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# Biophysiologic Effects of Warm Water Immersion

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Physiologic change associated with aquatic activity has been found to profoundly affect human function and health-related biologic alterations. Similar to sleep research, aquatics has emerged as an area ripe with human health and performance implications. Aquatic activity impacts the cardiovascular, musculoskeletal, autonomic nervous system (ANS) and endocrine systems in ways that have positive public health implications for issues confronting the nation, including obesity, diabetes and arthritis (Becker, 2004). Aquatic activity has tremendous application in the area of sports medicine and has great potential value to student athletes in both training and rehabilitation. The aquatic environment is a research area just emerging as a focus of physiologic importance with many health benefits that apply across the age span and could be widely accessed by the American public if both research support and understanding by the health professionals were to increase.

## Water Immersion and the Body

Immersion produces a dramatic shift of blood from the extremities to the chest, with approximately 2/3 of this volume in lung circulation and 1/3 within the heart (Arborelius, Balldin, Lilja, & Lundgren, 1972; Begin et al., 1976; Christie et al., 1990). This creates a major increase in cardiac filling volume, resulting in increased stroke volume and cardiac output (Begin et al., 1976; Christie et al., 1990). The resulting effect of immersion is that the heart pumps effectively the same amount of blood per minute at rest as it does during the initiation of aerobic exercise. Therefore immersion may be a useful way of beginning cardiac rehabilitation or recovery from severe debility (Cider, Svealv, Tang, Schaufelberger, & Andersson, 2006). At the same time, immersion decreases peripheral resistance, reducing the amount of work the heart must do to move this volume of blood, so the effort required to circulate blood decreases while cardiac efficiency increases (Arborelius et al., 1972; Gabrielsen, Johansen, & Norsk, 1993). This supports the rationale that aquatic exercise may be beneficial for cardiac rehab following ischemic

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heart injury (Gabrielsen et al., 2001; Gabrielsen, Sorensen et al., 2000; Hanna, Sheldahl, & Tristani, 1993; Heigenhauser, Boulet, Miller, & Faulkner, 1977; Jiang et al., 1994; Magder, Linnarsson, & Gullstrand, 1981; McMurray, Avery, & Sheps, 1988; Meyer & Leblanc, 2008).

Circulation to deep muscle structures is also increased significantly in water immersion, improving oxygen flow to tissues and potentially facilitating healing of muscle, bone, and joint injuries (Balldin & Lundgren, 1972; Balldin, Lundgren, Lundvall, & Mellander, 1971). Improved blood flow is also relevant to processes that alter tissue circulation, including diabetes and some auto-immune diseases. Neck-depth immersion may enhance brain blood flow through reduction in peripheral vascular resistance combined with increased cardiac output. This may improve brain functions, including cognition and memory, which could potentially aid in head trauma recovery or stroke rehabilitation (Bonde-Petersen, Schultz-Pedersen, & Dragsted, 1992). Renal efficiency also improves, producing diuresis through increased excretion of sodium and potassium, aiding in reduction of edema when present (Epstein, 1976, 1992).

The autonomic nervous system (ANS) is an important homeostatic mechanism in several of the body's regulatory functions. The ANS is the major control mechanism for cardiovascular regulatory activity, including heart rate and arterial pressure. In addition, it controls the gastrointestinal motility and secretion, renal and bladder function, visual alterations, thermoregulation, and a number of mental processes. Essentially, it functions as the motherboard for human bioregulation. The two major subdivisions of the ANS are the sympathetic and the parasympathetic systems. The functions of the sympathetic nervous system (SNS) are to control the fight or flight responses of the body while the functions of the parasympathetic nervous system (PNS) are to control the relaxation and repose responses. The anatomic location of these systems is in the brainstem, the spinal cord, and the hypothalamus.

The anatomy, interconnections, and neural regulatory mechanisms are quite complex, with many reflex triggers and feed-back mechanisms. The response speed is dramatic, potentially capable of doubling heart rate in a matter of a few seconds. There are two major neurotransmitters activated by the ANS: acetylcholine and norepinephrine, categorized as catacholamines. A number of methods have been used to measure the function of the ANS components, including blood hormones such as corticosteroid and catecholamine levels, galvanic skin responses (polygraphs), salivary cortisols, and heart rate variability (HRV). While measurement of blood hormones is useful, because ANS changes are so instantaneous, a running measurement of sympathetic/parasympathetic influence is technically difficult outside of a laboratory. Polygraphs are used in legal work but not commonly in research applications. HRV has emerged as a major method of assessing autonomic activity because it is noninvasive, inexpensive, and offers real-time information about the influence of the two subdivisions of the ANS (Lombardi, 2002; Rajendra Acharya, Paul Joseph, Kannathal, Lim, & Suri, 2006; Stauss, 2003; Thayer & Brosschot, 2005). Research has shown that stress and fear increases SNS activity, whereas relaxation, meditation, and neutral water immersion decrease SNS activity and increase PNS (Ditto, Eclache, & Goldman, 2006; Mano, Iwase, Yamazaki, & Saito, 1985; Perini & Veicsteinas, 2003; Ziegelstein, 2007). Increased SNS activation is associated with adverse cardiac events, includ-

ing arrhythmias, whereas increased vagal activation (PNS) is associated with a decrease in adverse cardiac events (Lombardi, 2002; Thayer & Brosschot, 2005; Thayer & Lane, 2007). As a result of these findings, HRV has become a major tool in the assessment of ANS activity and is commonly used in coronary care units for this purpose.

HRV analysis is based upon the understanding that a normal heart beats regularly, but with instantaneous variation. This variation is dependent upon respiratory frequency and ANS activity, including the interplay between the SNS and PNS subdivisions. By studying the variation using mathematical analysis, the variation may be broken into frequency spectra. By measuring the power of these various spectra, the influence of the two subdivisions may be assessed. Typically, it is believed that low and very low frequency spectral power in the rage from 0.15 to 0.4 Hz represents SNS, and that high frequency spectral power in the 0.15–0.4 Hz represents PNS influence. Further analysis of these variables can be used to examine the relationship between these two subdivisions, referred to as sympathovagal balance (Lombardi, 2002; Rajendra Acharya et al., 2006; Stauss, 2003; Thayer & Brosschot, 2005).

Comparable to meditation, aquatic immersion in warm water temperatures has been shown to exert an effect upon the ANS, decreasing sympathetic power while increasing vagal influence (Miwa, Sugiyama, Mano, Iwase, & Matsukawa, 1997; Nishimura & Onodera, 2000, 2001; Perini & Veicsteinas, 2003). A limited amount of research has been done to assess the effects of immersion temperature upon autonomic bioregulation. Most of the current literature has subjects in a supine floating position, rather than in the common seated position used while bathing or hot-tubbing (Nishimura & Onodera, 2000, 2001). This study examines water immersion impacts on the sitting position by contrasting ANS regulation measures in warm water to those in cool and neutral temperatures.

Immersion in warm water is generally found to be pleasurable, creating an almost universal feeling of relaxation. The ANS is the most rapidly responsive bioregulatory control function within the body. Using HRV, these changes can be clearly measured. It is known that positive emotional states are associated with increased sympathovagal balance, while negative emotions and stress will decrease HRV and sympathovagal balance (Brosschot, Van Dijk, & Thayer, 2007; Lane et al., 2008; Thayer & Lane, 2008; Thayer & Sternberg, 2006). The purpose of this study was to address whether the ANS would show changes that mirrored these positive emotion responses in HRV. In addition, we examined physiologic changes that are ANS-mediated, including blood pressure, heart rate, and core temperature.

# Methods

This study protocol was reviewed and approved by the Institution's Investigational Review Board. Sixteen healthy, college-aged participants volunteered for this study, consisting of eight males and females. Resting measurements of heart rate and blood pressure were taken using a standard automated plethysmometer (OMRON HEM-755, Omron Healthcare, Inc, Bannnockburn IL). Participants

ingested a radiofrequency core temperature transmitter (CorTemp, HQInc, Palmetto, FL) that continuously monitored core temperature during the study. To collect HRV data, subjects were connected to a BioPac biologic monitoring system (BioPac Systems Inc, Goleta CA) that continuously measured heart rate and electrocardiogram (ECG) with electrodes placed on right supraclavicular, right iliac, and the left apex.

Participants rested poolside for six minutes before initial measurements were taken. Data collection began with 6 minutes of baseline data and then subjects were immersed in the cool (31.1 °C) tub for 24 minutes (minutes six through 29). Vital signs (i.e., heart rate, core temperature, and blood pressure) were measured four times while in the cool tub: at the tail-end of the 11th, 17th, 23rd, and 29th minutes. Afterward, subjects exited the cool water and rested poolside for 12 minutes (minutes 30–41). Vitals were recollected half way through this first recovery period. Participants then immersed in neutral (36 °C) water for 24 minutes (minutes 42–65), followed by poolside recovery for 12 minutes (minutes 66–77). As before, vitals were retaken at the half-way point of the recovery period. Finally, participants immersed in the warm (39 °C) tub for 24 minutes (minutes 78–101) before sitting at poolside for a final 12 minutes (minutes 102–114). Vitals were taken at both the half-way point and end of the third recovery period.

BioPac data were cleaned before employing a fast-Fourier transform of the ECG into HRV. HRV data contained power spectrum data in very low frequency (VLF = 0.04HZ), low frequency (LF = 0.04–0.15HZ), and high frequency (HF = 0.15–0.4HZ), as well as sympathetic power, vagal power, and autonomic balance. Like vital measures, HRV data were assessed for the baseline and recovery periods and at the same time segments of the immersion periods. Table 1 provides descriptive statistics. Values listed for immersion and recovery periods represent the average of the measures taken.

As the underlying goal of the paper is to further explore potentially unique physiological effects of warm water immersion, paired-samples T tests were conducted. Paired-samples T tests are an appropriate statistical method because data were derived from the same subjects experiencing different conditions at different time points. Moreover, T tests allow for the inference of significance when sample size (and consequently statistical power) is low—a valuable feature considering the limited sample size of this study. Changes over time are graphed as a means to visually represent the relationships between water temperature and physiological response to immersion.

# Results

Table 2 presents results of paired-samples T tests analyses that compare vital and ANS measures taken while in warm water to those taken in other immersion states. Results strongly suggest that warm water has a significant effect on human bioregulatory processes. Physiological responses to warm water not only tend to significantly differ from baseline and recovery values, but the effects of warm water immersion also tend to differ from cool and neutral water immersion in meaningful ways. Key findings of the analyses are reviewed below.

## Heart Rate

Heart rate increased significantly in warm water when compared with cool and neutral immersion temperatures. Additional analyses (not shown here) reveal that immersion in neutral water did not significantly change heart rate from the first-recovery period. Immersion in cool water had the reverse effect of warm water because it lowered heart rate by an average of 4.735 beats per minute, but the magnitude of cool water's impact on heart rate is not nearly as great as warm water. Immersion in warm water increased participant's heart rate by an average of 21.573 beats per minute. As evidenced in Figure 1a, it appears that heart rate recovers significantly from its elevated warm water state once removed for even a few minutes, returning to a level close to those measured in baseline and recovery periods.

## Core Temperature

Core temperature also appears to increase significantly in warm water compared with cool or neutral temperatures. Analyses not shown here indicate that immersion in cool or neutral water did not significantly alter participants' body temperature, though limited sample size may partially account for the nonfinding. In any case, immersion in warm water increased participants' core temperature by 0.45 °C. Figure 1b provides a visual presentation of the change. Lastly, unlike for heart rate, removal from warm water did not significantly reduce core temperature. Thus, it appears the body requires a longer period of time to return to baseline core temperature than it does to restore baseline heart rate once removed from warm water.

## Systolic Blood Pressure

Warm water immersion significantly lowered participants' systolic blood pressure. On average participants' systolic blood pressure decreased by 11.596 mmHg, while rising slightly toward the end of the warm immersion period. During immersion periods water temperature alone does not seem to be the major factor in this effect, due to systolic blood pressure dropping similarly in cold, neutral, and warm water. During recovery immersion temperature does appear to affect systolic pressure either. During the cool and neutral immersion recovery periods, systolic pressures rose significantly, while following warm water immersion the recovery rise was far smaller. Figure 2 presents a summary of the pattern.

## Diastolic Blood Pressure

Participants' diastolic blood pressure responded similarly to systolic pressure. On average participants' diastolic blood pressure decreased by 25.826 mmHg in warm water. Unlike subjects' systolic pressure response however, warm water lowered diastolic blood pressure significantly compared with cool and neutral water. As with systolic pressure, diastolic blood pressures increase was significantly less in the third recovery period. This again implies that diastolic blood pressure responds differently based on whether one exits warm water as opposed to cool or neutral temperatures. Figure 2 provides a visual summary of systolic

## Table 1   Descriptive Statistics

| Variable | Minimum | Maximum | Mean | sd |
|---|---|---|---|---|
| Baseline vitals heart rate | 48 | 83 | 67.412 | 10.000 |
| Core temp [$n = 8$] | 36.44 | 37.34 | 37.05 | 0.296 |
| Systolic BP | 98 | 130 | 112.118 | 8.690 |
| Diastolic BP | 56 | 86 | 72.235 | 8.164 |
| *ANS* VLF | 6.608 | 16.620 | 9.910 | 3.060 |
| HF | 0.631 | 2.598 | 1.079 | 0.584 |
| SVB | 2.011 | 4.280 | 3.602 | 0.611 |
| Cool vitals heart rate | 48 | 76.5 | 62.694 | 9.068 |
| Core temp [$n = 9$] | 36.80 | 37.648 | 37.212 | 0.281 |
| Systolic BP | 91 | 118 | 102.529 | 7.293 |
| Diastolic BP | 51.5 | 70.5 | 61.382 | 5.053 |
| *ANS* VLF | 6.608 | 16.620 | 9.910 | 3.060 |
| HF | 0.636 | 4.992 | 1.991 | 1.253 |
| SVB | 1.131 | 3.875 | 2.636 | 0.819 |
| 1st recovery vitals heart rate | 45 | 76 | 60 | 9.738 |
| Core temp [$n = 9$] | 36.79 | 37.63 | 37.269 | 0.292 |
| Systolic BP | 94 | 134 | 113.118 | 10.487 |
| Diastolic BP | 61 | 84 | 75.235 | 6.440 |
| *ANS* VLF | 4.212 | 22.228 | 12.233 | 4.576 |
| HF | 0.684 | 2.421 | 1.344 | 0.568 |
| SVB | 2.684 | 5.765 | 3.791 | 0.781 |
| Neutral vitals heart rate | 46.3 | 75.3 | 60.406 | 7.933 |
| Core temp [$n = 9$] | 36.58 | 37.448 | 37.111 | 0.261 |
| Systolic BP | 87.8 | 112.80 | 98.176 | 6.883 |
| Diastolic BP | 48 | 67.5 | 56.632 | 5.545 |
| *ANS* VLF | 19.695 | 48.321 | 30.560 | 8.190 |
| HF | 0.596 | 3.542 | 1.563 | 0.888 |
| SVB | 1.608 | 3.903 | 2.864 | 0.700 |
| 2nd recovery vitals heart rate | 50 | 69 | 59.882 | 5.529 |
| Core temp [$n = 8$] | 36.79 | 37.41 | 37.167 | 0.211 |
| Systolic BP | 100 | 132 | 113.647 | 8.485 |
| Diastolic BP | 67 | 94 | 79 | 6.748 |
| *ANS* VLF | 8.282 | 20.159 | 13.335 | 3.955 |
| HF | 0.794 | 3.077 | 1.533 | 0.678 |
| SVB | 1.947 | 4.277 | 3.553 | 0.644 |
| Warm vitals heart rate | 68 | 101 | 81.588 | 8.711 |
| Core temp [$n = 10$] | 37.335 | 38.05 | 37.643 | 0.205 |
| Systolic BP | 83.5 | 120 | 102.610 | 9.014 |
| Diastolic BP | 40.75 | 65.5 | 53.176 | 6.109 |
| *ANS* VLF | 9.637 | 19.580 | 15.394 | 2.982 |

*(continued)*

**Table 1** *(continued)*

| Variable | Minimum | Maximum | Mean | sd |
|---|---|---|---|---|
| HF | 0.271 | 1.323 | 0.587 | 0.291 |
| SVB | 1.857 | 4.782 | 3.893 | 0.710 |
| 3rd recovery vitals heart rate | 51 | 92 | 67.941 | 9.384 |
| Core temp [*n* = 9] | 37.475 | 38.075 | 37.728 | 0.189 |
| Systolic BP | 92.5 | 131 | 108.559 | 10.484 |
| Diastolic BP | 56 | 80 | 66.941 | 6.169 |
| *ANS* VLF | 7.032 | 18.033 | 10.548 | 2.894 |
| HF | 0.619 | 2.114 | 1.038 | 0.387 |
| SVB | 2.364 | 4.956 | 3.920 | 0.685 |

**Table 2    Paired-Samples *T* Test Analyses of Warm Water Effects by Water Immersion Status⁺**

| Variable | Baseline | Cool | 1st Recovery | Neutral | 2nd Recovery | 3rd Recovery |
|---|---|---|---|---|---|---|
| Heart rate | +14.043*** | +18.779*** | +21.455*** | +21.073*** | +21.573*** | +13.69*** |
| Core temp | +0.700** | +0.423** | +0.367** | +0.524*** | +0.450*** | −0.093 |
| Systolic BP | −4.890 | −0.434 | −11.066*** | +3.904 | −11.596*** | −6.508*** |
| Diastolic BP | −19.059*** | −8.206*** | −22.059*** | −3.456* | −25.826*** | −13.588*** |
| VLF | +5.484*** | −16.956*** | +3.162** | −15.166*** | +2.060* | +4.847*** |
| HF | −0.492*** | −1.405*** | −0.757*** | −0.976*** | −0.947*** | −0.451** |
| SVB | +0.291* | +1.246*** | +0.101 | +1.029*** | +0.339* | −0.028 |

⁺Horizontal axis is reference category.

*p <.05; **p <.01; ***p <.001, 2-tailed.

*n* = 8–18

and diastolic blood pressure graphed together. This allows comparisons between pulse bandwidth, and shows it increased successively during neutral and warm water immersion.

## Very Low Frequency Power Spectral Data (VLF)

Warm water immersion significantly raises VLF HRV, though not nearly to the degree of cool and neutral water. Warm water immersion increased VLF power by an average of 2.060HZ. While this is a significant change, warm water's impact is far smaller than cool and neutral water, which raised participants' VLF values by 22.441HZ and 18.328 HZ, respectively. Figure 3a presents the difference.
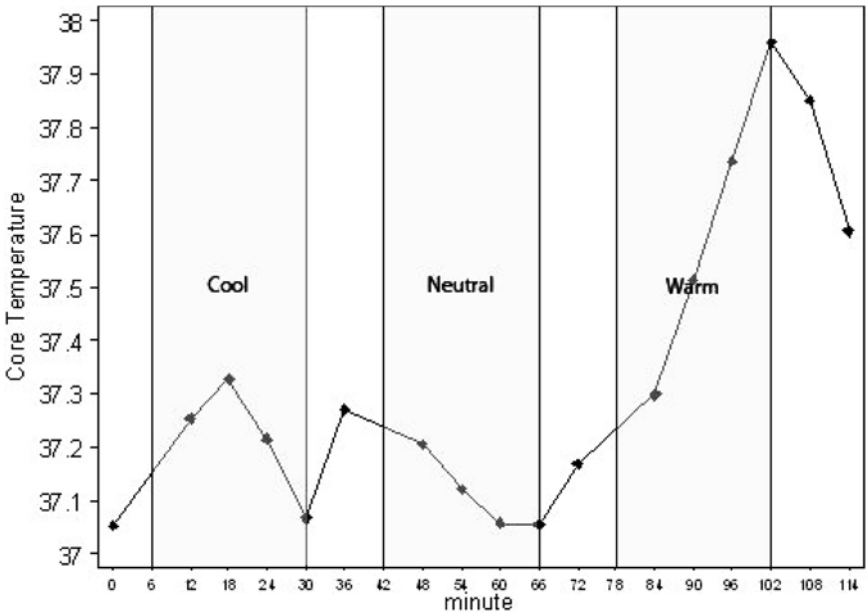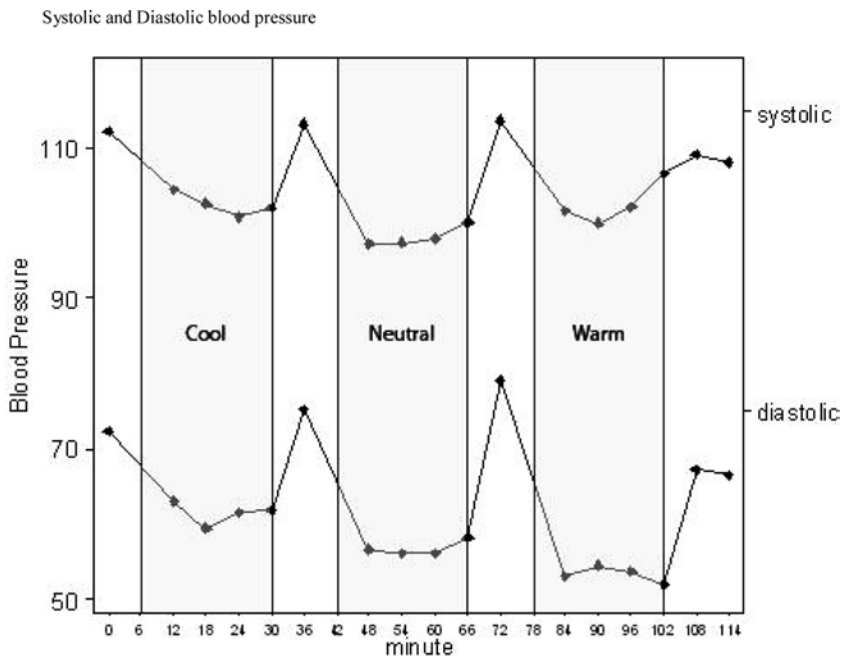
**(1a.)**
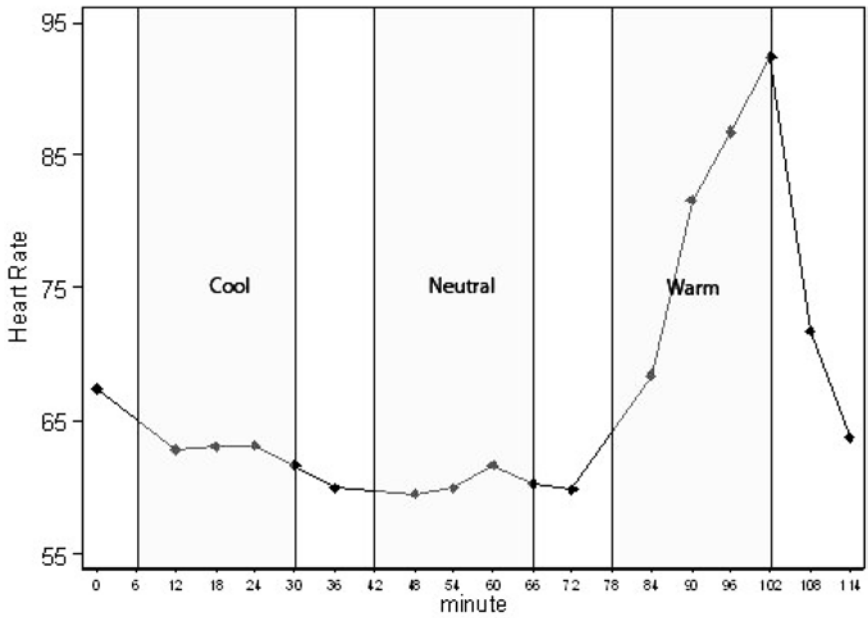


**(1b.)**



**Figure 1** — Vitals (a. heart rate; b. core temperature).

Systolic and Diastolic blood pressure



**Figure 2** — Systolic and Diastolic blood pressure.

## High Frequency Power Spectral Data (HF)

HF HRV power is heavily influenced by water immersion status. Unlike cool and neutral water, both of which appear to significantly increase the response in HF power spectrum, exposure to warm water is associated with a significant decline in HF power. Figure 3b shows warm water immersion decreased participants' HF power by 0.947HZ.

## Sympathovagal Balance (SVB)

Results for SVB reverse those of HF power spectral analysis. Cool and neutral water significantly decreased SVB, but warm water immersion significantly increased SVB. Figure 3c shows warm water immersion increased SVB by 0.339HZ.

# Discussion

The purpose of this study was to address whether the ANS would show changes in HRV. In addition, we examined physiologic changes that are ANS-mediated including blood pressure, heart rate, and core temperature. HRV has been used extensively to monitor ANS function, because it is safe, noninvasive, and relatively inexpensive (Sinski, Lewandowski, Abramczyk, Narkiewicz, & Gaciong,

(3a.)



(3b.)



(3c.)
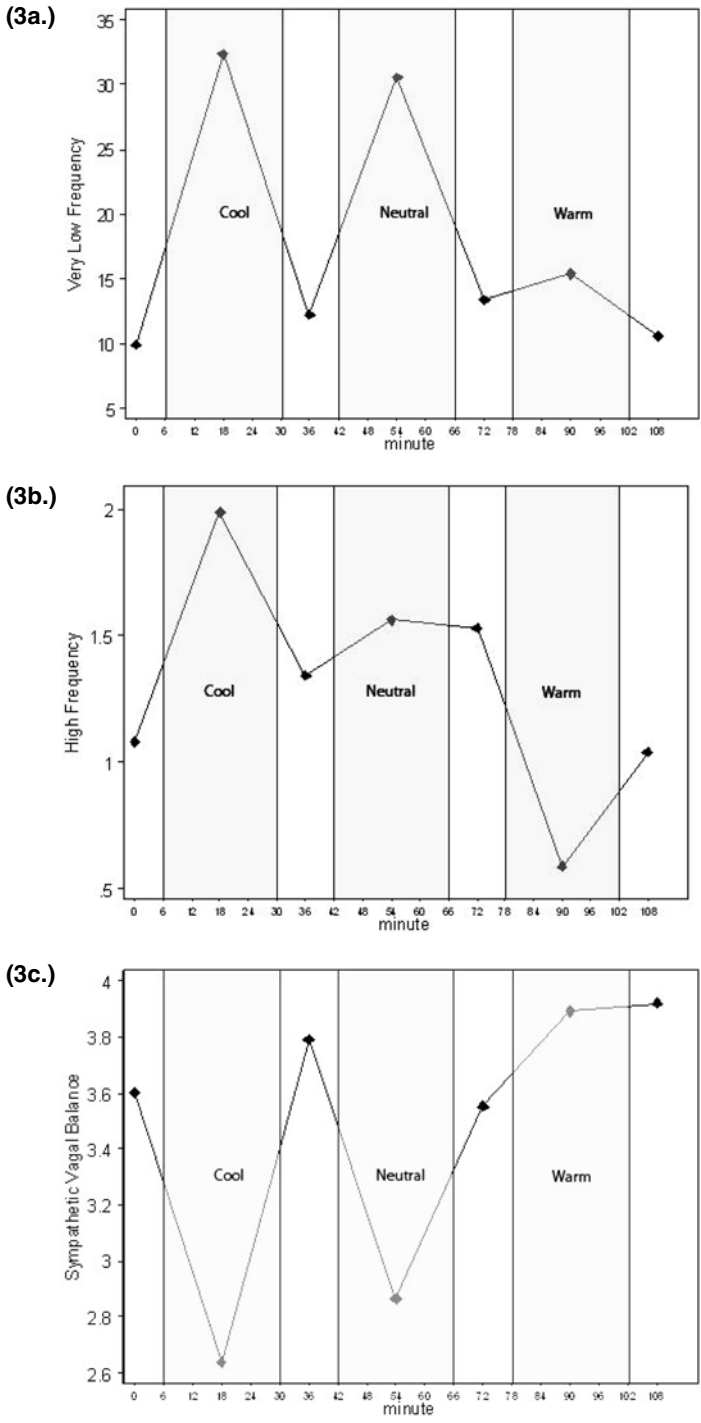


**Figure 3** — ANS (a. VLF power spectral data; b. HF power spectral data; c. Sympathova-

2006). The impact of autonomic dysfunction has been associated with a great number of diseases and health issues (Thayer & Lane, 2007; Thayer & Siegle, 2002; Thayer & Sternberg, 2006). Methods of ANS alteration to increase HRV and to decrease the influence of the SNS in particular have been shown to have positive effects on critical elements of bioregulation. This includes a number of important mood and cognitive processes (Eskandari & Sternberg, 2002; Lane et al., 2008; Thayer & Brosschot, 2005; Thayer & Siegle, 2002; Thayer, Newman, & McClain, 1994; Tiller, McCraty, & Atkinson, 1996; Ziegelstein, 2007). Safe, easily available nonpharmacologic methods of achieving this autonomic adjustment could have potential utility and applicability over a range of health care issues. Warm water immersion has been shown in some previous studies to have some significant effects upon the ANS, reducing SNS activity, and increasing PNS influence upon the ANS (Mourot et al., 2008; Mourot et al., 2007; Nagasawa et al., 2001; Nishimura & Onodera, 2000, 2001). The mood and cognitive effects of these autonomic adjustments reduce anxiety, increase working memory, increase executive function (a complex group of cognitive skills), and attentional regulation (Thayer & Brosschot, 2005). It is perhaps of note in this context that Winston Churchill, a prolific writer but also someone who suffered from depression, was known for doing a great deal of his writing in the bathtub.

In healthy college-aged adults immersion in water produced a significant number of important physiologic changes that may provide health benefits. These include changes in blood pressure, HRV, and core temperature. The authors believe that the cascade of these changes is intimately involved with ANS bioregulation. Further, these changes seem to be influenced by immersion temperatures, as a statistically significant relationship between ANS activity manifested by HRV and water temperatures was found. Cool water produced a rise from baseline in SNS activity, with a drop in sympathovagal balance. This likely represents a physiologic stress response. Somewhat surprisingly, when compared with cool and neutral immersion, warm water immersion still produced a rise in sympathetic power (while smaller) with a small drop in sympathovagal balance from baseline. This elevation of sympathovagal balance lasted throughout the postimmersion period of study. A rise in sympathovagal balance is associated with stress reduction, positive emotions, relaxation, and meditation (Thayer & Lane, 2000; Thayer & Siegle, 2002; Thayer et al., 1994). Such a physiologic change causes a decrease in cardiac irritability, a reduction in blood pressure, and a decrease in anxiety (Thayer & Brosschot, 2005; Thayer & Lane, 2000; Thayer & Siegle, 2002; Thayer & Sternberg, 2006; Ziegelstein, 2007).

We observed a decrease in both mean blood pressure and diastolic pressures during the immersion period, most pronounced during the warm water cycle and subsequent to it. This has been seen in a number of prior studies (Allison & Reger, 1998; Arborelius et al., 1972; Coruzzi, Musiari, Mossini, Ceriati, & Novarini, 1993; Gabrielsen, Warberg et al., 2000; Nishimura & Onodera, 2000; Park, Choi, & Park, 1999; Robiner, 1990).

This study assessed 16 college-aged individuals, a relatively small number of subjects. The subjects were all healthy and none took regular medications. A larger sample might have given somewhat different results as would have an older group of subjects. For this study, we chose water temperatures that were within a narrow range. During initial work we attempted a lower temperature but found

that subjects first chilled and then shivered by the conclusion of the 24-min immersion period. This greatly affects the ECG pickup, masking the signal with muscle artifact. Shivering often persisted into the second tank, thus creating signal artifact that made the HRV measurement unusable. We initially attempted 40 °C for the warm temperature, but found that none of the subjects were able to remain in the water for the desired 24 min immersion cycle, as core temperatures were elevated as has been found in prior research (Allison & Reger, 1998). The 24-min immersion cycle was chosen because HRV measurement requires at least a 5 min steady state cycle, and we were interested in assessment over a period of initial accommodation, followed by physiologic responses during full equilibration. The four 6-min periods left us a margin of error for data cleaning.

## Conclusions

We believe this to be the first study examining the effects of water immersion upon the ANS using various temperatures. There were striking differences between the three immersion states, with a pronounced increase in sympathovagal balance during the warm water immersion period, with a reduction in both diastolic and mean blood pressure. Core temperature also increased significantly in warm water compared with cool or neutral temperatures. The results showed substantial individual variation in magnitude, but not in direction. There may be clinical utility for the effects seen during warm water immersion.

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