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Hemodynamic Observations on a Yogic Breathing Technique Claimed to Help Eliminate and Prevent Heart Attacks: A Pilot Study

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ABSTRACT

Objective: This pilot study investigated the hemodynamics of a yogic breathing technique claimed "to help eliminate and prevent heart attacks due to abnormal electrical events to the heart," and to generally "enhance performance of the central nervous system (CNS) and to help eliminate the effects of traumatic shock and stress to the CNS."

Design: Parameters for (4) subjects were recorded during a preexercise resting period, a 31-minute exercise period, and a postexercise resting period.

Settings/location: Parameters for subjects were recorded in a laboratory at the University of California, San Diego.

Subjects: Parameters for 3 males (ages 44, 45, 67) and 1 female (age 41) were recorded. One (1) subject (male age 45) had extensive training in this technique.

Interventions: This yogic technique is a 1 breath per minute (BPM) respiratory exercise with slow inspiration for 20 seconds, breath retention for 20 seconds, and slow expiration for 20 seconds, for 31 consecutive minutes.

Outcome Measures: Fourteen beat-to-beat parameters were measured noninvasively and calculated for body surface area to yield: stroke index (SI), heart rate (HR), cardiac index, end diastolic index, peak flow, ejection fraction, thoracic fluid index, index of contractility, ejection ratio, systolic time ratio, acceleration index, and systolic, diastolic, and mean arterial pressures (MAPs). Left stroke work index (LSWI) and stroke systemic vascular resistance index (SSVRI) were calculated.

Results: We report on SI, HR, MAP, LSWI, and SSVRI and how they can help to describe hemodynamic-state changes. This technique induces dramatic shifts in all hemodynamic variables during the 1 BPM exercise and can produce unique changes in the postexercise resting period after long-term practice that appears to have a unique effect on the brain stem cardiorespiratory center regulating the Mayer wave (0.1–0.01 Hz) patterns of the cardiovascular system.

Conclusions: Preclinical studies are warranted to examine the possible long-term effects of this technique that appear to reset a cardiorespiratory brain-stem pacemaker. We postulate that this effect may be the basis for the purported yogic health claim.

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INTRODUCTION

The effects of respiration on the cardiovascular system were first reported 270 years ago in the scientific literature. In 1733, Hales (Hales, 1733) described the fluctuation of arterial pressure with respiration. And von Haller (1760) reported fluctuations of heart rate (HR) with respiration. Approximately 1 century later, Ludwig (1847) reported respiratory-related fluctuations in HR and arterial pressure and showed that HR increases with inspiration and decreases with expiration, with the opposite occurring for blood pressure (BP). However, numerous studies have shown that respiratory movements alone are not solely responsible for the rhythmic patterns of the cardiovascular system but that there is a complex interaction between respiration and circulation (Daly, 1986).

The autonomic, cardiovascular, and respiratory systems appear to be interlocked. Koepchen, Klussendorf, and Sommer (1981) discussed the intracentral coupling of the cardiovascular and respiratory brain-stem control centers and their coupling to autonomic rhythms. They stated that "cardiovascular neuronal activity also can be rhythmic on its own in different ranges of frequencies and also with frequencies in the range of the respiratory rhythm. The interaction in that case is similar to the interaction between coupled oscillators, where in most cases the respiratory rhythm is the leading one. But also the reverse can be observed." Clearly, cardiac and respiratory activities are the result of the mutual interaction of central oscillatory systems and not just the result of respiratory influences on cardiovascular tone.

However, the option of controlling respiratory patterns as a means to influence cardiovascular activity is an attractive one. Hirsh and Bishop (1981) have characterized the effects of tidal volume and breathing frequency on respiratory sinus arrhythmia (RSA) and demonstrated that the relationship is the same independent of whether it is spontaneous or voluntarily controlled. They also showed that RSA amplitude-to-respiratory frequency and RSA amplitude-to-respiratory volume relationships appear to be independent. Novak, Novak, De Champlain, Le Blanc, Martin, and Nadeau (1993) showed that a systematic slowing of respiration can pace hemodynamic fluctuation. They used equal times for inspiration to expiration and studied the changes in the low–(0.015-0.15 Hz) to high–(0.15-0.3 Hz) frequency rhythms. Using beat-to-beat measures, these researchers demonstrated how lengthening the respiratory period from 2.17 seconds to 20 seconds over an 8.5-minute interval paced the respiratory fluctuations in HR intervals, systolic blood pressure (SBP), and diastolic blood pressure (DBP) over the entire range of respiratory periods. Shannahoff-Khalsa, Kennedy, and Ziegler (1993) have studied the effects of varying the inspiration to expiration ratio for 10-minute periods using beat-to-beat measures to explore hemodynamic changes. These researchers compared two patterns with baselines. The first pattern used an inspiration period of 5 seconds with an expiration period of 20 seconds and the second pattern was the reverse. The researchers found that the prolonged expiration-to-inspiration pattern had the larger effect on the hemodynamic state which led to a 12% decrease in SI and a 6.5% increase in mean arterial pressure (MAP) compared to a 2% decrease in SI and a 3.4% increase in MAP. Yogis believe that the pattern with longer expiration produces a more "relaxing" effect on the body and that the pattern with prolonged inspiration is more "energizing."

Lepicovska, Novak, Drozen, and Fabian (1992) studied the effects of a continuous pattern of 8 seconds of inspiration with 32 seconds of breath holding and 8 seconds of expiration for 25 minutes on BP and HR in healthy subjects. The researchers showed the entrainment of the slow 0.03-Hz oscillations by repetitive breath holding and the occurrence of 0.1 Hz and 0.2 Hz respiratory components in both HR and BP. Telles and Desiraju (1991) reported the effects of two different yogic respiratory patterns on oxygen consumption. Both patterns were used for 4 minutes and compared to baselines. The first pattern had an inspiration, to hold, to expiration pattern of "about 1:1(or less):1" at about 3.2 breaths per minute (BPM). The second pattern "was about 1:4:2" with about 2.2 BPM. The first pattern increased oxygen consumption by 52% compared to baseline and the second pattern reduced oxygen consumption by 19%. These results show that slightly different patterns can lead to very different oxygen consumption rates. Most recently, Miyamura, Nishimura, Ishida, Katayama, Shimaoka, and Hiruta (2002) demonstrate the effects of 1 BPM for an hour with a single highly trained subject. The researchers found oxygen consumption at 256 mL/minute and a HR mean of 75 compared to a resting respiratory rate of 6 breaths/minute, 230 mL/minute, and an HR mean of 69. They described the respiratory maneuver as a "thoracic type of breathing; abdominal muscles play a passive role . . . a very deep inspiration is taken slowly with the glottis partially closed and the head erect. When the lungs are full, the head is bent forward until the chin touches the jugular notch firmly. After slow and deep expiration a new cycle is begun without pause." They also measured arterial blood CO_2 and O_2 partial pressure, oxygen saturation, and hydrogen ion concentration. They conclude that reduced hypercapnic chemosensitivity in the well-trained yogi may be related to an adaptation to low arterial pH and/or to high partial pressure CO_2.

One popular yogic breathing pattern uses the selective use of one nostril to differentially affect autonomic tone. Unilateral forced nostril breathing (UFNB) through the right nostril at either 6 BPM or 2–3 breaths per second (a yogic breathing pattern called "breath of fire" or *kapalabhatti*) increased HR compared to left UFNB, while left UFNB comparatively increased end diastolic volume (Shannahoff-Khalsa and Kennedy, 1993). These two techniques differ-

entially stimulate lateralized sympathetic tone and probably the sino–atrial or atrio–ventricular nodes, respectively, thus demonstrating other unique autonomic-related respiratory effects on cardiovascular function.

The pilot data presented here is an effort to investigate the possible hemodynamic changes using another yogic respiratory exercise that requires a 1 BPM rate for prolonged periods. The pattern involves an inspiration period lasting 20 seconds, with breath retention for 20 seconds, followed by expiration over 20 seconds while sitting erect. This pattern (20:20:20 breath) when perfected supposedly both "helps eliminate and prevent heart attacks that can be triggered by abnormal electrical events" (personal communication, Yogi Bhajan, Master of Kundalini Yoga) and is also said to "help eliminate the effects of traumatic shock and stress to the central nervous system."

MATERIALS AND METHODS

Subjects

Three males, ages 44, 45, and 67, and one female age 41 volunteered and were paid 25 dollars to participate. All subjects had significant experience (1–20 years) with a variety of yogic breathing techniques. However, only subject 1 (a male, age 45) had practiced this particular technique more than once prior to the experiment. The other 3 subjects, while being highly accomplished in similar but different yogic breathing techniques, were able to complete this specific 1 BPM pattern successfully at their first attempt without difficulty prior to the experiment. This was required to assure themselves and the authors that they would be able to repeat this performance under laboratory conditions without duress.

Equipment and calculations

All subjects were studied with simultaneous beat-to-beat recordings from both the BoMed NCCOM3-R7 Cardiodynamic Monitor (CardioDynamics International Corp., San Diego, CA) and the Finapres Blood Pressure Monitor 2300 (Ohmeda, Louisville, CO). The BoMed NCCOM3-R7 monitor measures thoracic electrical bioimpedance noninvasively and can measure global blood flow and parameters of left ventricular performance with adequate clinical accuracy (Sramek, 1988a, 1988b, 1991). Beat-to-beat values were recorded for stroke index (SI) (SI = stroke volume/body surface area [BSA]) (where SV [mL] = VEPT × VET × IC, VEPT = volume of electrically participating tissue, VET = ventricular ejection time, IC = index of contractility [s^{-1} = $(dZ/dt)_{max}$/TFI, TFI = thoracic fluid index]); HR (beats/minute); cardiac output, (CO) (liters/minute) = HR × SV; end-diastolic volume, EDV (mL) = 100 × SV/EF; peak flow, PF (mL/second = VEPT × IC × constant; ejection fraction, EF (%) = SV/EDV = (0.84 − [0.64 × STR]) × 100; thoracic fluid index, TFI (ohms) = Z_o = total impedance of thorax; index of contractility, IC (sec^{-1}) = $(dZ/dT)_{max}$/TFI; ejection ratio, ER (%) = 100 × VET/HRP, HRP = 60/HR; systolic time ratio, STR (%) = PEP/VET × 100, PEP = preejection period (second); and acceleration index, ACI (sec^{-2}) = (d2Z/dt2)max/TFI. In this study we used SI instead of SV to help compare values across subjects. The Finapres measures SBP, DBP, MAP, and HR at beat-to-beat intervals. Recording at beat-to-beat intervals made it possible to also calculate systemic vascular resistance index [SVRI = 80 × (MAP-3/CO)/CI {dyn × sec × cm^{-5} × $meter^2$}]; stroke systemic vascular resistance index [SSVRI = MAP − 3)/SI × 80 {dyn × sec × cm^{-2}}]; left cardiac work index [LCWI = (MAP − 6) × CI × 0.0144 {kg × m}]; and left stroke work index [LSWI = (MAP − 6) × SI × 0.0144 {g × m}] (Sramek, 1995, 2002). All data (except for that of subject 1, run 1) was collected in an "indexing" mode that is a calculation based on body-surface area to allow comparable measures between subjects. Also, run 1, subject 1, was the only recording with only the BoMed NCCOM3-R7 Cardiodynamic Monitor.

The computer and software for the BoMed NCCOM3-R7 and Finapres recordings was developed by the Sleeptrace Corp. (Dallas, TX). Data analysis was performed using DaDisp 2002 Software, (DSP Development Corp., Cambridge, MA) and Spectre, Version 5.0, (Solana Beach, CA). Graphics were displayed using Slide Write Plus Version 6.1 (Advanced Graphics Software, Encinitas, CA) and Harvard ChartXL, version 2.0 (Harvard Graphics, Hudson, NH).

Each subject was seated in a chair at a desk and had chest and neck electrodes applied (Littman 3M Snap diagnostic EKG electrodes, No. 2350). The Finapres cuff was applied to the third digit of the left hand between the first and second joint. After a minimum of 30 minutes of rest a baseline was recorded for 10–20 minutes followed by the 31-minute respiratory exercise (20:20:20 breath), followed by 10–20 minutes of final baseline. Subjects 2–4 with little laboratory recording experience had 20-minute pre- and postrecording periods to help ensure acclimatization and a resting state. However, only the last 10 minutes of the preexercise period and the first 10 minutes of postexercise rest periods were analyzed to match the 10-minute pre- and postexercise data sets of subject 1 who had long-term experience with this experimental set up. Each subject was trained to observe a digital timer to monitor the respective 20-second respiratory phases. The subjects were observed for compliance. Eyes were open during the experiment and it was conducted in an air-conditioned room at 23°C.

RESULTS

The time series of subject 1 for SI, HR, and MAP for the 10-minute preexercise, 31-minute 20:20:20 breath exercise,

and 10 minute postexercise periods, respectively, are presented in Figures 1, 2, and 3 for the third experimental run with this subject. The raw beat-to-beat data had been treated with a rolling average over 25 points to help elucidate the endogenous Mayer wave activity exhibited at approximately 0.1–0.01 Hz that clearly exhibits a wide variance under normal resting conditions. In Figure 2, note that, during the 20:20:20 breath exercise period, the three cardiovascular measures all show a unique entrainment with the respiratory cycle and a dramatic variance compared to resting states. However, while the 1-BPM exercise entrains each cardiovascular measure during the exercise period, the Mayer wave frequency pattern is not linked to the resting respiratory cycle during the preexercise period. The Mayer wave is always slower than, and independent of, the respiratory rate (Polosa, 1984). Figure 3 also shows there is a unique sinusoidal Mayer wave pattern that is most clear in the SI and HR data in the postexercise period. We consider this to be an important and perhaps informative result. This effect was only observed after long term practice by this subject. The results of the other three subjects do not exhibit this unique sinusoidal result in either the pre- or postexercise periods. Figure 4 shows the pre- and postexercise SI patterns for all 4 subjects that first include the three separate runs for subject 1 (in descending order based on the time intervals between practice) recorded at different periods during his

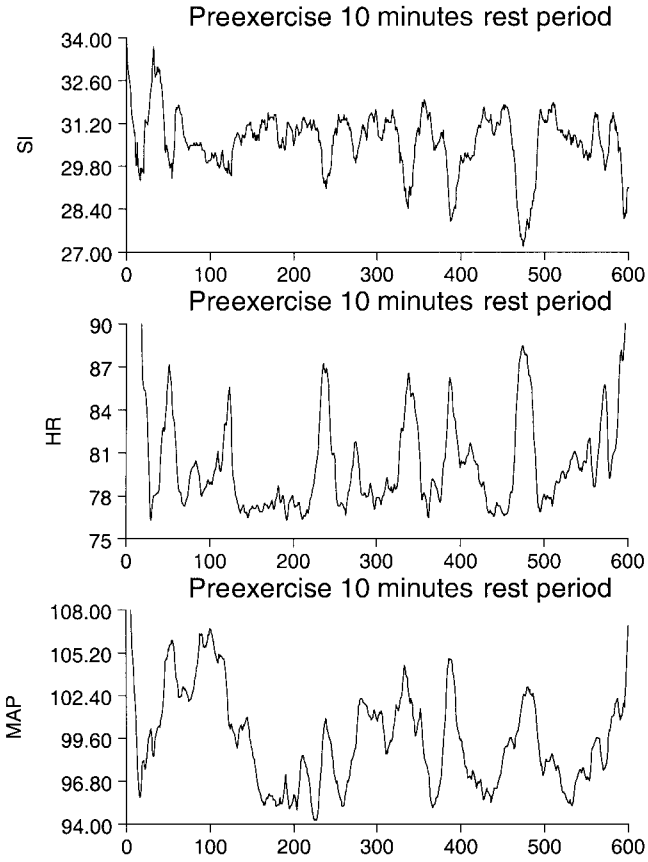

FIG. 1. Three graphs of simultaneous measurements from the BoMed NCCOM3-R7 Cardiodynamic Monitor (CardioDynamics International Corp., San Diego, CA) and the Finapres Blood Pressure Monitor (Ohmeda, Louisville, CO). Stroke index (SI) and heart rate (HR) were captured from the BoMed monitor and mean arterial pressure (MAP) was captured by the Finapres monitor. The beat-to-beat data from each instrument were then subjected to a rolling average over 25 data points to help exhibit the natural endogenous Mayer wave rhythmic activity (0.01 Hz–0.1 Hz). The top graph shows 10 minutes of resting SI data, the middle graph shows HR, and the bottom shows MAP. These data are for subject #1 for the 10 minutes of preexercise rest; it is the third experiment conducted after extensive practice of the 20:20:20 breath technique.

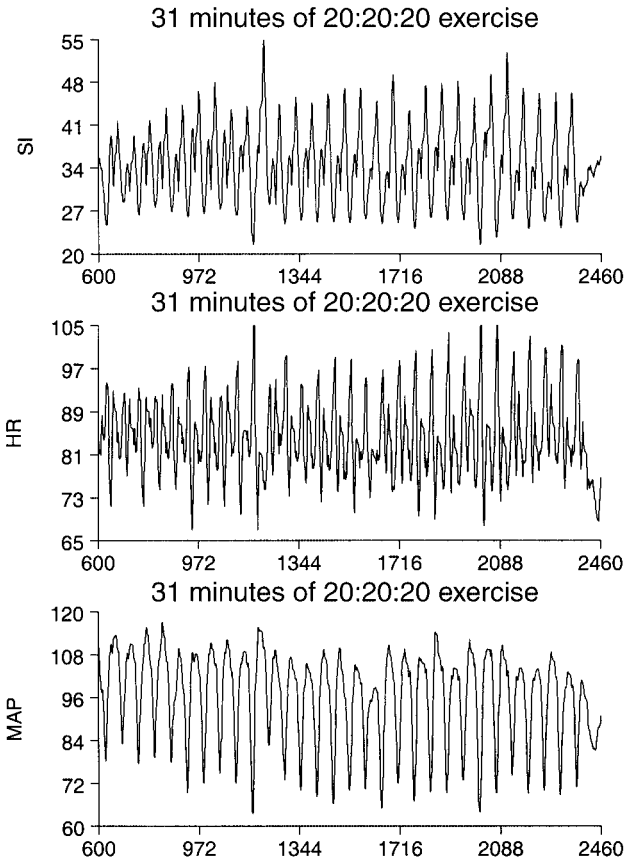

FIG. 2. Three graphs of simultaneous measurements from the BoMed NCCOM3-R7 Cardiodynamic Monitor (CardioDynamics International Corp., San Diego, CA) and the Finapres Blood Pressure Monitor (Ohmeda, Louisville, CO). Stroke index (SI) and heart rate (HR) were captured from the BoMed monitor and mean arterial pressure (MAP) were captured by the Finapres monitor. The beat-to-beat data from each instrument were then subjected to a rolling average over 25 data points to help exhibit the natural endogenous Mayer wave rhythmic activity (0.01 Hz–0.1 Hz). The top graph shows the 31-minutes of the 20:20:20 breath SI data, the middle graph shows HR, and the bottom shows MAP. This data are for subject #1 for the 31 minutes of the exercise period; it was the third experiment conducted after extensive practice of the 20:20:20 breath technique.

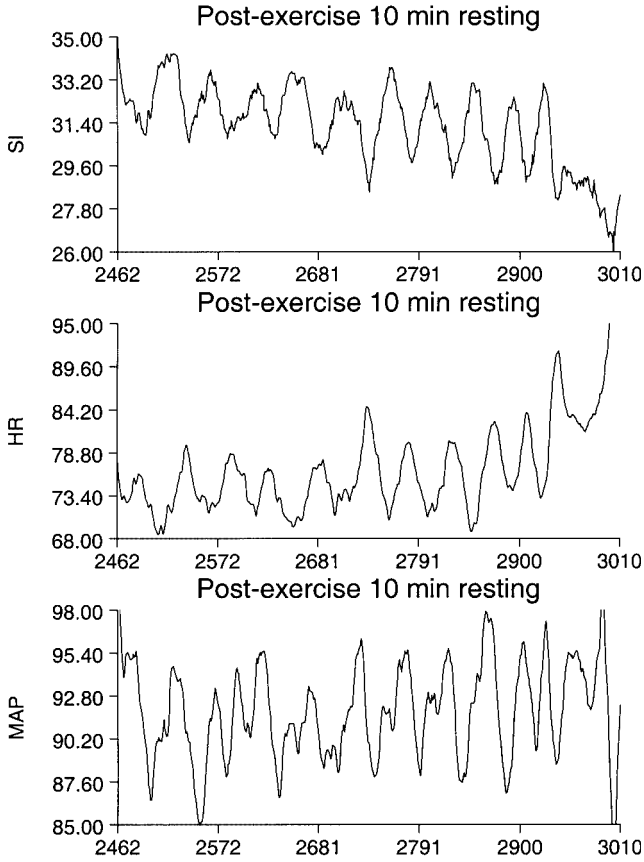

FIG. 3. Three graphs of simultaneous measurements from the BoMed NCCOM3-R7 Cardiodynamic Monitor (CardioDynamic International Corp., San Diego, CA) and the Finapres Blood Pressure Monitor (Ohmeda, Louisville, CO). Stroke index (SI) and heart rate (HR) were captured from the BoMed monitor and mean arterial pressure (MAP) was captured by the Finapres monitor. The beat-to-beat data from each instrument were then subjected to a rolling average over 25 data points to help exhibit the natural endogenous Mayer wave rhythmic activity (0.01 Hz–0.1 Hz). The top graph exhibits the 10 min of the postexercise resting SI data, the middle graph shows HR, and the bottom shows MAP. These data are for subject #1 for the 10 minutes of the postexercise period; it is the third experiment conducted after extensive practice of the 20:20:20 breath technique. Note the highly sinusoidal Mayer wave pattern in all three parameters.

training followed by the male subject age 67, the female subject age 41, and the male age 44. It is apparent that only run three in subject 1 (age 45) demonstrates this sinusoidal effect and it occurs only in the postexercise phase. This unique sinusoidal-like result does not occur in the data of the other runs and there is a slow progression toward this sinusoidal effect that appears to emerge somewhat in run 2 compared to run 1 for subject 1, but clearly exhibits in run 3. The time between run 1 and run 2 is approximately 8 months, and the time between run 2 and 3 is 1 month. The practice schedule for subject 1 between runs 1 and 2 was sporadic with a frequency of about one time per week, the practice schedule between runs 2 and 3 was about once every 2–3 days.

Color figure 5 (subject 1, runs 2 and 3 and subjects 2, 3, and 4) shows 3-dimensional plots of SI (x-axis) versus MAP (y-axis) versus HR (z-axis) for the three respective recording periods of the experiment. The data here for subject 1, run 1, was recorded with only the BoMed NCCOM3-R7 and not the Finapress measures, and thus is not profiled because

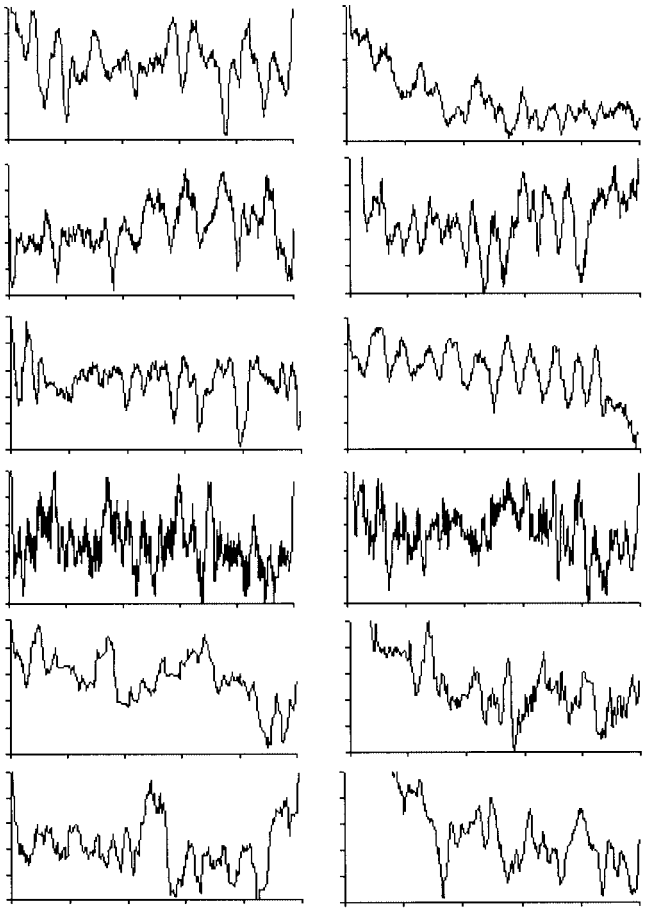

FIG. 4. Two columns and six rows of data. The left column is from the 10-minute preexercise resting data for stroke index (SI). The right column is from the 10-minute postexercise resting data for SI. The left and right graphs in the same row are from the same subject in one experiment, before and after the 20:20:20 breath exercise. The SI data are expressed here as the rolling average over 25 points of the raw data. The first row is subject 1 (experiment 1), run before significant or any regular practice of the 20:20:20 breath exercise. Row 2 (experiment 2) is subject 1 approximately 8 months after weekly practice, and row 3 (experiment 3) is 1 month after experiment 2 in which there was practice at a frequency of approximately once every 2–3 days. Row 4 is a male subject (age 67), row 5 is a female (age 41), and row 6 is a male subject (age 44). These three subjects were all well-trained in slow-breathing techniques at 1 BPM but after only one prior run with this specific breath exercise technique before the laboratory recording. The ticks on the x-axis are 2-minute markers. The data are displayed to help accentuate the Mayer wave patterns of activity. Note the occurrence of the unique sinusoidal pattern in row 3, column 2.

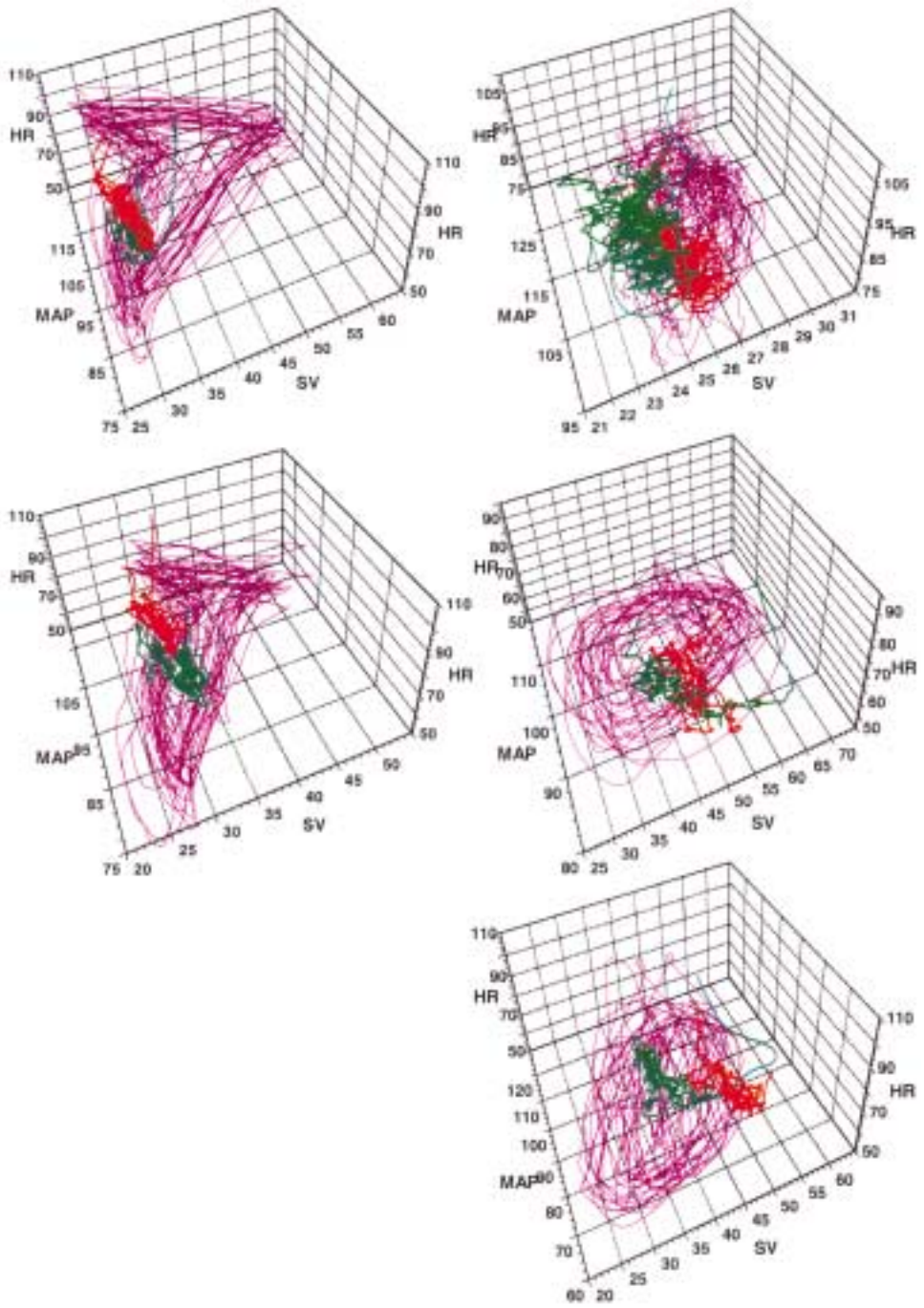

FIG. 5. Figure 5 shows five 3-dimensional color plots. There are 2 columns, in the left column there are 2 figures, the upper figure is of subject 1, experiment 2, and the bottom left figure is subject 1, experiment 3. The right column shows 3 subjects: top, male age 67; middle, male age 44; and bottom, female age 41. The x-axis is the stroke index (SI) data paired with the y-axis data of the mean arterial pressure (MAP) measure plotted against the z-axis of HR. Each point is an x-y-z plot of the simultaneous measures of SI, MAP, and HR. The red lines are from the 10-minute preexercise resting data. The green lines are from the 10 minutes of postexercise resting data, and the purple lines are from the 31 minutes of the 20:20:20 breath exercise data. Note the return maps for all 3 phases of the experiment each have their own respective loci of points and that the exercise phase has a much larger excursion of data points to define the hemodynamics during that phase of the study. Also, note that the postexercise phase of the recordings usually is more central to the 3-dimensional figure. This may indicate a more normalized hemodynamic state. Further studies are required to help determine a "normalized" 3-dimensional expression of the hemodynamic state.

MAP is missing. The beat-to-beat data is plotted again using the smoothing average of 25 to help elucidate the basic patterns of cardiovascular activity. These plots help to define the hemodynamic state better during the three phases of the experiment and clearly show that the three variables interact differently in these three phases when using this 3-dimensional array. While one variable cannot describe a physiologic state, we present the data here in a way that can help define a hemodynamic state. The red lines represent the 10-minute preexercise resting state, the purple lines represent the 31-minute exercise state, and the green lines represent the final 10-minute postexercise resting state. In addition to the broad areas covered by the 20:20:20 exercise, note the relative differences in the pre- and postexercise periods. The postexercise (green lines) state is usually more central in the 3-dimensional plot.

Hemodynamic analysis of the 20:20:20 exercise for subject 1, run 3 (Figure 6)

In Figure 6, the starting point of the 20:20:20 breath exercise, marked as point 1 in the two dimensional plot (be-

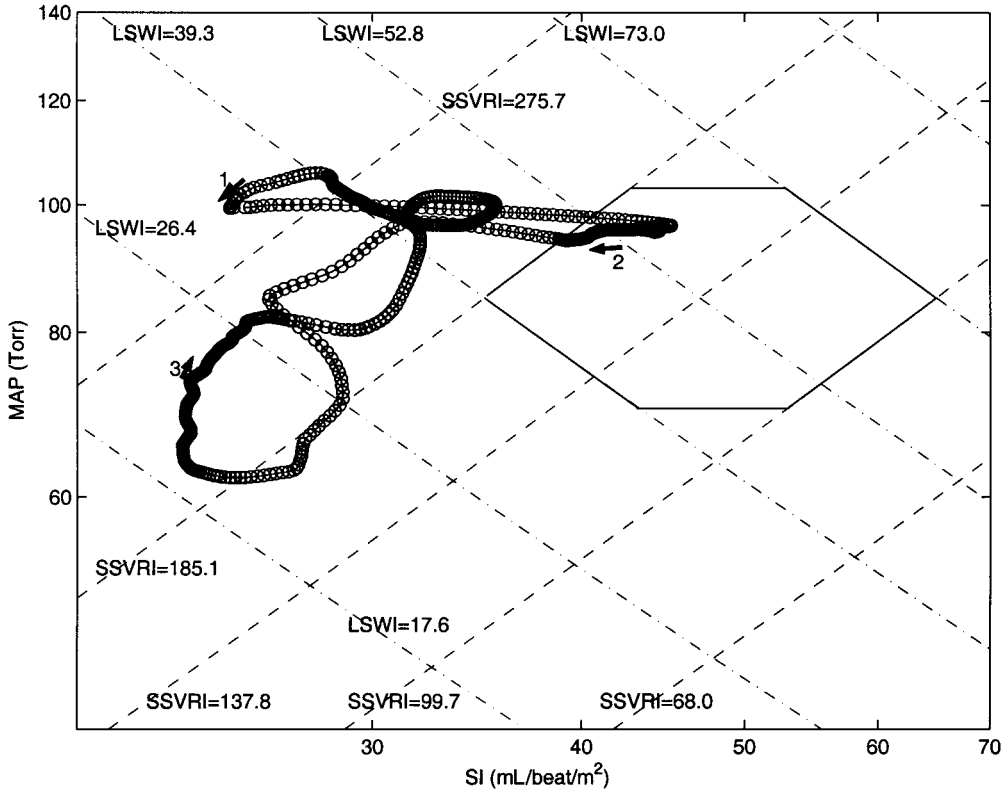

FIG. 6. This is a hemodynamic management chart structured for beat-to-beat parameters (see Sramek 1988b, 2002). Subject 1's (run 3) hemodynamic state is presented while performing the 20:20:20 breath exercise in an erect sitting posture plotted in an orthogonal system with the x–y coordinates of mean arterial pressure (MAP) and stroke index (SI). The south-west to north-east diagonal lines are isolines of systemic vascular resistance index (SSVRI), documenting the effects of vasoactivity (afterload) on the hemodynamic state. The north-west to south-east diagonal lines are isolines of left stroke work index (LSWI), documenting the combined effects of volume + inotropy on the hemodynamic state. The center of the hexagon represents the ideal normotension (ideal MAP) and the normodynamic state/beat (ideal SI) for a supine resting adult with normovasoactivity, normovolume, and normoinotropy. The return map here for Subject 1 performing the 20:20:20 breath exercise is shown as a series of intersecting circles and the arrows and numbers indicate the starting point (point 1) of the inspiration phase, the starting point of the breath retention phase (point 2), and the starting point for the expiration phase (point 3) for the 31 minutes of the exercise period breathing at one breath/minute. The return map calculation of subject 1, run 3, presented in Figure 6 was created as follows. The raw data for SI and MAP came at every heartbeat interval and were, thus, at uneven time points and, hence, were interpolated linearly into a fine, even-time grid with a resolution of 0.1 second. These fine resolution data were then averaged point-wise over the 31 1-minute exercise cycles, that is, at each moment in the 60 seconds of one 20:20:20 breath cycle, we found the arithmetic average over the 31 replicas to provide the average waveform over the breathing cycle. This was done independently for both SI and MAP and the multiple channels were plotted on different axes to yield a phase–space plot of the average synchronized-to-breathing dynamics. This procedure highlights any consistent behavior over the various phases of the breathing cycle and suppresses replica-to-replica fluctuations. However, using this procedure for cardiac patients may obscure important short-term changes, although it may also help elucidate any consistent abnormal variations.

ginning at inspiration) is defined by the highest intra-thoracic pressure and characterized by the following hemodynamic coordinates: MAP = 99 Torr @ SI = 24 ml/m^2.

Hemodynamics during the 20-second inspiration phase (points "1" → "2")

The 20-second inspiration phase is characterized by a balance between changes in contractility (volume and inotropy) and vasoactivity. The first half of the inspiration phase is characterized by a near doubling of SI (from SI = 24 to SI = 45 mL/m^2) at an almost constant value of MAP (MAP >> 99 Torr). This is caused initially by almost a linear increase in intravascular volume (the result of increasing venous return), followed by a gradually increasing inotropic state. The combined increase in contractility is approximately 85% (from LSWI = 31.8 to LSWI = 58.9 g.m/m^2). This decrease in contractility is accompanied by a near linear 94% increase in vasodilatation (from SSVRI = 316 to SSVRI = 163 dyn.sec.cm^{-5}.m^2), which explains the almost constant MAP value during this phase.

The second part of the 20-second inspiration phase is characterized by ~21% decrease in SI (from 45 to 38 mL/m^2) with a mild decrease (3%) in MAP (from 97 to 94 Torr). This is caused by both a small decrease in intravascular volume (a further inspiratory decrease in intrathoracic pressure does not produce a corresponding increase in venous return) and by a decrease in inotropy. A simultaneous vasoconstriction, however, still maintains MAP at almost the same level.

Hemodynamics during the 20-second breath retention phase (points "2" → "3")

The majority of the breath retention phase is characterized by almost a constant level of vasoactivity (~232 dyn.sec.cm^{-5}.m^2) accompanied by profound changes in volume and inotropy. The intrathoracic pressure remains at its minimal level during this entire phase. Gradually decreasing intravascular volume, which already started at the end of preceding phase, is initially compensated by a small increase in vasoconstriction, producing a small increase in MAP (from 94 to 97 Torr) and a decrease in SI (from 38 to 32 mL/m^2). However, from this point, both MAP and SI drop precipitously to their lowest levels during the entire cycle (to MAP = 63 Torr; SI = 23 mL/m^2). The original cause of this process is a profound volume depletion at almost a constant level of vasoactivity.

The remainder of the breath retention phase produces a gradual increase in MAP (from 63 to 75) at almost a constant level of SI (SI >> 24 mL/m^2). This is attributable mostly to an increase in inotropic state.

Hemodynamics during the 20-second expiration phase (points "3" → "1")

The increasing intrathoracic pressure in the first half of the expiratory phase is responsible for gradually decreasing venous return, which is accompanied by an increasing level of inotropic state; the vasoactivity, at the same time, stays at almost the constant level (~232 dyn.sec.cm^{-5}.m^2) it was during the breath retention phase. The second half of the expiratory phase is characterized by a still-decreasing venous return, compensated by a further increase in inotropy, so that the total level of the ejection phase contractility is almost constant (LSWI >> 41.5 g.m/m^2); this hemodynamic modulator activity is accompanied by an increase in vasoconstriction (to SSVRI = 316 dyn.sec.cm^{-5}.m^2).

Summary of the hemodynamic changes of the 20:20:20 breath

The "T-shape" return map of the hemodynamic point trajectory during the entire cycle of the 20:20:20 respiratory exercise shows a controlling effect of ejection phase contractility during one part of the 1-BPM cycle while vasoactivity is essentially unchanged. The other part of the cycle is then controlled by a variation in vasoactivity, while the contractility parameters (volume and inotropy) either complement each other or stay at almost constant level. The effects of contractility and the effects of vasoactivity are about 90° out of phase during the 1-BPM cycle. When this 1-BPM respiratory cycle is practiced over an extended period of time, it appears to produce a hemodynamic "flywheel-like" phenomenon, which tries to maintain the 1-minute hemodynamic modulator cycles in the subsequent postexercise resting period. This resetting of the pacemaker functions of the brain stem cardiorespiratory control center may be why this 20:20:20 respiratory exercise has the claimed beneficial effects.

DISCUSSION

It is now well-known that the autonomic nervous system plays a critical role in the genesis of sudden cardiac death, (Lown, 1979; Schwartz and Stone, 1982; Corr et al., 1986; Schwartz et al., 1992). And this is especially true when there is ischemic heart disease already present. Sudden cardiac death resulting from ventricular tachycardia can be induced by an imbalance in sympathetic versus parasympathetic activity. Specifically, sympathetic hyperactivity promotes the occurrence of ventricular tachycardia, (Schwartz and Priori, 1990) and augmented vagal tone exerts a protective and antifibrillatory effect (Vanoli et al., 1991).

The unique and rare result we see in the one case here with the induced sinusoidal effect on the Mayer wave pattern that occurs in SI, HR, and MAP suggests that the brainstem cardiorespiratory control center has been altered here after the long-term practice of the 20:20:20 breath. The Mayer wave was first described in 1876 (Mayer, 1876) and is known to be centrally regulated (Preiss and Polosa, 1974) and is an expression of autonomic activity (Polosa, 1984) mediated through the sympathetic and parasympathetic ner-

vous systems (Polosa, 1984; Koizumi et al., 1984). To our knowledge, this effect on the brainstem control center may be the first example of an endogenous pacemaker of the body being reset using the breath. We believe that this latent effect here is attributable to a resetting of the brainstem control center. The yogic claim is that, in addition to "helping to eliminate and prevent heart attacks," this breath technique also "enhances performance of the CNS and helps to eliminate the effects of traumatic shock and stress to the CNS" (personal communications, Yogi Bhajan). Because Mayer wave patterns have also been observed in the human EEG and are postulated to reflect spontaneous periodic changes of cortical excitability with control at the brainstem level (Novak et al., 1992), it may be that the cortical expression of the Mayer wave activity is also reset with the extensive use of this yogic technique. It may be that during high levels of prolonged stress or under acute severe stress, that breathing patterns become quite irregular, unstable, and potentially lethal under circumstances in which autonomic tone has already been compromised and, thus, this feedback on the brainstem cardiorespiratory control center can then lead to an abnormal electrical event or accident triggering ventricular tachycardias resulting in death. The 20:20:20 breath technique may be a useful tool to help reduce the occurrence of sudden cardiac death and the latent effects of stress on the central nervous system.

Because this technique ultimately requires an ability to reduce respiration to one BPM to obtain the apparent observed benefits, cardiac patients would require substantial training and practice to make maximum use of this technique. However, it is expected that, if the ratio is held constant and equal for the inspiration to breath retention to expiration phases, it may also be a helpful tool for cardiac rehabilitation at rates of less than one BPM in the early phases of training. Preclinical trials are warranted to help further explore the effects of this technique in additional normal subjects along with the versatility and safety of this tool in cardiac patients.

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