ± 4.3 | 17.3 ± 3.6* | 19.6 ± 4.1‡ |
| % emptying | 38 ± 11 | 35 ± 10 | 39 ± 15 |
| Left ventricle | | | |
| Diastolic | 37.7 ± 6.9 | 36.3 ± 6.1* | 38.4 ± 6.3*‡ |
| Systolic | 25.5 ± 6.9 | 24.9 ± 5.9 | 26.3 ± 6.2†‡ |
| % emptying | 33 ± 9 | 32 ± 8 | 32 ± 10 |
| Total cavity area | | | |
| Diastolic | 84 ± 14 | 84 ± 13 | 83 ± 19 |
| Systolic | 81 ± 15 | 79 ± 14† | 83 ± 15*‡ |

†$p < 0.05$ and *$p < 0.01$ vs. baseline; ‡$p < 0.01$ recovery vs finish. Total cavity area = sum of all four cavity areas.

**Table 2.** Velocities of Right and Left Ventricular Inflow at Baseline, Race Finish and Recovery in 18 Atheletes

| | Baseline | Race Finish | Recovery |
|---|---|---|---|
| Tricuspid valve (cm/s) | | | |
| E | 37 ± 9 | 44 ± 10 | 45 ± 13 |
| A | 26 ± 7 | 38 ± 7* | 26 ± 12 |
| E/A | 1.56 ± 0.69 | 1.17 ± 0.23† | 1.91 ± 0.47‡ |
| Mitral valve (cm/s) | | | |
| E | 54 ± 10 | 69 ± 18* | 72 ± 19* |
| A | 38 ± 8 | 54 ± 11* | 44 ± 10‡ |
| E/A | 1.53 ± 0.36 | 1.33 ± 0.36 | 1.81 ± 0.61*‡ |

†$p < 0.05$ and *$p < 0.01$ race finish and recovery vs. baseline; ‡$p < 0.01$ recovery vs. race finish. A = atrial filling velocity; E = rapid filling velocity.

**Table 3.** Prevalence and Severity of Valvular Regurgitation at Baseline, Race Finish and Recovery in 21 Athletes

| | Baseline | Race Finish | Recovery |
|---|---|---|---|
| Tricuspid regurgitation | | | |
| Present | 18 (86%) | 11 (52%) | 16 (76%) |
| JA/RA | 15.7 | 17.1 | 14.3 |
| Pulmonary regurgitation | | | |
| Present | 3 (14%) | 0 (0%)* | 9 (43%) |
| Mitral regurgitation | | | |
| Present | 16 (76%) | 0 (0%)* | 10 (48%) |
| JA/LA | 5.8 | 0 | 4.6 |
| Aortic regurgitation | | | |
| Present | 3 (14%) | 3 (14%) | 3 (14%) |

*$p < 0.01$ vs. both baseline and recovery. JA/RA = maximal jet area to right atrial area ratio; JA/LA = maximal jet area to left atrial area ratio.

exercise, the prevalence and severity of regurgitation were assessed. Tricuspid regurgitation was commonly present at prerace study and its incidence was slightly but not significantly reduced at finish, returning to baseline during recovery. In patients with tricuspid regurgitation, the severity, as assessed by the maximal jet area to right atrial area ratio (19), was similar at all three data recordings. In contrast, mitral regurgitation, while also commonly present at baseline, was not found in any subject at race finish and the incidence returned toward baseline values at recovery. The average severity of mitral regurgitation was similar at baseline and recovery. Pulmonary regurgitation was found in a small number of subjects at baseline, was absent at race finish and was again detected at recovery. Aortic regurgitation was present in an identical number of athletes at all three data recordings.

*Overall changes in each chamber area, or in total cardiac cavity area,* did not correlate with exercise duration or with changes in left or right ventricular inflow patterns (all $r < 0.70$). Similarly, changes in each chamber area did not correlate with those in other chambers. Neither the presence nor the severity of regurgitation was related to chamber size, shortening or heart rate, or changes in these variables.

*Similarly, heart rate was not related to any mitral or tricuspid inflow variable* (E, A or E/A for each valve) nor were changes in heart rate from baseline to finish related to changes in any of these variables (all $r < 0.60$).

## Discussion

**Right versus left ventricular response to exercise.** Although the right ventricle, as well as the left, must alter its performance to meet the increased circulatory demands of exercise, its response has not been extensively studied. We found that the right ventricle responds differently to prolonged competitive exercise than does the left; the right ventricle enlarges rather than becoming smaller, its diastolic filling pattern is altered, displaying a greater dependence on atrial systole than does the left, and the incidence of tricuspid regurgitation does not vary, whereas that of mitral regurgitation decreases sharply.

**Pulmonary versus systemic circulation response to exercise.** The response of the pulmonary circulation to exercise appears to be different from that of the systemic circulation; although it is less well studied (8–12). Investigators (8,9) have reported 50% to 70% increases in systolic, diastolic and mean pulmonary artery pressures with brief (<15 min) exercise, with a mean pulmonary artery pressure of 52 mm Hg documented in a normal subject (12). At the same time, mean and diastolic systemic blood pressures increased much less (11% to 26%) and systolic pressure increased only moderately (23% to 45%). Exercise caused a smaller decrease, if any, in total pulmonary resistance than was found in the systemic circulation (0% to 30% reduction compared with 35% to 51%). Most importantly, right ventricular work was increased 3.6 to 5.2-fold, whereas during the same exercise, left ventricular work increased only 2.1 to 2.8-fold over baseline. The greater increase in hemodynamic load imposed on the right ventricle may explain the differing right and left ventricular responses to exercise.

**Right and left ventricular systolic function.** Because the assessment of systolic function may be misleading without consideration of afterload, our data cannot fully address changes in right ventricular systolic function. Unfortunately, the incidence of tricuspid regurgitation at race finish was low and the tricuspid regurgitation envelopes incompletely visualized so as to preclude estimates of right ventricular pressure in most athletes. The larger diastolic size (or increased preload) suggests that ejection fraction should increase. That shortening did not change while end-systolic area increased implies either an appropriate response to increased afterload or a reduction in contractility at race finish, paralleling the left ventricular systolic dysfunction observed after prolonged exhaustive exercise (3–7).

Although ischemia does not seem to be a factor (3), many contributors to apparent left ventricular dysfunction are identical in both circulations; a greater right ventricular responsiveness to such contributors would be needed if these were to provide the mechanism of the differing right and left ventricular changes after exercise (1–3,6). In addition, left ventricular function may be negatively affected by right ventricular enlargement through a positional shift of the interventricular septum or abnormal septal wall motion (22,23). Although right and left ventricular interactions are difficult to study under race conditions, the stability of total diastolic cardiac chamber area suggests reciprocal changes in right and left ventricular sizes, perhaps in an effort to minimize right ventricular work and increase cardiac efficiency (14).

**Right and left ventricular diastolic function.** Although Doppler indexes cannot provide a complete description of diastolic function, changes in peak velocity in rapid filling (E) height have been closely related to indexes of active relaxation such as tau and maximal rate of pressure decrease (−dP/dt max) (24), and a filling pattern similar to that seen after exercise has been associated with increased myocardial stiffness occurring with age and pressure overload hypertrophy (25,26). Although inflow velocities can be affected by physiologic conditions such as preload and heart rate, which were altered at race finish (27), these should have affected right and left ventricular filling similarly. Instead, the pattern of right but not left ventricular filling was altered.

**Right versus left AV valve regurgitation.** An additional area in which we found differences was that of atrioventricular valve regurgitation. The incidence and severity of tricuspid regurgitation were unchanged and right atrial size decreased, indicating that increased valvular dysfunction did not provide a volume load that could have contributed to right ventricular enlargement. The cause of different changes in valvular regurgitation is unclear. Factors such as reduced central blood volume secondary to dehydration and redistribution to muscle and skin (28) and higher heart rate at race finish (although still well within the normal range) should lead to similar reductions in regurgitation of both mitral and tricuspid valves. However, the known increase in pulmonary artery pressure with exercise might tend to increase tricuspid regurgitation as is seen in both acute and chronic pulmonary hypertension (29).

**Limitations of the study.** The restrictions imposed on our study by the nature of the exercise involved, the study subjects and the remote location are important. We cannot conclusively provide the mechanisms for the observed differences in right and left ventricular responses to exercise. Changes in right ventricular shape or compliance (pressure-volume relation) cannot be excluded, nor do the data address possible differences in right and left ventricular recovery times after exercise. Other methodologic considerations restricted the data collection because examination of valvular regurgitation and ventricular inflow velocities could not be performed in the same individuals. Equipment availability was limited and race finish studies were purposefully abbreviated to better approximate exercise rather than recovery conditions. Instead, studies were performed on two otherwise identical groups undergoing the same amount of exercise and with similar changes in cardiac size and function. Heart rate and blood pressure may also influence our findings; however, differences in these variables were small and well within the normal range.

Echocardiographic assessment of right ventricular size is difficult because of its complex geometric structure and inaccessibility to imaging (15). Rather than attempting to calculate exact right ventricular or stroke volumes and perhaps introducing errors by geometric assumptions, we chose to report all our data as they were obtained and, therefore, compared the planar areas of each of the cardiac chambers. Although we cannot exclude rotational errors, planar emptying fraction calculated from two-dimensional echocardiography correlates closely with ejection fraction obtained using nuclear and cineangiographic techniques (15,16).

**Conclusions.** Although the present study does demonstrate that the right and left ventricles respond differently to exercise, mechanisms are unclear and the roles of pericardial restraint or ventricular interaction conjectural. Additionally, changes in shape or compliance may be important. The differences in the right and left heart responses to exercise may assume clinical importance in some athletes and under extreme environmental conditions such as high altitude. Further study of the exercise physiology of the right heart chambers and the pulmonary circulation is necessary to fully understand their response to exercise in both health and disease.

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