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EFFECTS OF AEROBIC CONDITIONING ON CARDIOVASCULAR SYMPATHETIC RESPONSE TO AND RECOVERY FROM CHALLENGE

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Abstract

Objective—Exercise has widely-documented cardioprotective effects but the mechanisms behind these effects are still poorly understood. Here, we test the hypothesis that aerobic training lowers cardiovascular sympathetic responses to and speeds recovery from challenge.

Methods—We conducted a randomized controlled trial contrasting aerobic versus strength training on indices of cardiac (pre-ejection period, PEP) and vascular (low-frequency blood pressure variability, LF-BPV) sympathetic responses to and recovery from psychological and orthostatic challenge in 149 young, healthy and sedentary adults.

Results—Aerobic and strength training did not alter PEP or LF-BPV reactivity to or recovery from challenge.

Conclusions—These findings, from a large randomized controlled trial using an intent-to-treat design, show that moderate aerobic exercise training has no effect on PEP and LF BPV reactivity to or recovery from psychological or orthostatic challenge. In healthy young adults, the cardioprotective effects of exercise training are unlikely to be mediated by changes in sympathetic activity.

It is well-established that aerobic exercise has significant cardioprotective effects for the general population. Recent meta-analyses have demonstrated that in 513,472 individuals, high and moderate levels of leisure time physical activity are associated with significant reduction in risk of heart disease (27% and 12% respectively) (Sofi, Capalbo, Cesari, Abbate, & Gensini, 2008) and that in 883,372 individuals followed for 20 years, physical activity was associated with a 35% reduction in the risk of cardiovascular mortality (Nocon et al., 2008). Consensus panels consistently recommend physical activity as an essential

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component of a healthy lifestyle (Haskell et al., 2007; Nelson et al., 2007; Pearson et al., 2003; Pearson et al., 2002).

Several biological mechanisms responsible for this cardioprotective effect are well understood, such as reduced metabolic demands on the myocardium and increased electrical stability (Scheuer & Tipton, 1977), but some remain more elusive. Modulation of responses to challenging events, both physical and psychological, is a pathophysiological mechanism that has received considerable attention. Although there is evidence to the contrary, a number of large cross-sectional and prospective studies have shown that greater reactivity to and prolonged recovery from psychological challenges predict future blood pressure status (Carroll, Phillips, Der, Hunt, & Benzeval, 2011; Steptoe & Marmot, 2005; Tuomisto, Majahalme, Kähönen, Fredrikson, & Turjanmaa, 2005), markers of atherosclerosis (Gianaros, Onyewuenyi, Sheu, Christie, & Critchley, 2011; Heponiemi et al., 2007; Jennings et al., 2004), and left ventricular mass (Kapuku et al., 1999). A recent meta-analysis confirms that greater responses to (relative risk (RR) = −.091, p <.001) and slower recovery (RR = −.096, p <.001) from laboratory challenges predict future cardiovascular disease (Chida & Steptoe, 2010). While these effects are small, they generally are consistent with the hypothesis.

Cardiovascular responses to and recovery from psychological challenge depend at least in part on the autonomic nervous system and in a recent report, we tested one element of this hypothesis. Healthy participants were randomized to a 12-week aerobic or strength training program. Indices of heart rate (HR) and cardiac parasympathetic modulation (HF-HRV) in response to and recovery from psychological and orthostatic challenge were collected before and after conditioning, as well as after 4 weeks of sedentary deconditioning. Contrary to expectation, aerobic conditioning produced no significant change in any of these indices. These findings suggest that aerobic training has its cardioprotective effects through some mechanism other than cardiac parasympathetic modulation of reactivity and recovery.

One candidate for this other mechanism is attenuated sympathetic nervous system (SNS) responses and recovery. The contribution of the SNS to cardiovascular disease and hypertension is unequivocal. Sympathetic nervous system dysfunction plays a significant role in the etiology of heart failure and hypertension (Parati & Esler, 2012) and in diabetes and metabolic syndrome (Straznicky et al., 2012). SNS activation is causally implicated in sudden cardiac death (Schwartz & Zipes, 2000). The increased incidence of myocardial infarction in the morning has been attributed to morning rise in SNS activity (Muller, 1999).

In addition, some evidence suggests that aerobic exercise training attenuates indices of SNS activity. For example, in post-myocardial infarction patients and untreated hypertensives, aerobic training significantly reduced muscle sympathetic nerve activity measured at rest (MSNA) (Laterza et al., 2007; Martinez et al., 2011). However, the effect of exercise training in normal participants may be different and indeed, some studies report that aerobic exercise training does not attenuate indices of SNS responses to psychological challenge (Cleroux, Peronnet, & de Champlain, 1985; Ray & Carter, 2010). However, these studies generally have had small and/or all male samples. In this paper, we examine the role of the

SNS in reactivity to and recovery from psychological challenge in a large sample of sedentary, young and healthy men and women.

Impedance cardiography (ICG) is a non-invasive method of measuring cardiac hemodynamic indices and systolic time intervals. Among these, pre-ejection period (PEP) is often used to assess myocardial β-adrenergic activity (Kelsey, Alpert, Patterson, & Barnard, 2000; Richter, Baeriswyl, & Roets, 2012; Sherwood et al., 1990). PEP is defined as the time interval between ventricular depolarization and the opening of the aortic valve. Increases in sympathetically driven myocardial contractility result in decreases in PEP. In support of this understanding, Mezzacappa et al. demonstrated that administration of epinephrine in healthy subjects significantly decreased PEP compared to placebo (Mezzacappa, Kelsey, & Katkin, 1999). Blockade of β-adrenergic receptors leads to an increase of PEP (Schachinger, Weinbacher, Kiss, Ritz, & Langewitz, 2001), further validating it as a non-invasive index of cardiac sympathetic tone. Postural changes affect PEP as well, as myocardial contractility is dependent on preload. For example, head-up tilt has been shown to increase PEP duration (Stafford, Harris, & Weissler, 1970).

Some evidence suggests that it is also possible to non-invasively measure vascular sympathetic regulation. Like heart rate (HR), blood pressure exhibits periodic oscillations at high (0.15–0.50 Hz, HF) and low frequencies (0.04–0.15 Hz, LF), a result of the regulatory control systems having different response times to changes in blood pressure (Harald M Stauss, 2007). While HF blood pressure variability (HF-BPV) is a direct product of respiratory-induced mechanical fluctuations of cardiac output which change arterial pressure (deBoer, Karemaker, & Strackee, 1987), some studies suggest that LF-BPV reflects vascular sympathetic drive (Cevese, Grasso, Poltronieri, & Schena, 1995; Dimier-David, Billon, Costagliola, Jaillon, & Funck-Brentano, 1994; Ditor et al., 2005; Montano et al., 1992; Schächinger, Weinbacher, Kiss, Ritz, & Langewitz, 2001). For example, Schächinger et al. demonstrated that in healthy volunteers, systolic BPV in the low-frequency range increased during nitroprusside-induced hypotension and decreased during norepinephrine-induced hypertension, reflecting central SNS output (Schächinger et al., 2001). Also, significant increases in LF-BPV amplitude in response to orthostatic challenge have been observed (Cooke et al., 1999; Mukai & Hayano, 1995). These data are consistent with the view that LF-BPV reflects vascular sympathetic control. In this paper, we test the hypothesis that in contrast to strength training, aerobic exercise attenuates the sympathetic response to and shortens the recovery from psychological and orthostatic challenge.

Method

Study Design

The study was a randomized controlled trial of aerobic vs. strength training on cardiovascular autonomic regulation in response to and recovery from challenge. All subjects provided informed consent. The Institutional Review Boards of Columbia University Medical Center and St. John's University approved this study.

Previously, we reported findings from this trial on the effects of aerobic training on cardiac parasympathetic reactivity to and recovery from psychological and orthostatic challenge (R.

P. Sloan et al., 2011). Because that paper provided a comprehensive account of the research methods, below we present a less detailed description.

Study Participants

Study participants were healthy, sedentary young adults, 18–45 years of age. Participants were eligible if they did not exercise regularly or exceed American Heart Association standards for average fitness (VO_2max ≤ 43 and 37 ml/kg/min for men and women respectively, established by cardiopulmonary exercise testing (CPET)). 149 participants met enrollment criteria and were randomized to either the aerobic (N=74) or strength training (N=75) group. Participants received a 6-month membership in a fitness facility and $300 for participation in the study.

Assessment of Aerobic Fitness

Maximum aerobic fitness (VO_2max) was assessed by a 30 Watts (W) every 2 minute graded exercise test on an Ergoline 800S cycle ergometer (SensorMedics Corp., Anaheim, CA), until VO_2max criteria (RQ ≥ 1.1, increases in ventilation without concomitant increases in VO_2, achievement of maximum age-predicted heart rate, and/or volitional fatigue) were reached. The highest VO_2 value attained was considered VO_2max (Buchfuhrer et al., 1983). Minute ventilation was measured by a pneumotachometer connected to a FLO-1 volume transducer module (PHYSIO-DYNE Instrument Corp., Quogue, NY). Ventilatory gas analysis was assessed using paramagnetic O_2 and infrared CO_2 analyzers connected to a computerized system (MAX-1, PHYSIO-DYNE Instrument Corp.). All systems were calibrated against known medical grade gases.

Experimental Protocol

Both training programs were 12 weeks in length. Before training, all subjects met individually with a trainer to review their exercise regimens. After that, they exercised on their own, 3–4 times per week, in designated facilities. They were permitted to construct individualized exercise programs so long as they met the criteria below. Adherence to training programs was documented by weekly logs and computerized attendance records.

Subjects were tested on three occasions: before training, immediately after completion of training, and again after 4 weeks of sedentary deconditioning during which they were to abstain completely from any form of exercise. Data collection staff were blind to training group assignment.

Conditioning Programs

Aerobic conditioning—Subjects chose from a series of activities, e.g., cycling on a stationary ergometer, running on a treadmill, or climbing on a Stairmaster. Subjects were instructed to exercise at 70% of their maximum heart rate (220-age for men, 226-age for women). They were given an initial goal of at least 20 mins aerobic exercise per session and increased duration gradually over two to three weeks, up to 45–60 min.

Strength training—At the initial session, subjects established a level of effort that permitted them to complete three sets of 10 repetitions for each of the following exercises:

bench presses, shoulder presses, quadriceps extensions, biceps curl, lateral pulls, triceps presses, and hamstring curls exercise. Subjects were instructed to increase the weight loads for these exercises by five pounds every two weeks.

Psychophysiology Testing Sessions

Testing sessions were scheduled for prior to randomization, after completion of training, and after completion of deconditioning. Participants were tested in the Behavioral Medicine Laboratory after eating a light breakfast and abstaining from caffeinated beverages. ECG electrodes were placed on the right shoulder, on the left anterior axillary line at the 10th intercostal space and in the right lower quadrant. Stretch bands were placed around the subject's chest and abdomen for measurement of respiration. Band electrodes for impedance cardiography (ICG) were placed on the upper neck and the root of the neck and on the upper abdomen and at the level of the xiphoid process. A Finapres blood pressure cuff was placed on the middle finger of the non-dominant hand.

After instrumentation, the subject rested quietly in the seated position during a 10 min baseline period followed by a 5-min period for preparation of their public speaking task (see below), the 5-min public speaking period and a 5-min recovery period. Then the subject was placed in the supine position on a Midland electric tilt table, modified to suspend a computer monitor in the subject's visual field for display of the psychological tasks. A numeric keypad, for responding to the arithmetic and Stroop tasks, was secured in a comfortable position relative to the dominant hand. The subject then rested quietly for 6 min of adaptation to position, followed by a two minute period for calibration of monitoring devices, and a second 10 min quiet, resting baseline. Subjects then, in fixed order, performed the 5-min mental arithmetic task, a 5-min recovery period, the 5-min Stroop color-word task, and another 5-min recovery period. Subjects were instructed to remain silent throughout the procedures. After the second recovery period, the tilt table was moved to the 70° head-up position and the monitoring devices were recalibrated. Physiological signals were collected for 10 min in the upright position.

Psychological Stressors

Public speaking task—Subjects selected one of five controversial topics, e.g., abortion, welfare. They were informed that their performance would be video-recorded for evaluation and a camera was placed prominently in their view. They then spent five min preparing for the speech followed by a 5-min period when they delivered it.

Mental arithmetic—The task required subjects to subtract serially by 7's starting with a 4 digit number presented on the monitor. At one min intervals, subjects received verbal prompts from the laboratory technician, e.g., "please subtract faster." Subjects were instructed to subtract as quickly and accurately as possible. If they made mistakes or lost their place, a new 4-digit starting number was provided.

Stroop color-word task—The computer presented color name words (blue, green, yellow, red) in a color which was either congruent or incongruent with the name. The task was to press a key on the keypad corresponding to the color of the letters, not the color

name. The task was paced by the computer so that subjects achieved a 67% correct response rate. Thus, if they performed poorly, the presentation rate slowed and if they performed well, it increased.

Recovery periods—A five-minute recovery period followed each challenge.

Measurement of Physiological Signals

ECG and Impedance Cardiography Analog ECG signals were digitized at 500 Hz by a National Instruments 16 bit A/D conversion board and passed to a microcomputer. ICG data were collected using the Minnesota 304B system with no gain. The impedance signal (Z0) and the first derivative of pulsatile impedance acquisition (dZ/dt) were digitized at 250 Hz and 500 Hz respectively by the NI A/D board and collected by the microcomputer. Mindware software (MindWare Technologies LTD, Gahanna, Ohio) was used to analyze ECG and ICG signals in 60-second epochs. PEP was measured as the time interval between the Q wave of the ECG and the B point of the dZ/dt wave. Errors in marking of R waves in the ECG signal and B, Z and X points in the dZ/dt waveform were corrected by visual inspection. Epochs in which more than 20% of the ECG or ICG signals were unreadable due to artifact were not scored. Only baselines (10 min) with 6 60-second epochs of adequate data and other periods (5 min) with 3 60-second epochs of adequate data were submitted to analysis.

Low frequency blood pressure variability—The beat to beat BP waveform was captured using a Finapres noninvasive BP monitor. The analog BP waveform was captured by the NI A/D board and was sampled at 500 samples/sec. Systolic and diastolic values for each cardiac cycle were identified using custom-written software resulting in a BP-time series. Mean BP and spectral power in the low (0.04–0.15 Hz) frequency band of the blood pressure power spectrum for both SBP and DBP were computed from these time series. Because the servo adjustment of the Finapres monitor was enabled during the last minute of each 300 sec period, spectra were calculated on 240 second epochs using an interval method for computing Fourier transforms similar to that described by DeBoer, Karamaker, and Strackee (deBoer, Karemaker, & Strackee, 1984). Prior to computing Fourier transforms, the mean of the BP series was subtracted from each value in the series and the series then was filtered using a Hanning window (Harris, 1978) and the power, i.e., variance (in $mmHg^2$), over the LF band was summed. Estimates of spectral power were adjusted to account for attenuation produced by this filter (Harris, 1978).

Computation of Reactivity and Recovery

For each variable, reactivity to each task was computed as the difference between the mean value during the task and the mean of the preceding baseline. Recovery was computed as the difference between challenge and recovery period. For each baseline, the two 300 sec epochs were averaged to yield a single value. To increase response stability, data from the arithmetic and Stroop tasks were averaged, as were the recovery periods that followed them (Kamarck et al., 1992). The speech task was treated separately because it was delivered in the seated, not the supine, position. To allow for complete equilibration to the upright

position, data from the first 5 min epoch after tilt were excluded from analysis. No recovery data were collected after tilt.

Statistical Analysis

To examine relationships in the prediction of systolic LF BPV, diastolic LF BPV, and PEP, the data were analyzed by performing separate three-way analyses of variance. Each model examined the prediction by group (aerobic versus strength), session (baseline, post training, and after deconditioning), and period within session (baseline, public speaking, arithmetic, and Stroop tasks, tilt, and recovery), while controlling for gender and age. To model the correlation among repeated measures, an unstructured covariance matrix was used. This matrix was selected according to the Akaike criterion (Akaike, 1974). Comparisons regarding recovery from challenge were also treated separately for speech task and for combined math/Stroop tasks.

For all main effects, an alpha level of less than .05 was considered to be statistically significant. All analyses were conducted using mixed modeling software (SAS 9.2 Proc Mixed) to generate restricted maximum likelihood estimates, using all available data.

Results

Descriptive Data

A total of 149 healthy men (n = 58) and women (n = 91) were randomized and tested before training. Technically acceptable data for analysis of PEP and LF-BPV were available on 144 of these participants. The two groups were well balanced (see Table 1). A total of 101 participants completed training, yielding a dropout rate of 32%, and 87 participants completed deconditioning. The dropout rates were the same for both groups. Dropouts were significantly younger (28.42 years versus 32.17 years, p = .003) and there were significantly more women dropouts (42 versus 17, p = .037). However, they do not differ in other demographic and physical characteristics.

Effect of Aerobic Conditioning

Analysis revealed significant effects of group assignment (F1,503=3.83; P<.001), testing session (F2,503=68.95; P<.001), gender (F1,503=46.16; P<.001) and age (F1,503= 8.84; P<.001) on aerobic capacity. Women had lower VO_2max than did men, which was consistent with the gender differences in the Heart Association fitness standards used as inclusion criteria. Body mass index and age were inversely related to VO_2max. Most importantly, the group X session interaction was highly significant (F2,503=26.80; P<.001). Aerobic capacity increased after training and decreased after deconditioning only in the aerobic-conditioning group.

Impact of Training on PEP Reactivity to and Recovery from Challenge

Figure 1 presents PEP values for the aerobic (top panel) and strength training (bottom panel) throughout the psychophysiology protocol. Table 2 presents the results of the statistical analysis. As the Table indicates and as Figure 1 makes clear, there was a significant effect of period on PEP, as expected. During the speech task, PEP shortened compared to the seated

baseline and recovery period. Similarly, PEP shortened during the aggregated math/Stroop task relative to the supine baseline and recovery periods. Finally, Figure 1 illustrates the effect on PEP of moving from the supine to the 70° head up position. The absence of a significant group X session X period interaction indicates that aerobic training did not differ from strength training in the effect on PEP reactivity to or recovery from psychological or positional challenge.

Impact of Training on LF BPV Reactivity to and Recovery from Challenge

Figures 2 and 3 present low frequency systolic and diastolic pressure variability respectively. For both BPV indices, there was a highly significant effect of period, as there was for PEP. However, the group X session X period interaction failed to achieve significance, indicated that as for PEP, the two training conditions did not differ in their effect on BPV reactivity to and recovery from challenge.

Interestingly, however, as Figures 2 and 3 show, the effect of the speech task was different than that of the aggregated math and Stroop task. Post-hoc contrasts confirm that during the speech task, LF SBPV and DBPV increased compared to baseline (all ps < .01) except for non-significant increases in the strength-training group at times 2 and 3. In contrast, BPV fell in response to the aggregated math/Stroop task (all ps < .001) at in every session in each group.

To assess the relationship between PEP and LF-SBPV or LF-DBPV as putative SNS indices, we computed Pearson correlation coefficients for these variables for each period (14) and each session (3) throughout the study. Of the 84 such Pearsons, only 3 reached statistical significance.

Discussion

Abundant evidence demonstrates that psychological stress increases vulnerability to cardiovascular events. Examples from the real world abound. For instance, on June 30, 1998, the day that England lost a World Cup football match to Argentina in a shoot-out, admission to English hospitals for acute myocardial infarction increased by 25%, compared to control days (Carroll, Ebrahim, Tilling, Macleod, & Smith, 2002). The risk of cardiac emergencies in Munich more than doubled on days when the German Football team was playing in the 2006 World Cup (Wilbert-Lampen et al., 2008). On the day of the 1994 Northridge earthquake in California, the number of sudden cardiovascular deaths increased 6-fold compared to a control period (Leor, Poole, & Kloner, 1996). In a patient undergoing 24-hour ambulatory blood pressure monitoring, systolic and diastolic pressure rose to 150 mm Hg and 122mm Hg respectively and heart rate rose to 150 bpm at the time of the strongest tremor from an earthquake that struck central Italy in March 1998. On the first day of the 1991 Iraqi missile attacks, total mortality in Israel increased by 58% largely due to cardiovascular events (Kark, Goldman, & Epstein, 1995).

Psychological stress in the laboratory, while less extreme, nonetheless produces physiological effects that may be in the causal pathway to CVD events. Laboratory challenges increase heart rate and blood pressure (Al'Absi et al., 1997) as well as

sympathetic nervous system activity measured as norepinephrine measured in serum (Grillot et al., 1995; R.P. Sloan et al., 1996) or spillover rate, indicative of whole body sympathetic activity (Esler et al., 2004) and muscle sympathetic nerve activity (MSNA) (Anderson, Sinkey, & Mark, 1991; Hjemdahl et al., 1989). Mental challenges also lead to withdrawal of cardiac parasympathetic modulation (Gianaros, Van Der Veen, & Jennings, 2004; R. P. Sloan, Shapiro, & Gorman, 1990; R.P. Sloan, Korten, & Myers, 1991; Richard P. Sloan et al., 1997).

Because aerobic exercise is known to promote cardioprotection, we sought to examine whether it might do so by reducing sympathetically mediated responses to and recovery from laboratory challenge. In a randomized controlled trial of aerobic exercise vs. strength training in young, healthy, sedentary adults, we found no evidence that improvements in aerobic conditioning had an effect on myocardial sympathetic nervous system responses to or recovery from psychological or orthostatic challenge. Pre-ejection period, an index of sympathetically driven myocardial contractility, shortened as expected in response to the speech, math, and Stroop tasks, consistent with evidence that these tasks elicit sympathetic activation. However, there was no effect of treatment assignment, indicating that improvements in aerobic capacity did not attenuate the sympathetic response to and recovery from psychological challenge.

Seemingly paradoxically, PEP increased in response to tilt, suggesting a reduction in myocardial sympathetic activation. Positional change is widely recognized to elicit a powerful sympathetic response to compensate for the pooling of blood in the lower limbs (Cooke et al., 1999; Furlan et al., 2000). However, PEP is an unreliable index of myocardial sympathetic nervous system activity in response to postural shifts. Because moving from the supine to the upright position displaces blood to the lower limbs, it lowers cardiac preload. Through the Frank-Starling mechanism, myocardial contractile force is reduced, leading to a lengthening of PEP (Houtveen, Groot, & De Geus, 2005).

Low frequency blood pressure variability, a putative index of vascular sympathetic control, rose as expected in response to positional change. As indicated above, data suggest that laboratory-based psychological challenges also elicit sympathetic activation. While LF-BPV rose in response to and then recovered from speech, it unexpectedly fell in response to math and Stroop and then recovered. Improvements in aerobic capacity had no impact on this pattern of response and recovery.

Relatively few studies have examined the BPV response to psychological challenge and in those few, the findings are inconsistent. Mulder et al. reported that mental stress led to a reduction in BPV in the 0.01–0.06 Hz frequency band but did not alter power in the 0.07–0.14 Hz band (Mulder, Veldman, Ruddel, Robbe, & Muler, 1991). Tulen et al. (Tulen, 1999) also found that 0.07–0.14 Hz systolic pressure variability as well as total systolic pressure variability fell during a 10-min Stroop task administered in both the supine and seated positions, despite the fact that the task produced increases in both SBP (~11%) and DBP (~12%) (Tulen, 1999) but no change in plasma epinephrine and norepinephrine. Fauvel et al. (Fauvel et al., 2000) reported no change in the standard deviation of systolic pressure or in 0.07–0.14 Hz variability in response to a 5-min Stroop task administered to healthy men

studied in the seated position and a systolic pressure increase of 22.4 mmHg (~14%). In contrast, Grillot et al. found that in subjects in supine position, a 10-min Stroop led to an increase in 0.07–0.14 Hz variability and in systolic pressure (31 mmHg, 25%) accompanied by significant increases in plasma epinephrine and norepinephrine. Moreover, the increases in blood pressure variability and epinephrine were significantly correlated ($r = 0.69$) (Grillot et al., 1995).

Competing views of the origins of LF-BPV exist. Some have suggested that it is the product of the feed-forward influence of HRV (Toska & Eriksen, 1993; Veerman, Imholz, Weiling, Karemaker, & van Montfrans, 1994). Others assert that it reflects a central sympathetic oscillator (Cevese et al., 1995; Montano et al., 1992). Still others argue that it merely is a resonance phenomenon, a product of the delay in the β-adrenergic vasoconstrictor response mediated by the baroreceptors (Bertram, Barres, Cuisinaud, & Julien, 1998; Cerutti, Barres, & Paultre, 1994; van de Borne et al., 2001) Hammer, 2005 #8134).

Our findings suggest that none of these accounts is complete and that LF BPV during psychological challenge reflects the interplay of two factors. Parasympathetically-mediated oscillations in HR and the associated changes in cardiac output, under certain conditions, can contribute to oscillations in BP (Toska & Eriksen, 1993; Veerman et al., 1994). Indeed, in the supine position, with relatively little SNS activity, most BPV is a product of the feed-forward influence of HRV (Saul et al., 1991).

During mental stress, HRV falls due to withdrawal of cardiac parasympathetic modulation. As HRV falls, so does the feed-forward contribution to oscillations in BP, thus reducing BPV. However, mental stress also causes LF-BPV-enhancing vascular sympathetic activation commensurate with the degree of stressfulness of the challenge. In our study, the math and Stroop tasks led to relatively small increases in SBP (10 mmHg, 8%), similar to the data of Tulen, who also reported a reduction in BPV. This modest SNS activation is insufficient to offset the feed-forward based reduction in BPV produced by the reduction in HRV.

In contrast, SBP increased substantially more (28 mmHg, 24%) in response to the speech task, suggesting that this task elicited considerably greater SNS activation and by this account, was sufficient to offset the loss of feed-forward influence of HRV on BPV. Thus, in response to this more evocative stress, LF-BPV rose. Grillot et al. similarly reported that LF-BPV rose during a Stroop task that was stressful enough to produce a 31 mmHg SBP increase (Grillot et al., 1995). This interpretation raises questions about the validity of LF-BPV as an unambiguous index of vascular sympathetic modulation, suggesting instead that it reflects both parasympathetic regulation of HR and sympathetic vascular regulation.

Previously, we reported that the PNS responses of subjects in this protocol (R. P. Sloan et al., 2011) and demonstrated that there were no training group differences in PNS reactivity to or recovery from psychological or orthostatic challenge. Here we report no training group effect on SNS activation during psychological challenge. Collectively, these findings support for the conclusion that the mechanism of exercise-induced cardioprotection is not attenuation of the autonomic response to or recovery from challenge, at least those that can

be delivered in a laboratory. This conclusion is consistent with other evidence, too. Ray and Carter recently reported the results of a small study demonstrating that although aerobic training resulted in an 18% improvement in aerobic capacity, it had no effect on MSNA responses to mental arithmetic (Ray & Carter, 2010). In samples of similar aged and poorly conditioned participants, de Geus et al. found no effect of training on PEP (E. J. de Geus, van Doornen, & Orlebeke, 1993; E. J. C. De Geus, van Doornen, de Visser, & Orlebeke, 1990). In a small training study, the increased plasma epinephrine response to mental challenge was not altered by improvements in aerobic capacity. Plasma norepinephrine (NE) did not increase in response to mental challenge but as expected, moving to the upright position lead to an increase in NE but this effect was not changed by training (Cleroux et al., 1985). In a small sample of young Black men, aerobic training did not change BPV in the 0.04–0.15 Hz frequency band in response to the Stroop task (Bond et al., 2008).

Some studies have presented findings consistent with the hypothesis that improvements in aerobic fitness attenuate responses to psychological challenge. For example, Hamer and Steptoe reported that greater fitness was associated with smaller parasympathetically mediated reductions in HRV during mental stress. However, these data were based on cross-sectional analyses rather than from a randomized controlled trial (Hamer & Steptoe, 2007). In a six-week randomized trial of aerobic training vs. strength training or a no treatment control condition, the heart rate and rate pressure product response to and recovery from mental stress were reduced in the aerobic group but not in other two groups (Spalding, Lyon, Steel, & Hatfield, 2004). Other indices of autonomic responses to challenge were not collected in this study.

Limitations

Participants had limited oversight during the study. They received an initial training session with a research assistant but then exercised on their own. During training sessions, they measured their heart rate by palpation to determine if they were exercising at the recommended intensity. These limitations notwithstanding, the improvements in aerobic capacity in the aerobic but not the strength training group suggest that participants exercised as instructed.

It also is possible that PEP and LF-BPV are not valid indices of myocardial and vascular sympathetic drive, respectively. Supporting evidence differs for the two. In the case of PEP, pharmacological and clinical studies demonstrate that it provides an index of sympathetic modulation of myocardial contractility. Beta-adrenergic blockade lengthens PEP, as expected (Cacioppo et al., 1994; Winzer et al., 1999). In clinical conditions known to be associated with elevations in sympathetic activity such as diabetes (Berntson, Norman, Hawkley, & Cacioppo, 2008; Licht et al., 2010) and metabolic syndrome (Licht et al., 2010), PEP is reduced, as expected. Thus, it is unlikely that the failure to find an effect of aerobic training on myocardial sympathetic modulation is due to shortcomings of PEP as a valid index.

Evidence that LF-BPV is an index of vascular sympathetic activity is more limited. In rats, phentolamine decreased mid-frequency blood pressure variability, consistent with the hypothesis that it reflects β-adrenergic-mediated vasomotor function (Yoshimoto et al.,

2011). Other blockade studies also support this view (Julien, Malpas, & Stauss, 2001; H. M. Stauss & Kregel, 1996; H. M. Stauss, Persson, Johnson, & Kregel, 1997; Zhang et al., 2002). Moreover, low frequency blood pressure oscillations are consistent with the time delays that characterize sympathetic modulation of vascular tone (Harald M Stauss, 2007). However, other studies suggest that LF-BPV is merely is a resonance phenomenon, a product of the delay in the β-adrenergic vasoconstrictor response mediated by the baroreceptors (Bertram et al., 1998; Cerutti et al., 1994; deBoer et al., 1987; Hammer & Saul, 2005; van de Borne et al., 2001). Evidence also suggests that under certain conditions, LF-BPV is the product of the feed-forward effects of RR interval variability. Thus, the status of LF-BPV as an index of cardiovascular sympathetic modulation is less well established than PEP.

Finally, because the psychological challenges were presented three times over the course of the study, it is possible that participants habituated to them, thus obscuring group differences in response to training. Examination of the figures reveals that the PEP response to the speech task was smaller during the second and third testing sessions. However, because the PEP responses to the math/Stroop task and the LF-BPV responses to all challenges were not appreciably different across the testing sessions, it is unlikely that habituation accounts for the failure to demonstrate training group differences in these indices.

To conclude, these findings, from a large randomized controlled trial using an intent-to-treat design, show that moderate aerobic exercise training has no effect on PEP and LF BPV reactivity to or recovery from psychological or orthostatic challenge. While this study raises questions about whether LF-BPV is an unambiguous index of vascular sympathetic activity, the PEP findings suggest that at least in healthy young adults, the cardioprotective effects of exercise training are unlikely to be mediated by attenuation of sympathetic nervous system responses to stress.

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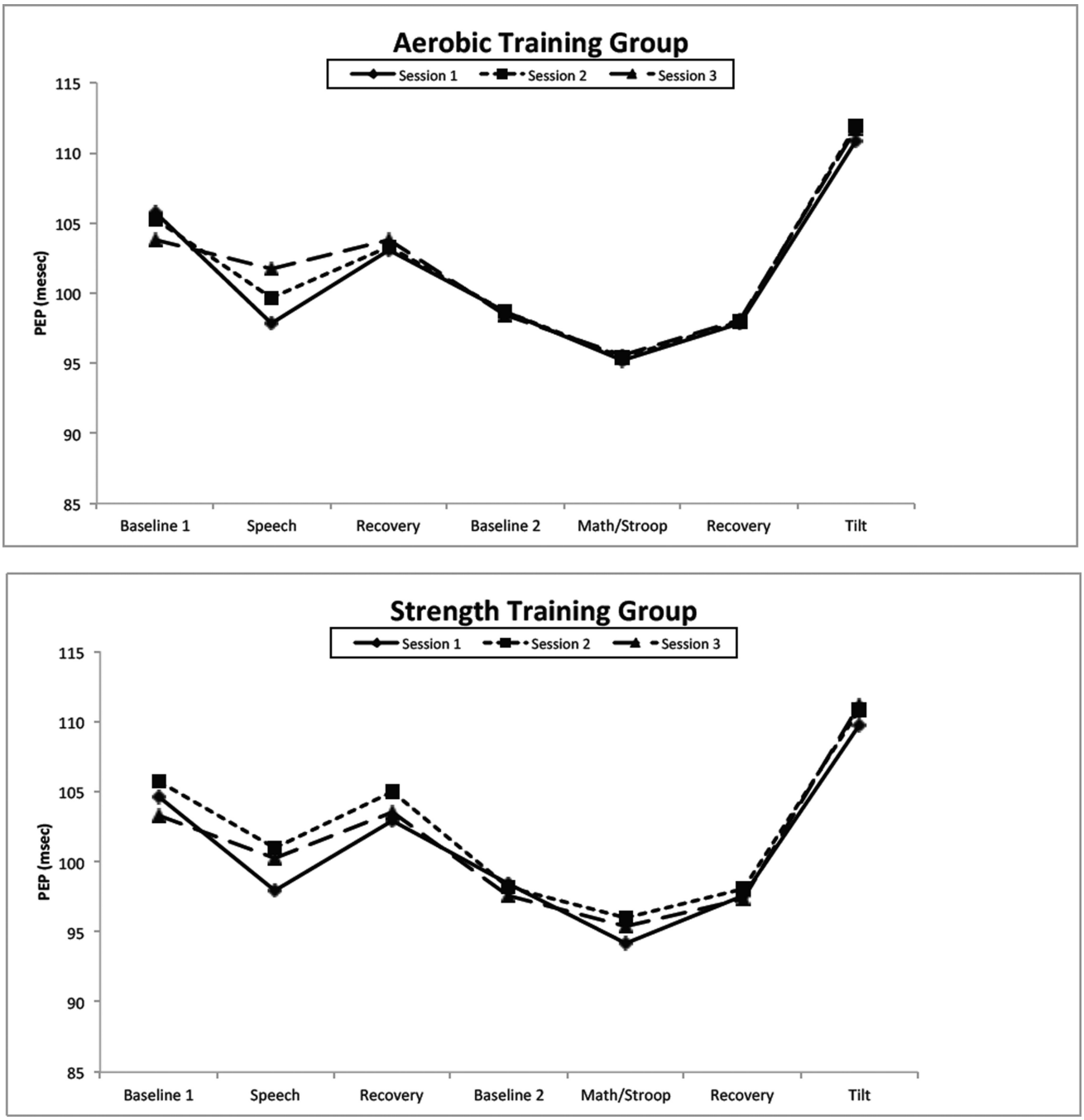


Figure 1.

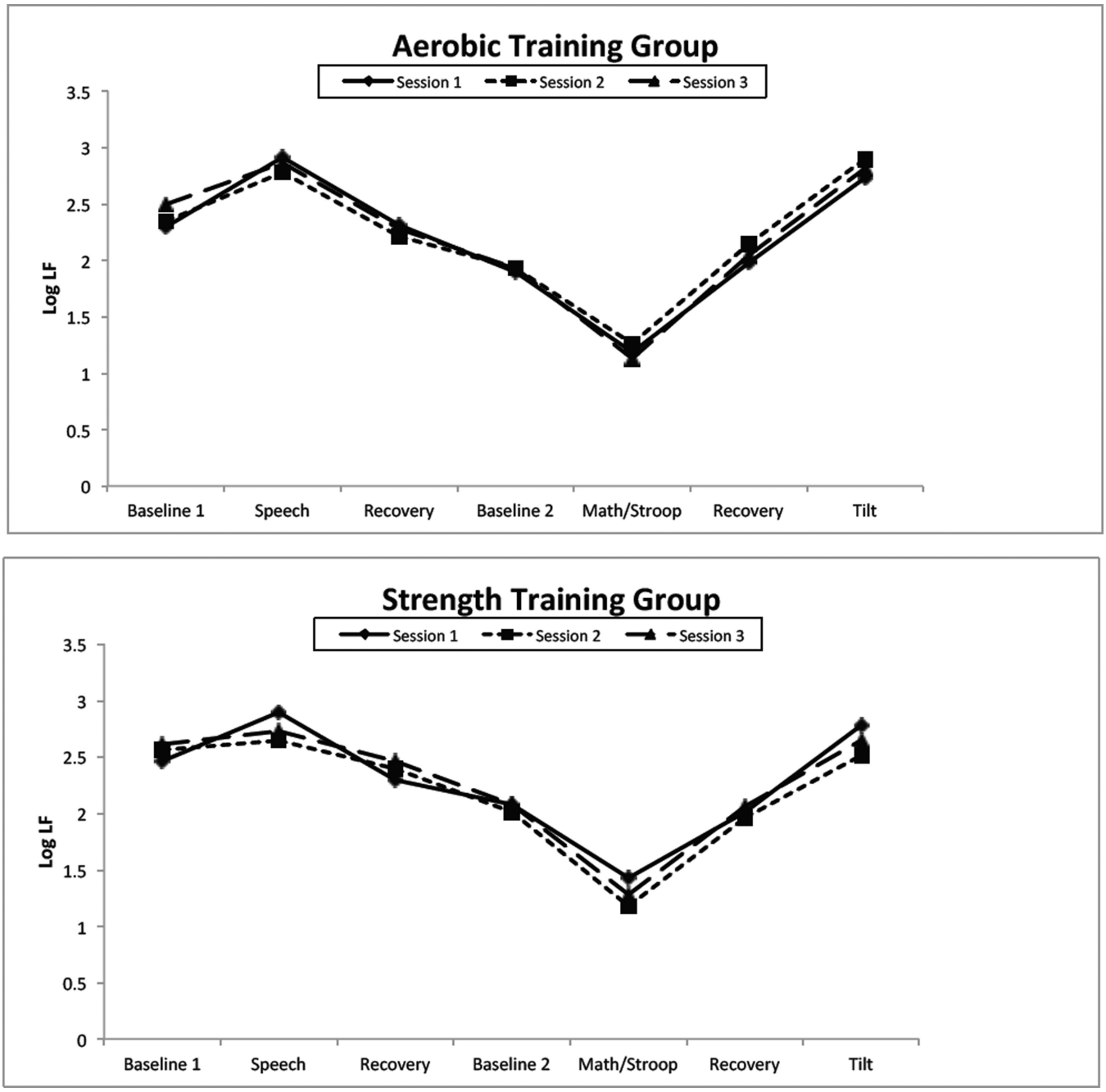
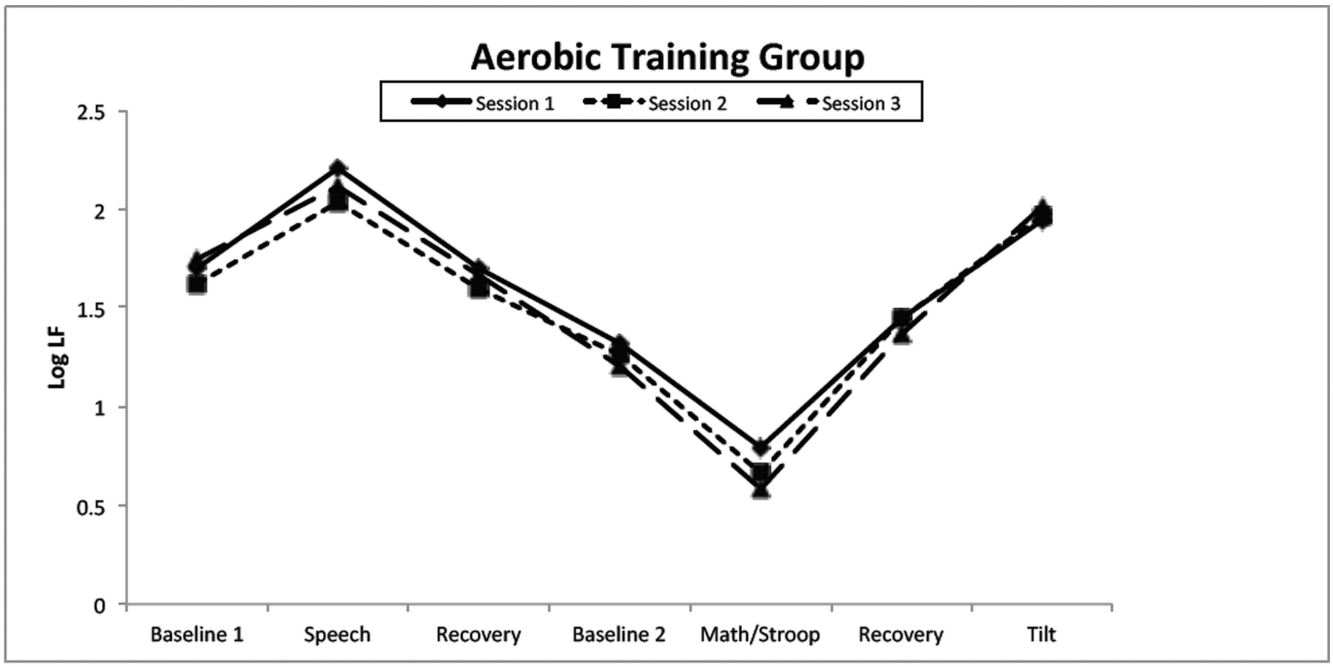


Figure 2.



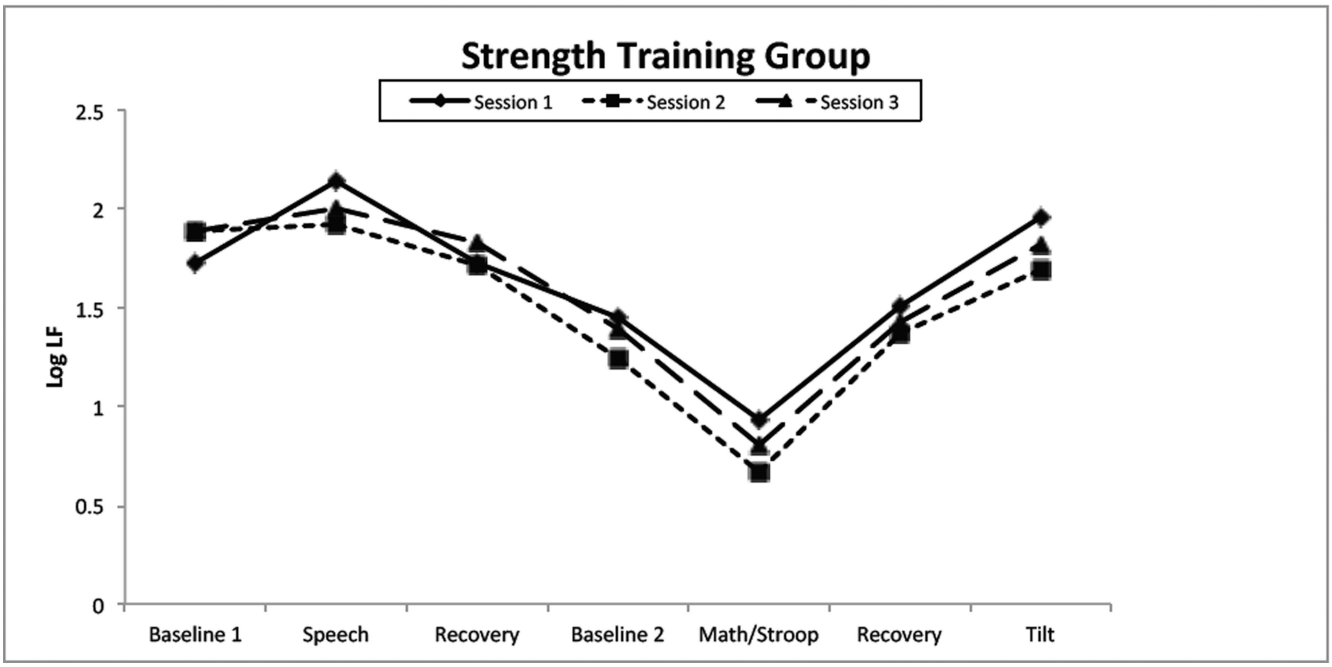


Figure 3.

Table 1

	Group=Aerobic			Group=Strength			
Variable	**N**	**Mean**	**SE**	**N**	**Mean**	**SE**	**p-value**
Age (years)	71	30.28	0.83	73	31.29	0.92	.42
VO_2max (mL/kg/min)	71	34.07	0.73	73	33.10	0.65	.32
SBP (mmHg)	71	109.82	1.26	72	110.25	1.40	.82
DBP (mmHg)	71	71.11	0.93	72	71.22	1.01	.94
HR (bpm)	71	70.97	1.07	73	71.84	1.07	.57
BMI (kg/m^2)	70	24.59	0.45	73	24.98	0.49	.55

Table 2

Type III F-Tests for Between-Time Models

	PEP	LF-SBPV	LF-DBPV
Session	1.21	0.42	3.47*
Group	.03	0.15	0.17
Period	126.98***	210.61***	180.77***
Group*Session	.36	1.04	0.52
Session*Period	4.65***	1.30	1.59
Group*Period	0.66	2.80*	2.68*
Group*Session*Period	0.91	1.26	1.38

All models adjusted for age and gender.

† p<.10

* p<.05

** p<.01

*** p<.0001

PEP = pre-ejection period; LF= low frequency; SBPV=Systolic Blood Pressure Variability; DBPV=Diastolic Blood Pressure Variability