Blunted Cardiac Output from Overtraining Is Related to Increased Arterial Stiffness

ALEXANDRA M. COATES1, PHILIP J. MILLAR2,3, and JAMIE F. BURR1

1The Human Performance and Health Research Laboratory, Department of Human Health and Nutritional Sciences, University of Guelph, Guelph, ON, CANADA; 2Department of Human Health and Nutritional Sciences, University of Guelph, Guelph, ON, CANADA; and 3Toronto General Research Institute, Toronto General Hospital, Toronto, ON, CANADA

ABSTRACT

COATES, A. M., P. J. MILLAR, and J. F. BURR. Blunted Cardiac Output from Overtraining Is Related to Increased Arterial Stiffness. Med. Sci. Sports Exerc., Vol. 50, No. 12, pp. 2459–2464, 2018. Purpose: Moderate overtraining has been characterized by decreased exercising HR and recently decreased exercising stroke volume (SV), independent of alterations to blood volume. The aim of this study was to assess changes in arterial stiffness and central hemodynamics, and their relationship to exercising SV, after 3 wk of overload training. Methods: Twenty-six cyclists and triathletes completed 3 wk of either regular training (CON; n = 13) or overload training (OL; n = 13). Testing took place before (PRE) and after regular or overload training (POST). Resting measures included brachial blood pressure, HR, carotid–femoral pulse wave velocity (PWV) to assess arterial stiffness, and carotid pulse wave analysis to assess wave reflections and central hemodynamics. An incremental cycle test was used to assess peak power, maximal HR, and maximal lactate to assess overtraining status. Cardiac output (Q̇), SV, and HR were assessed using cardiac impedance. Results: Resting arterial stiffness was unaltered in CON but increased with OL after increased training (CON −0.1 ± 0.6 m s−1 vs OL +0.5 ± 0.8 m s−1, P = 0.04). Resting blood pressure and central hemodynamics, including aortic pressures, augmentation index, and subendocardial viability ratio, did not change (all P > 0.05). Maximal SV (CON +3 mL vs OL −9 mL, P = 0.04), HR (CON −2 ± 4 bpm vs OL −9 ± 3 bpm, P < 0.001), and Q̇ (CON +0.32 L min−1 vs OL −1.75 L min−1, P = 0.01) decreased with OL from PRE to POST. A significant inverse relationship existed between changes in PWV and maximal Q̇ (r = −0.44, P = 0.04) and changes in PWV and peak power (r = −0.48, P = 0.01), and trended for SV and PWV (r = −0.41, P = 0.055). Conclusions: Overload training results in increased resting arterial stiffness and reduced SV during exercise, with no changes to resting central hemodynamics. Key Words: OVERTRAINING, OVERREACHING, ENDURANCE TRAINING, CARDIAC FATIGUE, PULSE WAVE VELOCITY

Endurance athletes train for the primary purpose of improving performance and, as such, are required to undergo a certain amount of training overload for physiological adaptations to manifest upon recovery (1). However, as a consequence of hard training blocks or training camps, athletes regularly demonstrate decrements to performance in the form of overreaching (2). Three weeks of overload training has been shown previously to reduce endurance exercise performance (3–5), exercising HR (3,5,6), lactic acid production (3), resting muscle sympathetic nerve activity (5), and increase the incidence of illness (6). Recently, evidence suggests that overload training in well-trained triathletes is also associated with reductions in stroke volume (SV), cardiac output (Q̇), and plasma epinephrine during exhaustive exercise, independent of an alteration in blood volume (4), suggesting a sympathetically mediated ionotropic impairment.

An alternative mechanism for reductions in Q̇ with overload training could involve impaired arterial–ventricular coupling and myocardial contractility secondary to increases in arterial stiffness (7–9). Increased central arterial stiffness permits a faster return of reflected pulse waves and can increase left ventricular afterload (8,10). Several studies have noted increases in central arterial stiffness, measured as carotid–femoral pulse wave velocity (cf-PWV), after eccentric exercise (11,12) and ultramarathon running (13). In addition, increased training loads during a weeklong running camp increased arterial stiffness (9), whereas 8 wk of increased training volume in triathletes increased measures of pulse wave reflection (14). It has also been reported that higher resting central arterial stiffness is associated with increased cardiac fatigue after high-intensity
exercise (15). Collectively, these results suggest that repeated high-intensity or prolonged endurance training efforts may reduce exercise performance and impair exercising VO2 by altering arterial–ventricular coupling.

The purpose of this investigation was to determine whether 3 wk of overload training in recreational endurance athletes blunts maximal SV and \( \dot{Q} \), and to assess the relationships with changes in central arterial stiffness. We hypothesized, based on previous evidence (4), that overload training would attenuate SV and \( \dot{Q} \) responses during maximal exercise and that these responses would be associated with increases in resting central arterial stiffness and peripheral pulse wave reflection.

**METHODS**

**Participants.** Thirty-three recreational triathletes and cyclists (19 male; 36 ± 10 yr [mean ± SD]) were recruited from local triathlon and cycling clubs. Inclusion criteria stipulated athletes must be 18–50 yr old, currently after a structured endurance training program, familiar with cycle pacing, and be free from disease, illness, or injury. Subjects were randomized by enrollment order to either the regularly training control group (CON; \( n = 16 \)) or the overload training group (OL; \( n = 17 \)) in a block-randomized fashion. A subset of these data has been published previously examining autonomic responses to overload training (5,16).

All subjects completed a PAR-Q+ questionnaire to ensure readiness for physical activity and provided written informed consent in accordance with the declaration of Helsinki. This study was approved by the University Research Ethics Board.

**Experimental protocol.** Subjects were required to complete a 4-wk training protocol, with the first week’s training performed at a ~50% reduction in their regular training volume to ensure subjects were recovered at baseline, followed by 3 wk performed at either 100% of their regular training volume (CON), or ~150% (OL). All subjects were given Polar A300 watches and heart straps (Polar Electro Oy, Kempele, Finland), as well as online accounts through the device manufacturer’s website to track their training volumes and intensities and to monitor adherence.

**Training.** To achieve adequate overload training in this population, and to attain ~150% of their training volume, OL athletes were required to complete their regular training and to perform three additional cycle training sessions, two of which were in the laboratory under standardized conditions. The first additional session was a high-intensity interval workout consisting of 4 × 30 s. Wingate anaerobic tests on an electromagnetically braked cycle ergometer (Racermate Velotron, Seattle, WA), at a load of 7.5% body weight, with 4 min of recovery between intervals. The second session was a 15-km virtual time trial performed on the same cycle ergometer over a standardized course with undulating terrain. The final session was a 2-h ride completed on the subject’s own time and tracked using the previously described HR monitoring. The ride was prescribed as four blocks of 10 min at an HR of 50%–60% of \( \dot{V}O_{2\text{max}} \) and 20 min at an HR of 66%–75% of their \( \dot{V}O_{2\text{max}} \). HR zones for this session were calculated from the initial \( \dot{V}O_{2\text{max}} \) testing. CON athletes were asked to complete their previously programmed workouts, and all sessions were tracked via their Polar Flow accounts for the 3-wk duration.

**Testing.** Participants completed testing sessions after the first week of reduced training (PRE) and after the 3 wk of either OL or CON training (POST). Participants were instructed to maintain consistent dietary choices on both days. To ensure consistency, participants were required to complete meal logs from the night prior and the day of the first testing session (PRE), which were replicated for the other visits. Participants were instructed to abstain from exercise, alcohol, smoking, and drugs for 24 h before testing. Lastly, subjects were asked to complete the Profile of Mood States Second Edition (POMS-2) (Multi-Health Systems Inc., NY) to assess mood states across the training protocol to evaluate overtraining status. The POMS-2 has a dose–response relationship with training load, with increased load equating to increased negative mood scores, but does not have a cutoff value for overtraining status (2). The total mood disturbance score was determined by subtracting the vigor–activity scale from the sum of 5 negative mood scales, and a constant of 100 was added to account for negative values (17).

**Resting cardiovascular assessment.** After voiding of the bladder and collection of basic anthropometrics (height at baseline, weight), participants were given 5 min of supine rest followed by two measures of resting blood pressure using a standard sphygmomanometer. If the difference between the two blood pressure measures was greater than 5 mm Hg, a third measure was taken, and the average was used. Participants rested quietly for a minimum of 10 min in standardized laboratory conditions maintained at 20°–23°C, before undergoing measurement of cf-PWV, the gold standard method for non-invasive assessment of arterial stiffness (18). The collection of PWV was facilitated using Sphygmocor CPVH (AtCor Medical Ltd., NSW, Australia), whereby PWV = distance (m) / transit time (s). The strongest carotid and the femoral arterial pulse points were located using manual palpation, and the shortest straight line from the suprasternal notch to the carotid and femoral arteries was measured with an anthropometric tape measure to the nearest millimeter. A correction factor was applied to the overall distance to subtract carotid–sternal distance (19). Consecutive pulse wave measures were taken at the carotid artery followed by the femoral artery using a high-fidelity Millar tonometer, with 10 s of consistent waveforms recorded at each site. Transit time was calculated automatically from the ECG-gated pulse waveforms. A minimum of two cf-PWV measures were collected and used for analysis unless the difference was greater than 0.5 ms−1, in which case a third measure was taken and the median was calculated (20).

To characterize the contribution of peripheral wave reflection to central blood pressure, a generalized transfer function (21) was applied to carotid artery pressure waveforms. The carotid artery site was selected due to its proximity to the aorta and closer similarity than radial waveforms to aortic waveforms. Central pressures were calibrated using brachial mean arterial pressure and Q˙ and T, which were calculated from the ECG-gated pulse waveforms. A minimum of two cf-PWV measures were collected and used for analysis unless the difference was greater than 0.5 ms−1, in which case a third measure was taken and the median was calculated (20).
pressure and diastolic blood pressures. Measures analyzed from the waveform analysis included: aortic blood pressure (aortic systolic, aortic diastolic, aortic mean arterial, and aortic pulse pressures), augmentation index (AIx) of pressure waves, ejection duration, and Buckberg’s subendocardial viability ratio (SEVR) to assess cardiac perfusion to work ratio (22).

**Exercise testing.** Following resting measures, subjects were fitted with an impedance cardiography device to measure HR, SV, and cardiac output (Q) during exercise (Physioflow Enduro, Manatec Bio-medical, Marcheren, France), which was calibrated before each test. This device has been previously validated against the direct Fick method during exercise (23).

Subjects performed a maximal incremental exercise test on a Velotron cycle ergometer (Racermate Velotron) with oxygen consumption measured via mixing chamber using a Moxus metabolic cart (Moxus, AEI Technologies, Pennsylvania, PA). Men followed a protocol starting at 100 W and increasing 1 W every 2 s, with women similarly starting at 100 W, but increasing 1 W every 3 s. Subjects were not tolled to stop upon reaching a plateau in oxygen consumption, and instead the test was terminated when they could no longer maintain a cadence above 40 rpm seated or standing. Maximal HR, Q, and SV were recorded, and finger stick lactate sampling (Lactate Plus, Nova Biomedical, Waltham, MA) was initiated at 60 s post-exercise with samples taken every 30 s until a peak was observed.

**Statistical analysis.** Descriptive participant characteristics were assessed between groups using unpaired Student’s t-tests. Training outcomes (peak power, VO2max, maximal lactate, and mood scores), resting cardiovascular measures (PWV, blood pressure, AIx, AIx@75, ejection duration, and SEVR), and exercising cardiac parameters (HRmax, SVmax, and Qmax) were examined using two-way repeated-measures ANOVA with Bonferroni corrections for post hoc testing. All data were assessed for normality visually and using the Shapiro–Wilks test. Change scores (Δ) used for linear regression analysis were performed by subtracting baseline values (PRE) from posttraining values (POST). Bivariate linear regression analysis without adjustment was used to examine relationships between ΔPWV and ΔQmax, ΔPWV and ΔSVmax, and ΔPeak power. Data are reported as mean ± SD with significance set a priori at P < 0.05. Statistical analysis was performed using Statistical Package for the Social Science (SPSS, version 24; IBM, Chicago, IL).

**RESULTS**

We recruited a total of 33 subjects; however, only 26 athletes completed the study. In the OL group, one subject was not able to follow the overload training protocol, one subject became ill, and two subjects had incomplete exercising cardiovascular data, resulting in 13 OL subjects. In the CON group, one subject was injured outside of the study and two subjects became ill, resulting in 13 CON subjects (13 OL:13 CON). Subject characteristics are presented in Table 1.

**Training outcomes.** Performance (peak power) was significantly increased from PRE to POST in the CON group, compared with a decrease in performance in the OL group (CON 9 ± 11 W vs -9 ± 13 W, interaction term P < 0.001). VO2 max did not change over the 3 wk with either CON or OL (CON +39 ± 203 mL·min⁻¹ vs OL +4 ± 199 mL·min⁻¹, P = 0.71). Maximal lactate was unchanged in CON and decreased nonsignificantly from PRE to POST with OL (CON -0.1 ± 2.0 mmol·L⁻¹ vs OL -2.0 ± 3.0 mmol·L⁻¹, interaction term P = 0.09). Lastly, POMS-2 negative mood states were increased from PRE in both groups (CON +10 ± 15 a.u. and OL +25 ± 23 a.u., main effect of time P < 0.001). The overtraining status of these individuals with the addition of the other two athletes is discussed in a previous publication (16).

**Resting cardiovascular measures.** Changes to cf-PWV from PRE to POST are presented in Figure 1. Brachial blood pressure, PWV, PWA, central blood pressures, and estimates of myocardial work and perfusion (SEVR) from PRE to POST training are presented in Table 2. Central arterial stiffness did not change in CON and was increased at POST with OL training (Interaction term P = 0.04). Resting HR decreased in both groups from PRE to POST (Main effect of time P = 0.03). There were no significant changes across the 3-wk training block between groups for any of the other resting cardiovascular measures.

**Exercising cardiovascular measures.** There was a significant decrease in SVmax from PRE to POST with OL, which did not occur with CON (Interaction term P = 0.04). HRmax did not significantly change with CON after training, but decreased with OL (Interaction term P < 0.0001), as well as Qmax (Interaction term, P = 0.01). When SVmax was normalized to body surface area, the interaction between OL and CON from PRE to POST was maintained (CON 60.9 ± 11.3 to

---

**TABLE 1. Baseline characteristics.**

<table>
<thead>
<tr>
<th>Group (n)</th>
<th>Age (yr)</th>
<th>Height (cm)</th>
<th>Weight (kg)</th>
<th>VO2 (mL·kg⁻¹·min⁻¹)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control (13)</td>
<td>36 ± 10</td>
<td>176 ± 9</td>
<td>72.5 ± 10.2</td>
<td>54.8 ± 8.8</td>
</tr>
<tr>
<td>Males (6)</td>
<td>37 ± 10</td>
<td>181 ± 6</td>
<td>78.5 ± 7.8</td>
<td>59.6 ± 7.6</td>
</tr>
<tr>
<td>Females (5)</td>
<td>34 ± 11</td>
<td>177 ± 7</td>
<td>69.3 ± 4.1</td>
<td>47.1 ± 5.5</td>
</tr>
<tr>
<td>Overload (13)</td>
<td>37 ± 8</td>
<td>175 ± 7</td>
<td>73.2 ± 11.4</td>
<td>55.5 ± 7</td>
</tr>
<tr>
<td>Males (8)</td>
<td>40 ± 6</td>
<td>178 ± 5</td>
<td>80.2 ± 6.7</td>
<td>59.4 ± 4.1</td>
</tr>
<tr>
<td>Females (5)</td>
<td>34 ± 10</td>
<td>170 ± 7</td>
<td>62.1 ± 7.5</td>
<td>49.2 ± 6.2</td>
</tr>
</tbody>
</table>

Data are presented as mean ± SD. No significant differences between groups.
cardiac loads (8). When arteries stiffen with age, hypertension, inflammation, and atherosclerosis (24), the heart requires more force upon ejection. Further exacerbating the situation, pulse waves that are reflected from downstream bifurcations in the vascular system return earlier in the cardiac cycle and can increase central systolic pressures (8) and decrease the available time for cardiac perfusion and filling (10). Central artery stiffness is recognized as a risk factor for heart disease in the general population (24), and a 1-m s$^{-1}$ increase in PWV has been demonstrated to be associated with a 15% risk of cardiovascular disease mortality (25).

Although arterial stiffness has never been assessed in the context of overtraining or overreaching with endurance athletes, there is a small body of research that suggests increased stiffness may occur acutely with prolonged exercise. Burr et al. (13) demonstrated ultramarathon runners had acute reductions in large artery compliance after a 120- to 195-km mountainous trail race. By contrast, Phillips et al. (26) did not see changes to cf-PWV after an ultramarathon, and Vlachopolous et al. (27) did not see changes to cf-PWV and saw a decrease in A$_i$ and A$_i@75$ bpm after a marathon. Importantly, all of these studies were performed acutely after prolonged races (13,26,27), coincident with a reduction in resting blood pressure. As blood pressure is involved in the stress–strain relationship, such postexercise hypotension could reduce cf-PWV and mask functional or structural vascular changes (18). By contrast, cross-sectional comparisons of marathon and ultramarathon runners (27,28) have reported higher resting PWV compared with recreationally active controls, with running distance per training session related positively to central stiffness (28). Further, after 8 wk of increased training, endurance athletes demonstrated a plateau in performance and increased A$_i$, with no changes in central blood pressure (14), whereas a week of increased running distance and intensity increased arterial stiffness in collegiate endurance runners (9). Our present investigation indicates that short-term overload training leads to increased arterial stiffness, independent of changes to blood pressure.

Several mechanisms have been proposed to explain increases in arterial stiffness after acute or chronic overload exercise, including increased inflammation (11,29) and vasoconstrictor tone (30). A causal role of inflammation on arterial stiffness has been demonstrated previously using

| TABLE 2. Measures of blood pressure and carotid pulse wave analysis in recreational endurance athletes after 3 wk of either regular training or overload training. |
|-----------------|-----------------|-----------------|-----------------|
|                  | Control         | Overload        |                 |
|                  | PRE  | POST | PRE  | POST |
| Resting HR (bpm) | 60  ± 7 | 57  ± 7 | 58  ± 8 | 56  ± 8 |
| Brachial SBP (mm Hg) | 118 ± 10 | 114 ± 9 | 113 ± 7 | 112 ± 15 |
| Brachial DBP (mm Hg) | 73  ± 6 | 74  ± 7 | 73  ± 5 | 72  ± 8 |
| Aortic SBP (mm Hg) | 109 ± 10 | 107 ± 10 | 105 ± 6 | 105 ± 14 |
| Aortic DBP (mm Hg) | 73  ± 6 | 74  ± 7 | 73  ± 5 | 72  ± 8 |
| Aortic MAP (mm Hg) | 87  ± 7 | 87  ± 6 | 86  ± 5 | 85  ± 10 |
| Aortic PP (mm Hg) | 37  ± 6 | 34  ± 11 | 32  ± 6 | 34  ± 8 |
| Aix (%) | 11 ± 13 | 10 ± 12 | 16 ± 11 | 12 ± 15 |
| Aix75 (%) | 1 ± 11 | 1 ± 12 | 7 ± 9 | 2 ± 13 |
| Ejection duration (ms) | 326 ± 15 | 330 ± 19 | 329 ± 14 | 331 ± 15 |
| SEVR (%) | 196 ± 45 | 209 ± 35 | 205 ± 37 | 218 ± 47 |

Data are presented as mean ± SD. *Main effect of time $P < 0.05$.

$60.7 ± 6.8$ mL m$^{-2}$ vs OL $59.6 ± 10.5$ to $55.2 ± 8.2$ mL m$^{-2}$, $P = 0.048)$. PRE to POST data are presented in Figure 2.

The changes in resting PWV demonstrated a trend for a negative association to the changes in $SV_{\text{max}}$ after training ($r = -0.41, P = 0.055$) and were related significantly to the changes in $Q_{\text{max}}$ ($r = -0.44, P = 0.04$) and peak power ($r = -0.48, P = 0.01$) after training (Fig. 3).

**DISCUSSION**

This study was the first to examine the effects of overload training in endurance athletes on central arterial stiffness, and whether these changes in PWV were associated with changes in SV and $Q$. In line with our hypothesis, 3 wk of overload training, but not regular exercise training, resulted in reduced HR, SV, and $Q$ at a maximal aerobic effort. These findings are consistent with previous observations (4). A novel finding was that overload training, but not regular exercise training, increased central PWV independent of a change in resting blood pressure or pulse wave reflection. Finally, the changes in PWV after exercise training were negatively correlated with decreases in $Q_{\text{max}}$, with a similar trend for an association with $SV_{\text{max}}$. These results demonstrate that overload training can induce stiffening of central vessels, and that these changes may relate to the attenuated cardiac responses during exercise in athletes undergoing overload training.

Arterial compliance is hallmark of a healthy cardiovascular system, as compliant arteries promote the conversion of pulsatile flow to steady flow at the capillaries, and attenuate

---

**FIGURE 2**—Maximal HR (A), SV (B), and cardiac output (C) responses to regular (CON) or overload (OL) training among recreational endurance cyclists and triathletes. Data presented as mean ± SD. *$P < 0.05$, **$P < 0.005$, ***$P < 0.0001$.**
In the present study, we did not assess inflammatory markers, we cannot specifically comment on the possibility of a causative relationship between inflammation and increased arterial stiffness after overtraining. A second possibility is that increased cf-PWV with overload training was secondary to increased vasomotor sympathetic outflow. We previously reported that a subset of our participants had increased resting muscle sympathetic nerve activity after overload but not control training (5). The independent role of peripheral sympathetic activation on arterial stiffness remains controversial (31), although we recently demonstrated that acute increases in muscle sympathetic nerve activity, without changing blood pressure, can elevate cf-PWV (32). Future studies are required to investigate the specific mechanisms responsible for increased cf-PWV after short-term overload training. Lastly, it is unknown whether a short overload protocol, such as the 3-wk protocol used in the present study, can induce structural changes to the vasculature.

To our knowledge, only one other study has assessed cardiac function during exercise with overtraining (4), demonstrating that overreached athletes had reduced SV and $\dot{Q}$ during exercise when assessed with cardiac impedance, in concert with decreased plasma concentrations of epinephrine during exercise (4). It was postulated that reduced catecholamines could explain the reduced cardiac response in overreached athletes (4). Although we did not assess plasma catecholamines in the present study, we previously reported that the muscle sympathetic response was not altered during static handgrip exercise after overload training (5). However, a lack of change in sympathetic outflow directed toward the muscle vasculature does not preclude a role for an alteration in the release of catecholamines from the adrenal medulla, and it has long been hypothesized that overtraining can result in hypothalamic–pituitary–adrenal dysregulation (2). Alternatively, or in combination, increases in central PWV could affect SV through changes in arterial–ventricular coupling. Using direct measurement with echocardiography, cardiac impedance can be used to indirectly and reliably assess SV measures during maximal exercise (23), providing a functional assessment for athletes. It is recognized that there are sex differences in cardiovascular function that could be influenced by menstrual cycles (36), and our small sample size did not allow for between-sex analysis. As our groups were sex and age matched, and as PRE–POST testing took place one month apart, it is assumed our female subjects would be in similar phases of their menstrual cycle. Further research is needed to determine between sex differences in physiological responses to overtraining. Lastly, we did not assess hydration status in the groups, other than indirectly through weight, which was unchanged across the intervention for both groups. As previous research demonstrated unchanged blood volumes and decreased $SV_{\text{max}}$ and $\dot{Q}_{\text{max}}$ with overreached athletes (4), we do not believe blood volume or dehydration to be a significant factor in the present investigation.

**CONCLUSIONS**

This was the first study to assess central arterial stiffness and hemodynamics after overload training in recreational endurance athletes. After overload training, it was determined that there is a moderate increase in resting cf-PWV, with no
significant changes to the magnitude of pulse wave reflection, central blood pressure, or estimates of cardiac perfusion at rest. Further, the increases in resting central arterial stiffness were related to decrements in maximal cardiac output (Q) and peak power output during exercise after overload training. This research provides evidence that vascular adaptations may be involved in exercise underperformance after periods of overtraining. Further research into the chronic cardiovascular effects of overtraining is warranted.

REFERENCES


This research was supported by a grant from the Natural Science and Engineering Research Council of Canada Discovery (no. 06019 to P. J. M. and no. 03974 to J. F. B.); the Ontario Ministry of Research, Innovation, Science (no. 34379 to P. J. M. and no 460597 to J. F. B.); and the Canadian Foundation for Innovation (no. 34379 to P. J. M. and no. 460597 to J. F. B.), A. M. C. was supported by a Queen Elizabeth II Graduate Scholarship in Science and Technology. The authors have no conflicts of interest to declare. The results of the study are presented clearly, honestly, and without fabrication, falsification; or inappropriate data manipulation. The results of the present study do not constitute endorsement by the American College of Sports Medicine.