Lifestyle Change Diminishes a Hypertensive Response to Exercise in Type 2 Diabetes

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ABSTRACT

SCHULTZ, M. G., M. D. HORDERN, R. LEANO, J. S. COOMBES, T. H. MARWICK, and J. E. SHARMAN. Lifestyle Change Diminishes a Hypertensive Response to Exercise in Type 2 Diabetes. *Med. Sci. Sports Exerc.*, Vol. 43, No. 5, pp. 764–769, 2011. **Purpose**: A hypertensive response to exercise (HRE) is common in patients with type 2 diabetes and is associated with increased left ventricular (LV) mass and mortality. This study aimed to determine whether lifestyle modification would improve exercise blood pressure (BP) and reduce LV mass in patients with type 2 diabetes. **Methods**: One hundred and eighty-five patients with type 2 diabetes were randomized to 1 yr of lifestyle intervention (n = 97, mean ± SD age = 54.7 ± 11.3 yr, 51% men) or usual care (control; n = 88, age = 53.8 ± 8.1 yr, 61% men). Brachial BP was measured at rest and during a graded maximal exercise test at baseline and 1 yr. Patients also underwent two-dimensional echocardiography to determine LV dimensions. A subgroup of 61 patients had resting and exercise central BP estimated from radial tonometry. An HRE was defined as a maximal exercise systolic BP of ≥210 mm Hg for men and ≥190 mm Hg for women. **Results**: At study entry, there were 101 patients (55%) with an HRE (n = 51 controls). Compared with controls, lifestyle intervention significantly reduced the propensity to develop an HRE in those participants who did not have HRE at baseline (29.8% vs 59.5%, P = 0.006). However, absolute values of exercise and resting (brachial and central) BP and LV mass were not significantly changed (all P values > 0.05). There were significant (all P values < 0.05) improvements in $\dot{V}O_{2max}$, body mass index, plasma glucose, insulin resistance, and HDL cholesterol after lifestyle intervention compared with control. **Conclusions**: Lifestyle intervention significantly attenuates the development of an HRE but does not reduce cardiac size after 1 yr in patients with type 2 diabetes. **Key Words:** LIFESTYLE MODIFICATION, EXERCISE, BLOOD PRESSURE, HYPERTENSION

Brachial blood pressure (BP) at rest is a potent predictor of cardiovascular morbidity and mortality. However, there is evidence to suggest that a hypertensive response to exercise (HRE) also independently predicts morbidity (including development of hypertension) and mortality (12,17,18,20,24). We recently reported that patients with type 2 diabetes mellitus have a high prevalence of an HRE (51%) and that this abnormal exercise response was associated with increased left ventricular (LV) relative wall thickness (RWT) and increased exercise central BP (22). These findings are likely to be of clinical significance because LV RWT predicts cardiovascular outcome in patients with type 2 diabetes (7). It has been shown that the cardiovascular and metabolic risk profile of patients with type 2 diabetes can improve after lifestyle change (11). Improved risk may also be achieved via vascular adaptations such as increased large artery compliance (4), reduced augmentation index (a marker of central systolic loading) (6), and improved conduit and resistance vessel endothelial function (16). These adaptations may expect to lower central BP and reduce cardiac size through improved ventricular–vascular interaction. Accordingly, the aim of this study was to determine the effect of lifestyle modification on resting and exercise BP (brachial and central) as well as LV mass in patients with type 2 diabetes.

METHODS

Participants and study design. This was a randomized controlled trial in a cohort of 248 otherwise healthy patients with type 2 diabetes (aged 24–75 yr), recruited via a local community advertisement program. Results of the main study relating to myocardial function were reported recently (Trial registration.anzctr.org.au; Identifier: ACTRN 12607000060448) (10), and this current report is an analysis of the exercise BP data. Inclusion criteria included a negative stress echocardiograph in which patients also needed to achieve an exercise HR ≥ 85% of age predicted maximum HR (220 − age × 0.85). Twenty-five patients returned a positive test and were excluded, leaving 223 patients who were randomized into either a program involving

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positive lifestyle changes (intervention, n = 112) or usual care (control, n = 111). From this cohort, 38 patients had no exercise BP data available, leaving 185 patients (control, n = 88; lifestyle intervention, n = 97; see Figure 1 for flow of study participants). From our previous report on exercise BP in patients with type 2 diabetes (22), with 88 patients per group, we can detect a between-group difference of 8.6 mm Hg in exercise systolic BP (SBP) and correlations of r = 0.291, with α = 0.05, β = 0.2. A subgroup of 61 patients (n = 30 controls) had central BP measured by radial tonometry at rest and after maximal exercise. From our previous exercise reproducibility study (9), with 30 patients we can detect a between-group difference of 10.3 mm Hg in exercise central SBP (with α = 0.05, β = 0.2). All measures were performed at baseline and 1 yr later. Patients randomized to usual care received standard medical advice through their general practitioners and the Diabetes Clinic at the Princess Alexandra Hospital. All participants gave their informed written consent to participate, and ethics approval was obtained from The Princess Alexandra Hospital and the University of Queensland. All investigations were carried out in accordance with the principles of the Declaration of Helsinki.

Lifestyle intervention—exercise component. Patients randomized to the intervention received a formal exercise training program in addition to standard care. The exercise program aimed to achieve a minimum of 150 min·wk^{-1} of at least moderate-intensity exercise and was delivered in two stages over the year. Stage 1 (initial 4 wk) of the exercise intervention consisted of two gym-based training sessions (1 h each) of combined moderate-intensity aerobic and resistance training per week. These sessions were prescribed and supervised by accredited exercise physiologists, and patients were asked to perform a minimum of 30 additional minutes of exercise at home. The exercise programs were individually tailored, taking into consideration the fitness level, comorbidities, and exercise preferences of each individual.

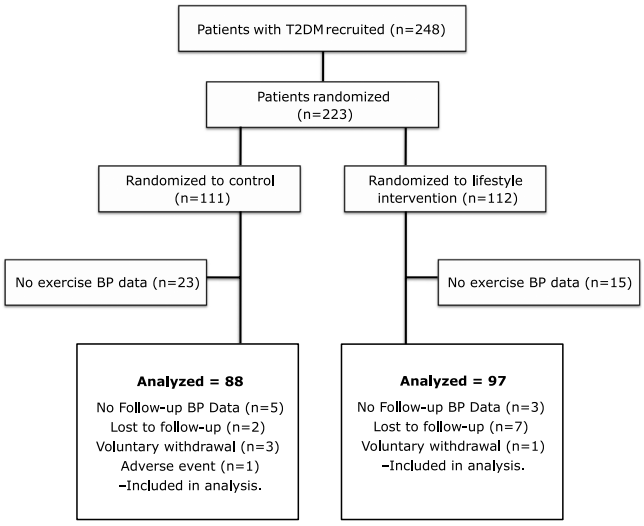

FIGURE 1—Flow of participants through the study. BP, blood pressure; T2DM, type 2 diabetes mellitus.

Moderate intensity was defined as a rating of perceived exertion of 12–13 on the Borg 20-point scale. Stage 2 (in the remaining 11 months) involved a home-based, self-directed diet and exercise program consisting of individually tailored exercise, including moderate-intensity walking, jogging, swimming, and/or resistance training. Weekly telephone follow-up was initiated to provide advice and to check for compliance to the program for the first 3 months, then every 2 wk until 6 months, and monthly thereafter.

Lifestyle intervention—dietary component. Patients randomized to the intervention also received some basic dietary advice in addition to exercise training. The dietary component of the intervention included individually tailored advice aimed at a 7% decrease in body weight. Qualified dieticians assessed the dietary composition of each participant at baseline, followed by monthly review sessions for 12 months. The dietary modifications aimed to achieve an intake of 15% protein, 45%–50% carbohydrates, <35% fat per day, with a 1:1:1 ratio of polysaturated, monosaturated, and saturated fat, respectively.

Protocol for cardiovascular examination. All participants attended the hospital on the morning after an overnight fast. Before attendance, participants were asked to avoid heavy exercise (within 24 h) and to refrain from caffeine and smoking (within 4 h). A blood sample was obtained, and biochemistry was performed by standard laboratory techniques (Queensland Health Pathology Service, Brisbane, Australia). Supine resting brachial and central BP measures were recorded after at least 5 min of rest. Resting echocardiography measures were then taken, after which subjects performed a treadmill exercise stress test to volitional fatigue. A breath-by-breath analysis of expired gas for maximal expired oxygen ($\dot{V}O_{2max}$) was also obtained (V29C SensorMedics, Yorba Linda, CA). Brachial BP was recorded at each stage, including the final minute of the exercise test, which was recorded as the maximal brachial BP. When the exercise test was terminated, the patient was quickly moved to the supine position in a bed located next to the treadmill. Central BP was then immediately recorded (<2 min) by radial tonometry.

Hemodynamics. The average of two BP measures recorded by mercury sphygmomanometer (Baumanometer; W.A. Baum Co., New York, NY) was used as the baseline brachial BP value. Central BP was estimated via radial applanation tonometry (SphygmoCor 7.01; AtCor Medical, Sydney, Australia), using a validated generalized transfer function (23), with good reproducibility during exercise (9). Resting radial waveforms were calibrated with the average brachial SBP and diastolic BP (DBP) acquired at a similar time (within ≈1 min). Postexercise radial waveforms were calibrated with the brachial BP at peak exercise. An HRE was defined as brachial SBP of ≥210 mm Hg for men and ≥190 mm Hg for women, in accordance with previous work (15,22). Pulse pressure was calculated by subtracting DBP from SBP. Augmentation index was derived from the difference between central P_2 and P_1, expressed as a percentage of pulse pressure.

Amplification of pulse pressure was defined as the ratio of peripheral (brachial) to central pulse pressure.

Echocardiography. Measures of LV dimensions were only available for this analysis in 162 patients (80 controls). Data were recorded via two-dimensional targeted M-mode echocardiography using a standard ultrasound machine (Vivid 7; GE Vingmed, Horten, Norway), with a 2.5-MHz phased array probe according to the American Society of Echocardiography protocol. LV mass was indexed to height$^{2.7}$ (LVMI g·m$^{-2.7}$), and LV RWT was calculated as the ratio of 2 × posterior wall thickness to LV diameter, each at end diastole. LV hypertrophy was defined from Lang et al. (14) as LV mass index of >55 g·m$^{-2.7}$ for men and >51 g·m$^{-2.7}$ for women.

Statistical analysis. All data were analyzed using the Statistical Package for the Social Sciences for Windows (Version 17.0; SPSS Inc., Chicago, IL). All variables were tested for normality of distribution, using the Kolmogorov–Smirnov measure and/or the Q-Q distribution plots and log transformed if required. Independent *t*-tests were used to compare the differences at baseline and for the change after 1 yr between the control and the intervention. ANCOVA was additionally undertaken to assess between-group differences, correcting for baseline differences or mean arterial pressure as indicated in the tables. Categorical variables were compared by the chi-square test for independence. Pearson product moment correlations were performed to assess relationships between variables. $P < 0.05$ was considered significant.

RESULTS

Clinical characteristics. At baseline, there were 101 patients (55%) with an HRE (control, $n = 51$; intervention, $n = 50$), with no significant differences for age (54.5 ± 8.1 vs 55.4 ± 11.3 yr) or sex ($n = 48$ men vs $n = 49$ men) between the control and the intervention groups, respectively ($P > 0.05$ for both). Mean duration of diabetes was not significantly different between the intervention and the control groups. (5.8 ± 6.4 vs 5.9 ± 5.8, respectively, $P > 0.05$), nor was the presence of hypertension (brachial BP ≥140/90 mm Hg) at baseline (41% intervention vs 30% controls, $P > 0.05$). There were also no significant differences in the number of patients at baseline with renovascular disease ($n = 8$ controls vs $n = 6$ intervention), diabetic retinopathy ($n = 6$ controls vs $n = 6$ intervention), neuropathy ($n = 5$ controls vs $n = 8$ intervention), deep vein thrombosis ($n = 0$ controls vs $n = 1$ intervention), peripheral vascular disease ($n = 0$ controls vs $n = 2$ intervention), or atrial fibrillation ($n = 1$ control vs $n = 2$ intervention), all P values > 0.05. The clinical and metabolic parameters of patients at baseline and 1 yr are compared in Table 1. At baseline, the control group had a greater $\dot{V}O_{2max}$ and LV RWT compared with participants in the intervention group ($P < 0.05$). There were no significant differences at baseline between the number of patients with LV hypertrophy ($n = 34$ controls vs $n = 28$ intervention, $P > 0.05$), nor any other clinical or metabolic variable ($P > 0.05$). After 1 yr of lifestyle intervention, there was a significantly greater improvement in $\dot{V}O_{2max}$, body mass index, plasma glucose, homeostasis model of insulin resistance ($HOMA_{IR}$), and HDL cholesterol compared with controls (all P values < 0.05). LV mass index and LV RWT failed to improve beyond that of the control group after lifestyle intervention ($P > 0.05$). The number of patients with LV hypertrophy after 1 yr did not significantly differ between groups ($n = 34$ controls vs $n = 38$ intervention, $P > 0.05$). In addition, the percentage of patients taking antihypertensive, lipid lowering, and/or medications for hyperglycemia did not differ between groups at baseline or 1 yr. Furthermore, the use of these

TABLE 1. Clinical and metabolic parameters of the study population at baseline and change over 12 months.

	Control ($n = 88$)			Intervention ($n = 97$)			
	Baseline, Mean ± SD or %	12 Months, Mean ± SD or %	Change, Mean ± SD or %	Baseline, Mean ± SD or %	12 Months, Mean ± SD or %	Change, Mean ± SD or %	*P*
Body mass index (kg·m^{-2})	31.5 ± 6.0	31.7 ± 5.9	0.2 ± 2.0	32.1 ± 5.4	31.6 ± 5.4	−0.5 ± 1.9	0.024
$\dot{V}O_{2max}$ (mL·kg^{-1}·min^{-1})	24.1 ± 7.0	24.2 ± 7.1	0.3 ± 4.9	21.0 ± 5.9	23.1 ± 6.6	2.1 ± 4.7	0.020[a]
Left ventricular mass index (g·m$^{-2.7}$)	50.4 ± 16.2	50.6 ± 14.9	0.1 ± 13.9	49.7 ± 18.5	51.7 ± 28.6	1.4 ± 14.7	0.556
Left ventricular RWT (ratio)	0.48 ± 0.13	0.44 ± 0.09	−0.04 ± 0.12	0.44 ± 0.10	0.44 ± 0.09	0.004 ± 0.10	0.314[a]
Total cholesterol (mmol·L^{-1})	4.84 ± 1.01	4.72 ± 1.03	−0.12 ± 0.74	4.88 ± 1.02	4.86 ± 1.10	−0.02 ± 0.85	0.438
Triglycerides (mmol·L^{-1})	1.75 ± 1.32	1.95 ± 1.76	0.20 ± 1.59	2.08 ± 2.49	2.26 ± 3.47	0.16 ± 1. 98	0.112
HDL cholesterol (mmol·L^{-1})	1.39 ± 0.43	1.38 ± 0.43	−0.01 ± 0.29	1.35 ± 0.36	1.45 ± 0.46	0.07 ± 0.33	0.043
Glycosylated HbA_{1c} (%)	7.54 ± 1.43	7.82 ± 1.66	0.28 ± 1.38	7.52 ± 1.48	7.52 ± 1.50	−0.08 ± 1.21	0.089
Glucose (mmol·L^{-1})	8.28 ± 2.73	8.97 ± 3.01	0.69 ± 2.45	8.56 ± 3.20	8.31 ± 8.46	−0.28 ± 2.78	0.003
$HOMA_{IR}$	5.48 ± 5.07	7.14 ± 9.97	1.60 ± 7.62	6.00 ± 4.12	5.43 ± 4.68	−0.48 ± 4.05	0.001
Medications (%)							
ACEi/ARB	39	44	6	45	46	5	0.818
Calcium channel blocker	7	7	2	7	7	2	0.886
Diuretic	3	3	0	4	4	2	0.201
Statin	36	40	8	43	43	7	0.839
Thiazolidinedione	2	3	3	5	6	3	0.897
Sulfonylurea	27	25	3	29	28	5	0.590

Data are presented as mean ± SD.

$HOMA_{IR}$, homeostasis model assessment of insulin resistance; RWT, relative wall thickness; ACEi, angiotensin converting enzyme inhibitor; ARB, angiotensin receptor blocker.

[a] Data were significantly different between groups at baseline ($P < 0.05$); therefore, data have been ANCOVA corrected for baseline values. Left ventricular mass index and LV wall thickness were recorded in 162 patients only (80 controls). $HOMA_{IR}$ was only available in 130 patients (62 controls). Significance is $P < 0.05$.

TABLE 2. Blood pressure parameters between control and intervention groups at baseline and 12 months.

	Controls (n = 88)			Intervention (n = 97)			
	Baseline	12 Months	Change	Baseline	12 Months	Change	P Value Between-Group Change
Rest							
Brachial SBP (mm Hg)	129 ± 15	128 ± 15	−1 ± 14	134 ± 17	133 ± 18	−2 ± 14	0.746[a]*
Brachial DBP (mm Hg)	80 ± 8	79 ± 9	−1 ± 9	83 ± 10	80 ± 9	−3 ± 9	0.775[a]*
Brachial PP (mm Hg)	48 ± 11	48 ± 12	0.3 ± 11	52 ± 14	53 ± 15	1.1 ± 14	0.605[a]*
HR (bpm)	82 ± 14	80 ± 13	−2 ± 21	85 ± 15	81 ± 14	−4 ± 21	0.625*
Mean arterial pressure (mm Hg)	97 ± 10	96 ± 10	−1 ± 9	100 ± 11	98 ± 10	−2 ± 9	0.986[a]*
Central SBP (mm Hg)	125 ± 18	125 ± 17	0.3 ± 17	126 ± 19	123 ± 14	−3 ± 18	0.536
Central DBP (mm Hg)	82 ± 9	77 ± 9	−5 ± 9	83 ± 9	80 ± 8	−3 ± 12	0.569
Augmentation index (%)	27 ± 8	25 ± 8	−2 ± 7	25 ± 11	24 ± 11	−1 ± 8	0.461
Central PP (mm Hg)	43 ± 13	48 ± 15	5 ± 13	43 ± 14	43 ± 14	−0.5 ± 15	0.235
PP amplification (ratio)	1.28 ± 0.13	1.30 ± 0.11	0.02 ± 0.11	1.33 ± 0.17	1.30 ± 0.11	0.01 ± 0.15	0.686
Postmaximal exercise							
Brachial SBP (mm Hg)	203 ± 21	207 ± 23	4 ± 23	193 ± 28	198 ± 26	6 ± 24	0.728[a]*
Brachial DBP (mm Hg)	90 ± 11	88 ± 16	−1 ± 17	89 ± 12	86 ± 10	−3 ± 12	0.725*
Brachial PP (mm Hg)	113 ± 22	119 ± 25	6 ± 26	104 ± 24	112 ± 23	8 ± 22	0.577[a]*
HR (bpm)	159 ± 18	156 ± 18	−3 ± 13	152 ± 23	149 ± 23	−3 ± 19	0.805[a]*
Time to maximal BP (min)	12 ± 4	13 ± 4	1 ± 3	10 ± 4	12 ± 4	1 ± 3	0.199
Central SBP (mm Hg)	160 ± 16	158 ± 17	−2 ± 19	156 ± 23	156 ± 17	0.3 ± 21	0.676
Central DBP (mm Hg)	94 ± 13	87 ± 12	−7 ± 14	92 ± 17	88 ± 9	4 ± 15	0.328
Augmentation index (%)	3 ± 10	0.2 ± 12	−3 ± 9	9 ± 14	2 ± 15	−7 ± 13	0.177
Central PP (mm Hg)	65 ± 14	71 ± 10	6 ± 17	64 ± 18	68 ± 13	4 ± 15	0.720
PP amplification (ratio)	1.73 ± 0.14	1.75 ± 0.15	0.03 ± 0.14	1.66 ± 0.21	1.72 ± 0.18	0.06 ± 0.17	0.435

Data are presented as mean ± SD.
SBP, systolic blood pressure; DBP, diastolic blood pressure; PP, pulse pressure.
[a] Data were significantly different between groups at baseline ($P < 0.05$)
* P value is ANCOVA corrected for mean arterial pressure, which was significantly different between groups at baseline (all P values < 0.05). Central BP measures were recorded in 61 patients (30 controls) only. Significance is $P < 0.05$.

medications did not change significantly between groups over the course of the study.

Hemodynamic response to intervention. The hemodynamic characteristics of the study population at baseline and 12 months are shown in Table 2. Resting brachial SBP, DBP, and pulse pressure were all higher in the intervention group at baseline, with exercise brachial SBP, pulse pressure, HR, and time to maximal BP all higher in the control group at baseline (all P values < 0.05). All other baseline hemodynamic variables were similar between the study groups. Absolute values of exercise and resting, brachial, and central BP parameters were not significantly different between groups after intervention when analyzed by independent t-test or after ANCOVA correction for mean arterial pressure (as indicated in Table 2) (all P values > 0.05). There was also no significant difference between groups for exercise BP at any stage of the exercise test ($P > 0.05$). However, lifestyle intervention significantly reduced progression toward the development of an HRE in those participants who did not have an HRE at baseline (29.8% vs 59.5% progression to HRE, $P = 0.006$; Fig. 2A). Of those patients who had an HRE at baseline (Fig. 1B), lifestyle intervention failed to significantly change HRE status (22.0% vs 23.5%, $P = 0.855$). The change in maximal exercise SBP was moderately correlated with the change in total cholesterol and change in resting SBP ($r = 0.21$ and 0.21, respectively, $P < 0.05$).

DISCUSSION

An HRE is a common finding in patients with type 2 diabetes and is associated with increased LV mass (22) as well as cardiovascular morbidity and mortality in other populations (17,18,20,24). To our knowledge, this is the first study to investigate the impact of lifestyle intervention on brachial and central exercise BP in patients with type 2 diabetes. The novel finding was that 1 yr of lifestyle modification,

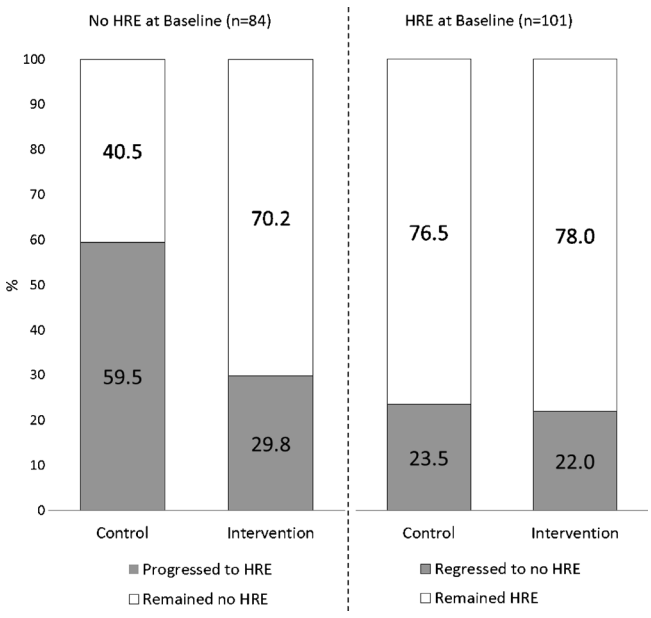

FIGURE 2—Progression or regression of an HRE (SBP ≥ 210 mm Hg for men and ≥190 mm Hg for women) from baseline to 1 yr in the control (n = 88) and intervention (n = 97) groups. Panel A indicates that of those participants who *did not* have an HRE at baseline, 1 yr of lifestyle intervention significantly attenuated the propensity to develop an HRE compared with controls (29.8% vs 59.5% progression to HRE, $P = 0.006$). Panel B indicates that of those participants who *did* have an HRE at baseline, 1 yr of lifestyle intervention failed to significantly change HRE status compared with controls (22.0% vs 23.5%, $P = 0.855$).

incorporating exercise training and dietary advice, significantly attenuated the propensity to develop a HRE. We did, however, find no significant change in absolute exercise BP values or LV mass. As expected, improvements to aerobic capacity ($\dot{V}O_{2max}$) and metabolic parameters including plasma glucose, HDL cholesterol, and $HOMA_{IR}$ were observed after lifestyle modification.

More than half the study population had an HRE at baseline, which is in accordance with our previous observations (22) as well as that of others (13). The clinical outcomes regarding such high prevalence in diabetic individuals have never been reported but are likely to be significant given the strong independent association between exaggerated exercise BP and cardiovascular mortality in other patient groups (12,19,20). Our finding that lifestyle intervention attenuated the development of an HRE only in those patients with a normal exercise BP response suggests that improvements to exercise hemodynamics may be more achievable before the development of overt exercise hypertension. The reasons for the lack of response in terms of being able to reverse an HRE to normal exercise BP are unknown. However, it is possible that an HRE represents an advanced state of cardiovascular irregularity that may not be amenable to major improvements in the short term. This study reinforces the pertinence of undertaking regular aerobic exercise for patients with type 2 diabetes and also highlights the need for further studies to understand the physiology underlying abnormal exercise BP.

Although this was not a mechanistic study, an exaggerated BP response to exercise may be due to several factors. Vascular properties, including increased aortic stiffness, which has a strong association with type 2 diabetes (21), may be crucial to the appropriate regulation of BP during exercise. Indeed, increased arterial stiffness has been reported to be associated with an HRE (25). This relationship may be expected because ejection of increased stroke volume (associated with exercise) into a stiff aorta could contribute to an exaggerated BP response. Furthermore, because arterial stiffness may be greater in the presence of dyslipidemia or hyperglycemia (21,26), metabolic characteristics may be of significance to the understanding of exercise BP. Indeed, hypercholesterolemia and insulin resistance have been associated with abnormal increases in exercise BP from resting conditions (3). Although we did not measure central artery stiffness, previous studies involving patients with type 2 diabetes have revealed improved large artery compliance and concomitant reductions in resting BP after participating in exercise and lifestyle modification (2,4). Also of note is the study by Chang et al. (5), which found evidence of brachial artery endothelial dysfunction in patients with exaggerated exercise BP during treadmill testing.

Although we were able to attenuate the propensity to develop an HRE in the intervention group, there was no significant difference in the absolute values of exercise BP between groups. As we have previously reported (10), the control group may have become more active over the course of the study (supported by the drop in resting brachial SBP of 1 mm Hg and slight improvement in $\dot{V}O_{2max}$ in control patients), which may partly explain the lack of BP change between groups. On the other hand, both the $\dot{V}O_{2max}$ and the time on the exercise treadmill were increased significantly in the intervention group. The increased treadmill time could have contributed to a greater increase in exercise BP, thereby masking an effect of the lifestyle intervention. However, the lack of change to submaximal exercise BP could also suggest otherwise.

Our previous research has shown an association between LV mass and HRE in patients with type 2 diabetes (22). Other studies also reported this in nondiabetic patients with normal resting BP (8). However, Lauer et al. (15) cautioned that this relationship may be confounded by age, resting BP, and body mass. We expected a reduction in LV mass after lifestyle change on the basis that improved vascular function would reduce LV afterload and improve ventricular–vascular interaction. Although this did not eventuate, an exercise-induced cardiac hypertrophy ("athlete's heart") can arise from participation in regular exercise, and this may have occurred in some patients. The likelihood of this being a major influencing factor is probably low, given that the intensity and duration of exercise required to induce athletic LV hypertrophy may be greater than the amount undertaken by patients in this study (1). Despite no significant change to cardiac size, we have previously reported that subtle indices of LV diastolic and systolic function can improve significantly in the short term in those patients who spend more time engaged in moderate and vigorous levels of activity (10).

SUMMARY AND CONCLUSIONS

An HRE is associated with cardiovascular morbidity and mortality (12,17,18,20,24) and increased LV mass (8,22). This study found that 1 yr of lifestyle intervention significantly improved metabolic profile, increased functional capacity, and importantly attenuated the progression toward an HRE in patients with type 2 diabetes and normal exercise BP. Given the high prevalence of an exaggerated exercise BP in patients with type 2 diabetes (13,22), our findings provide additional evidence for the cardiovascular protective effects of lifestyle modification that includes regular aerobic exercise in this patient group.

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Results and discussion presented in this article are those of the authors and do not constitute endorsement by the American College of Sports Medicine.

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