Cardiac function following prolonged exercise: influence of age

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1Faculty of Physical Education and Health and 3Heart and Stroke/Richard Lewar Centre of Excellence, Faculty of Medicine, University of Toronto, Toronto, Canada; and 2Division of Cardiology, Mt. Sinai Hospital, Toronto, Canada

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Banks L, Sasson Z, Esfandiari S, Busato GM, Goodman JM. Cardiac function following prolonged exercise: influence of age. J Appl Physiol 110: 1541–1548, 2011. First published March 24, 2011; doi:10.1152/japplphysiol.01242.2010. —This study sought to determine the influence of age on the left ventricular (LV) response to prolonged exercise (PE; 150 min). LV systolic and diastolic performance was assessed using echocardiography (ECHO) before (pre) and 60 min following (post) exercise performed at 80% maximal aerobic power in young (28 ± 4.5 years; n = 18; mean ± SD) and middle-aged (52 ± 3.9 years; n = 18) participants. LV performance was assessed using two-dimensional ECHO, including speckle-tracking imaging, to determine LV strain (LV S) and LV S rate (LV SR). LV strain was used to determine the magnitude of this response. We observed a postexercise elevation in LV S (young: −19.5 ± 2.1% vs. −21.6 ± 2.1%; middle-aged: −19.9 ± 2.3% vs. −20.8 ± 2.1%; P < 0.05) and increase the magnitude of this response.

Diastolic function was reduced during recovery, including the LV SR ratio of early-to-late atrial diastolic filling (SRe/a), in young (2.35 ± 0.7 vs. 1.89 ± 0.2; P < 0.05) and middle-aged (1.51 ± 0.5 vs. 1.05 ± 0.2; P < 0.01) participants, as were conventional indices including the E/A ratio. Dobutamine stress ECHO revealed a postexercise depression in LV S in response to increasing dobutamine dose, which was similar in both young (pre-exercise dobutamine 0 vs. 20 μg·kg⁻¹·min⁻¹: −19.5 ± 2.1% vs. −27.2 ± 2.2%; postexercise dobutamine 0 vs. 20 μg·kg⁻¹·min⁻¹: −21.6 ± 2.1% vs. −23.7 ± 2.2%; P < 0.05) and middle-aged participants (pre: −19.9 ± 2.3% vs. −25.3 ± 2.7%; post: −20.8 ± 2.1% vs. −23.5 ± 2.7%; P < 0.05). This was despite higher noradrenaline concentrations immediately postexercise in both groups.

Dobutamine stress ECHO revealed a postexercise depression in LV S in response to increasing dobutamine dose, which was similar in both young (pre-exercise dobutamine 0 vs. 20 μg·kg⁻¹·min⁻¹: −19.5 ± 2.1% vs. −27.2 ± 2.2%; postexercise dobutamine 0 vs. 20 μg·kg⁻¹·min⁻¹: −21.6 ± 2.1% vs. −23.7 ± 2.2%; P < 0.05) and middle-aged participants (pre: −19.9 ± 2.3% vs. −25.3 ± 2.7%; post: −20.8 ± 2.1% vs. −23.5 ± 2.7%; P < 0.05). This was despite higher noradrenaline concentrations immediately postexercise in both groups.

We have demonstrated a direct link between the degree of LV impairment and exercise intensity (1), and there appears to be a similar relationship between the duration of PE and LV impairment (6, 55). However, little is known about the effect of increasing age on the manifestation of cardiac fatigue (20); middle-aged endurance athletes engaged in PE may be more vulnerable to exercise-induced cardiac fatigue, given the evidence of a reduction in cardiac β-adrenergic sensitivity with increasing age (24). Given the aging population and increased participation in prolonged endurance events in this age group (39), further information about the effects of aging on the LV response to PE is warranted. Therefore, the purpose of this study was to investigate the effects of age on the LV systolic and diastolic response to 150 min of PE in endurance-trained athletes. We hypothesized that middle-aged adults would exhibit a greater decline in systolic and diastolic performance secondary to a more profound β-adrenergic desensitization following PE.

METHODS

Participants. A total of 18 young (age range: 19–35) and 18 middle-aged participants (age range: 45–60) were recruited from advertisements posted in local running and triathlon clubs. Participants were free of medications and had no previous known history of cardiovascular disease. All participants had a long-standing history of endurance participation (>3 years). Informed, written consent was obtained from all participants prior to their participation. The study was approved by the University of Toronto and Mt. Sinai Hospital research ethics boards (Toronto, Canada) and was in full conformity with the Helsinki Declaration on the use of human participants.

General experimental design. Recruited participants initially underwent graded exercise testing in a controlled laboratory setting to determine maximal aerobic power (VO2max). Results were used to establish a running velocity eliciting a heart rate (HR) of ~80% VO2max during exercise, which is typically completed by endurance-trained athletes during training and/or recreational races. Cardiac function was assessed using two-dimensional (2D) echocardiography (ECHO) at baseline 60 min before the exercise session (pre) and postexercise following a 60-min recovery. Dobutamine stress ECHO (DSE) was used to interrogate β-adrenergic sensitivity pre- and post-PE. Blood analysis was performed to examine changes in both hematocrit (Hct) and catecholamines during PE.

Maximal exercise testing. Graded treadmill exercise testing (Lode BV. Groningen-Holland Medical Technology, The Netherlands) was performed to exhaustion to assess VO2max and maximal HR (HRmax). Ventilatory variables were collected online using a metabolic cart (Moxus Modular VO2 System, Applied Electrochemistry, Pittsburgh, PA), and HR was measured continuously (Polar 810i) and recorded electronically (HRTrak II heart rate tracker, Equilibrated Bio Systems, Smithtown, NY). A plateau in oxygen consumption, despite an increase in work rate, was used to determine if maximal effort was
achieved. Secondary measures for the attainment of VO2max included the attainment of the age-predicted HRmax and a respiratory exchange ratio of 1.15 or higher.

**PE protocol.** Participants completed a high-intensity (80% VO2max) exercise challenge involving 150 min of running. This velocity was verified prior to the challenge on a treadmill with HR monitoring for a period of 15 min. The HR was then confirmed by a track observer using data from the HR monitor, and the pace was fine-tuned accordingly. All participants completed their exercise on a supervised 200-m track in a controlled environment with temperature and humidity maintained at ~22°C and 40%, respectively. Participants were weighed on a calibrated scale (Health O Meter, Bridgewater, IL) to determine dry body mass. To minimize changes in hydration state and cardiac-loading conditions, participants were encouraged to consume ~200–300 ml of water every 20 min. Fluid consumption was recorded every 20 min.

**Recovery period.** Prior to postexercise ECHO measures, participants rested and rehydrated in a controlled laboratory environment for 60 min and were allowed to consume fluids ad libitum.

**ECHO.** 2D and Tissue Doppler Imaging (TDI) studies were conducted using an M3S probe on a commercial system (GE Vivid 7 and Imaging System, Version BT03–5, GE Healthcare, Canada) in accordance with the American Society of Echocardiography (28). Each participant was fitted with a standard 12-electrocardiographic lead configuration (Case 16 exercise testing system, Marquette Medical Systems, Milwaukee, WI) for HR assessment and ECHO gating. All ECHO images were acquired with the participant placed in the semiprone position (60° table elevation) on an imaging table, oriented in the left lateral decubitus position. All ECHO data were analyzed offline by a single trained observer, who was blinded to each stage of the dobutamine protocol, using a commercially available proprietary workstation (EchoPAC, Version 7, GE Healthcare). A minimum of three cardiac cycles was captured and averaged for analysis. M-mode and 2D ECHO images were obtained from the standard parasternal and apical windows. All system settings were adjusted to produce an optimal signal-to-noise ratio and endocardial delineation. M-mode ECHO views were used to measure LV morphology, including the LV internal dimension during diastole (LVIDd, mm), the interventricular septum during diastole (IVSd, mm), and the LV posterior wall thickness during diastole (LVPWd, mm). Apical, four chamber views were used to calculate LV end-diastolic (EDV; mL) and end-systolic volumes (ESV; mL) to determine ejection fraction (EF; %) using the single-plane Simpson’s method (28). Sphygmomanometer-determined systolic blood pressure (SBP) was obtained simultaneously to LV imaging to determine the systolic pressure volume ratio (PVR; mmHg/mL), which was calculated as the ratio between SBP and ESV; this was used as a surrogate measure of LV contractility (21). Longitudinal strain (S) and S rate (SR) were acquired from three cardiac cycles. An average of the six LV segments was calculated with a frame rate of 70 frames/s (11). Regional systolic and diastolic LV systolic function.

**Participant Characteristics**

All participants completed the entire protocol without any adverse events during or following exercise. Young participants included 12 males and six females and had a mean age of 28 ± 5 years and a VO2max of 55 ± 6.8 ml·kg⁻¹·min⁻¹. Middle-aged participants included 15 males and three females and were 52 ± 3.8 years of age with a VO2max of 47 ± 7.2 ml·kg⁻¹·min⁻¹. LV morphology was similar among young and middle-aged participants (LVIDd: 4.9 ± 0.2; LVPWd: 0.9 ± 0.3 mm; IVSd: 1.2 ± 0.4 vs. 4.9 ± 0.4 mm; LVPWd: 0.9 ± 0.2 vs. 1.0 ± 0.2; P > 0.05 for all). All participants presented with normal ventricular morphology, resting function, and VO2max values typical of well-trained, recreational-endurance athletes. There were no significant sex differences in measures of systolic and diastolic function; therefore, a single-group analysis with sexes combined was completed.

**Cardiovascular Response to PE**

Changes in cardiovascular variables observed pre- and post-PE are shown in Table 1.

**Blood pressure.** Middle-aged participants had a higher resting SBP (P = 0.006) and diastolic blood pressure (DBP; P < 0.01) and demonstrated a greater postexercise hypotension (ΔSBP = −15 mmHg vs. −5 mmHg; ΔDBP = −10 mmHg vs. −2 mmHg) compared with young participants.

**LV systolic function.** Baseline and postexercise LV EF was significantly lower in the young group compared with the
middle-aged participants and remained unchanged following exercise in both cohorts (P < 0.01). Higher resting LV EF was related to higher resting SBP (r = 0.34; P < 0.04) and DBP (r = 0.37; P < 0.03) in the middle-aged participants relative to the younger participants. Changes in global LV S were similar at baseline and elevated following PE (P > 0.05) for the young (−19.5 ± 2.1% vs. −21.6 ± 2.1%) and the middle-aged participants (−19.9 ± 2.3% vs. −20.8 ± 2.1%; P < 0.05). Similar trends were observed for global LV SR young: −1.19 ± 0.1 vs. −1.37 ± 0.2; middle-aged: −1.20 ± 0.2 vs. −1.38 ± 0.2; P < 0.05) following PE.

LV diastolic function. Data for LV diastolic function pre- and postexercise are summarized in Table 2. Baseline E/A, E’/A’ septal, E’/A’ lateral, and LV SRe/a were lower in the middle-aged relative to the young participants (P < 0.001); these diastolic indices were reduced in both cohorts postexercise.

Table 2. Diastolic function following prolonged strenuous exercise by age

<table>
<thead>
<tr>
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<th>Pre-exercise</th>
<th>Postexercise</th>
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<tbody>
<tr>
<td></td>
<td>Young</td>
<td>Old</td>
</tr>
<tr>
<td>LV SRe</td>
<td>1.88 (0.24)</td>
<td>1.65 (0.30)*</td>
</tr>
<tr>
<td>LV SRe/a</td>
<td>0.84 (0.20)</td>
<td>0.71 (0.26)*</td>
</tr>
<tr>
<td>LV E (m/s)</td>
<td>2.35 (0.64)</td>
<td>1.51 (0.48)*</td>
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<tr>
<td>LV E/A</td>
<td>0.68 (0.12)</td>
<td>0.63 (0.15)</td>
</tr>
<tr>
<td>LV E/A</td>
<td>0.36 (0.06)</td>
<td>0.48 (0.10)*</td>
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<tr>
<td>LV E’ (m/s)</td>
<td>1.92 (0.38)</td>
<td>1.38 (0.34)*</td>
</tr>
<tr>
<td>LV E’ septal</td>
<td>−11.35 (1.58)</td>
<td>−7.90 (1.50)*</td>
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<tr>
<td>LV E’ septal</td>
<td>−5.44 (1.25)</td>
<td>−7.06 (1.83)*</td>
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<tr>
<td>LV E’ septal</td>
<td>−2.62 (0.52)</td>
<td>−3.92 (0.35)*</td>
</tr>
<tr>
<td>LV E’ lateral</td>
<td>−13.13 (1.61)</td>
<td>−9.29 (1.35)*</td>
</tr>
<tr>
<td>LV E’ lateral</td>
<td>−4.29 (1.83)</td>
<td>−6.39 (1.97)*</td>
</tr>
<tr>
<td>LV E’ lateral</td>
<td>3.56 (1.35)</td>
<td>1.64 (0.57)*</td>
</tr>
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Values represent mean (SD). *P < 0.05 between-group comparison of young vs. old; †P < 0.05 within group comparison of pre- vs. postexercise. LV E, left ventricular early filling phase; LV A, left ventricular late atrial filling; LV E/A, left ventricular early-to-late atrial filling ratio; LV E’, early peak myocardial tissue velocity (cm/s); LV A’, late atrial peak myocardial tissue velocity (cm/s); LV E’/A’, left ventricular early-to-late myocardial tissue velocity ratio.
cise \( (P < 0.001) \). Also, similar reductions in diastolic SR (LV SRe/a) were observed in young \( (P < 0.01) \) and middle-aged participants \( (P < 0.001) \).

**LV volumes** (Table 1). Changes in EDV were not significantly different between young and middle-aged at baseline and remained unchanged postexercise \( (P > 0.05) \). Baseline and postexercise ESV was higher in the middle-aged compared with the young participants \( (P < 0.001) \), and ESV did not significantly change from baseline in either group \( (P > 0.05) \). Pre-exercise SV was significantly higher in the middle-aged relative to the young group \( (P = 0.03) \). Whereas changes in SV did not change in the young participants, they were significantly reduced in the middle-aged following PE \( (P = 0.04) \). There was no change in pre-PE vs. post-PE Hct in the young \( (42.5 \pm 2.4 \text{ vs. } 42.4\% \); \( P = 0.79) \) or middle-aged \( (41.7 \pm 4.1\% \); \( P = 0.54) \) groups, with no age differences observed at baseline \( (P = 0.46) \) and during recovery \( (P = 0.32) \). Body mass was reduced similarly in both young and middle-aged during exercise (young: \( 66 \pm 9 \text{ vs. } 65 \pm 8 \text{ kg, } P < 0.01 \); middle-aged: \( 63 \pm 4 \text{ vs. } 61 \pm 4 \text{ kg, } P < 0.01 \)), but pre-exercise body mass was restored prior to recovery ECHO analysis (young: \( 66 \pm 9 \text{ kg} \); middle-aged: \( 62 \pm 4 \text{ kg, } P > 0.05 \)). Notably, the change in systolic or diastolic LV S or SR was unrelated to the changes in Hct, EDV, or body mass \( (P > 0.05) \).

**Catecholamines.** Baseline NA was higher in the middle-aged relative to the young cohort \( (P = 0.012) \) and remained higher during and following exercise \( (P < .01) \). However, there was no interaction effect, as NA remained elevated during and following PE in both middle-aged \( (P < .05) \) and young participants \( (P > .05) \). Plasma NA was not correlated to the changes in the LV S \( (r = 0.10; P = 0.60) \), LV SR \( (r = 0.17; P = 0.35) \), and LV SRe/a \( (r = -0.14; P = 0.43) \) from pre-PE to post-PE.

**Dobutamine stress** (Table 1). Changes in cardiovascular variables observed pre- and post-PE are shown in Table 2. A similar post-PE depression in systolic performance was observed in response to dobutamine stress as depicted in dose-response curves for LV EF (Fig. 1) and LV S (Fig. 2) in each group, regardless if expressed in absolute values (Table 1) or when expressed as a percentage change from baseline (Figs. 1 and 2). We failed to observe a significant percentage change from baseline for LV SR among middle-aged participants (Fig. 3). There were no changes observed following PE in any index of diastolic function (including E/A, SRE/A) at any dobutamine dose.

**DISCUSSION**

To our knowledge, this is the first study to directly examine the influence of age on LV systolic and diastolic performance following PE. Our findings confirm that PE can induce changes in LV...
systolic and diastolic function across a wide age span with both young and middle-aged participants, demonstrating a similar reduction in exercise-induced β-adrenergic receptor sensitivity.

**LV Systolic Function**

Recent speckle-tracking studies have shown a decline in LV S and/or LV SR following recovery from PE (17, 35), yet a recent study also failed to observe any decline in LV S (25). In contrast to these observations, we observed an increase in resting LV S following recovery from PE in both groups, with little change in LV EF. Disparate observations are likely due to variations in assessment conditions seen in field studies (vs. 60 min postexercise in the present study) and the uncontrolled nature of the exercise stress. As expected, we observed elevated plasma catecholamines during exercise, and while attenuated during recovery, they remained significantly higher than baseline measures after 60 min of recovery. This may have contributed to enhance inotropic and chronotropic stimulation under more favorable cardiac-loading conditions during recovery, as demonstrated by a profound postexercise hypotension that would lower afterload (Table 1). We did not use atropine during dobutamine stress, and unlike isoproterenol, dobutamine is highly specific to β1-adrenergic receptors, with only modest chronotropic effects compared with significant inotropic stimulation. Yet, chronotropic stimulation remained postexercise, likely due to the residual increases in catecholamines, core temperature, and modest dehydration. We also reported a higher resting EF and PVR among the middle-aged relative to the younger participants. This finding is common in middle-aged cohorts (38) and may be due to a hyperkinetic cardiac output secondary to higher resting SBP (15). This may reflect the need for an elevated cardiac output to compensate for increased arterial stiffness with age (26). Nonetheless, elevated EF occurred despite similar LV morphology and other measures of systolic function, including LV S and LV SR. EF and PVR also remained unchanged postexercise in response to dobutamine stress, which may, in part, be due to loading and HR dependency (40), respectively.

Our present data are consistent with prior studies of young participants, which reported a reduction in β-adrenergic receptor sensitivity regardless of exercise intensity (1). The present data indicate that increased age does not amplify the degree of exercise-induced β-adrenergic desensitization, despite the finding that middle-aged participants had higher catecholamine concentrations during exercise at the same relative intensity compared with their young counterparts. Age-related decreases in the percentage of β-adrenergic receptors in the high-affinity binding state, in addition to decreased G protein-mediated signal transduction, have been reported in aged human heart tissue (53), yet both young and middle-aged participants demonstrated a preservation of systolic function at rest. Cardiac adaptations in middle-aged endurance athletes may also influence systolic performance through the maintenance of cardiac output (38, 43) and β-adrenergic sensitivity (29), thereby attenuating the age-related declines in LV systolic function. Middle-aged endurance athletes are also known to have a greater reliance on the Frank-Starling mechanism during exercise, with a significant hemodynamic response characterized by an increase in EDV and SV (43). These hemodynamic adaptations in endurance athletes are characterized by mild LV hypertrophy (4, 46) and dilation induced by chronic volume overload (14, 43, 45), whereby an increased SV at peak exercise may mitigate the decline in cardiac output and systolic performance seen with increasing age. This is supported by observations that resting systolic function (including LV S) is maintained in both younger and middle-aged endurance athletes given the LV morphological adaptations to exercise (4, 46).

Systolic impairment following PE was only evident during dobutamine stress, with a significant decay in LV EF and S observed for both groups (Figs. 1 and 2). Prior studies have reported that the reduced β-adrenergic receptor responsiveness with elevated catecholamine levels during PE (12, 22, 41, 51, 52) is associated with reductions in myocardial contractility (17, 20, 41, 51). These findings are similar to those seen in canine-pacing models used to induce cardiomyopathy, notably tachycardia-induced declines in β-adrenergic receptor sensitivity (56). Both of these models demonstrate that acute down-regulation of β-adrenergic receptor sensitivity (16, 27) may be linked to chronically elevated catecholamine levels that lead to blunting of sympathetic activation (3). Notwithstanding, it is also possible that a reduced inotropic state is secondary to changes occurring downstream of the receptor itself.

**LV Diastolic Function**

To date, declines in diastolic function have been reported following PE in a mixed group of participants across the
lifespan (7, 23, 35, 36). Endurance training is known to enhance early diastolic filling at rest and during exercise. However, middle-aged athletes display significantly lower early diastolic filling patterns compared with young athletes (30), which is in agreement with our present data (Table 1). Normal physiological aging of the LV also includes a reduction in LV diastolic compliance and an increase EDV (14, 33, 38, 43, 47). There is general agreement that reduced contractility with aging is offset by an increased reliance on the Frank-Starling mechanism to maintain stroke volume at rest (14, 38), yet few data are available for exercise. The present data show declines in LV diastolic function regardless of age following PE. Similar to George et al. (17), we found a decline in LV SR_{c/a} following PE, which suggests impairment of ventricular relaxation. While they observed this after an ultra-marathon distance, we have identified similar results following an exercise duration (i.e., 150 min) more commonly performed by recreational athletes. We also found that diastolic function was not influenced by changes in preload, as LV SR_{E/A} was unrelated to EDV (P > 0.05). The mechanisms for this relaxation impairment remain speculative. Changes in LV chamber stiffness and calcium handling may have contributed to the reduction in both systolic and diastolic function. It has been suggested that diastolic cardiac fatigue may involve a delayed diastolic decline in cytosolic calcium concentration resulting from altered myofilament calcium ion (Ca^{2+}) sensitivity and/or decreased calcium ATPase in the sarcoplasmic reticulum (42). Furthermore, a short-term cardiomyocyte-pacing model has also demonstrated that intracellular Ca^{2+} concentration dynamics are not influenced by age (31), supporting our observations of impairment across a wide age span. The clinical relevance of these finding is unknown but warrants further investigation.

This study extends our knowledge of the physiological mechanisms contributing to exercise-induced cardiac fatigue. Moreover, it serves to illustrate some of the challenges of interpreting LV performance following exercise. Evidence supporting exercise-induced cardiac fatigue is challenged by the fact that postexercise cardiac function is influenced by changes in numerous factors, including preload, afterload, and catecholamines (20). In fact, our preliminary postexercise ECHO findings, made following 60 min of recovery and rehydration, revealed no changes in resting cardiac systolic but reduced diastolic performance. Cardiac interrogation using DSE demonstrates a reduction in β-adrenergic sensitivity following PE. While transient β-adrenergic desensitization may contribute to the declines in cardiac function, load-independent indices of LV function obtained during exercise may further reveal the effects of PE in future studies.

Limitations

Despite measures to control fluid intake during exercise and recovery, we could not fully control for potential changes in LV preload. Fluid intake was monitored during exercise and recovery, contributing to a fully restored Hct and only modest reductions in EDV and body mass, which indirectly reflect preload status. Notably, changes in LV S, LV SR, and LV SR_{E/A} were unrelated to changes in EDV and body mass. These efforts to control fluid status limited variations in preload compared with greater control of preload than field studies and may have mitigated, to some extent, the influence of loading conditions on LV systolic and diastolic function. A load-independent measure of contractility using noninvasive techniques remains elusive. Speckle-tracking-derived measures of S and SR are more sensitive measures of systolic and diastolic performance than traditional indices (i.e., EF) (2, 13, 19, 32). Notwithstanding, measures of SR and in particular, S are load-dependent (5, 50); however, corroborating data are provided by measures of the systolic pressure/ESV ratio, an accepted surrogate measure of contractility that has been used during dobutamine stress (21). The current study did not examine radial and circumferential S and SR, as these measures exhibit larger variability (37), particularly those obtained distal to the mitral valve plane. While our study included both male and female participants, the small number of middle-aged female participants precluded adequate power to comment on possible gender and age interactions and may have limited our ability to detect age-related catecholamine changes following PE. Finally, previous studies have indicated that the duration of exercise is related to the extent of cardiac fatigue (6); consequently, we chose to isolate the effects of exercise intensity and duration, precluding any control for the “volume” of exercise per se, which may have resulted in a greater exercise stimulus (running distance) in the younger cohort.

Conclusion

PE in both young and middle-aged recreational athletes induces a depression in the LV EF and S responses to dobutamine stress following exercise, indicative of a reduction in β-adrenergic sensitivity. A reduction in diastolic function following exercise was also observed for both young and middle-aged participants, suggesting that increasing age does not increase the likelihood of developing or the extent of LV dysfunction observed following PE.

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DISCLOSURES

No conflicts of interest, financial or otherwise, are declared by the author(s).

REFERENCES


J Appl Physiol • VOL 110 • JUNE 2011 • www.jap.org
9. Lang RM, Bierig M, Devereux RB, Flachskampf FA, Foster E, Pellikka PA, Picard MH, Roman MJ, Seward J, Skorton DJ, St John Sutton M, Tajik AJ. Recommendations for chamber quantification: a report from the American Society of Echocardiography’s Guidelines and Standards Committee and the Chamber Quantification Writing Group, developed in conjunction with the European Association of Echocardiography, a branch of the European Society of Cardiology. J Am Soc Echocardiogr 18: 1440–1463, 2005.


40. Tulloh L, Robinson D, Patel A, Ware A, Prendergast C, Sullivan D, Pressley L. Raised troponin T and echocardiographic abnormalities after...


