Cold-Water Face Immersion Per Se Elicits Cardiac Parasympathetic Activity

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Background  Cold-water face immersion (FI) is known to produce physiological changes, including bradycardia, by stimulating the parasympathetic system. However, other factors such as sympathetic activity, intrapleural pressures, and changes in chemical mediators may also contribute to these changes.

Methods and Results  Eight healthy volunteers underwent a series of experiments designed to observe the effects of FI on heart rate and its variability, as detected using wavelet transformation. Each subject was instructed to bend over and put the entire face into an empty basin with and without breathing (protocols 1 and 2, respectively), and then perform FI in warm-water (protocols 3 and 4, respectively) and cold-water (protocols 5 and 6, respectively) while breathing and breath holding. Change in the R-R interval with FI was only significantly greater for protocol 6 than for the control procedure (protocol 1). Also, changes in the natural logarithm of high-frequency power with FI were significantly greater for protocols 5 and 6 than the protocol 1.

Conclusions  Bradycardia associated with cold-water FI is mainly attributed to cardiac vagal activity, which is independent of both the change in body position caused by bending over a basin and breath holding. (Circ J 2006; 70: 773–776)

Key Words: Diving reflex; Face immersion; Heart rate variability; Wavelet transform

Diving into water is known to cause various physiological changes in mammals.1–3 Autonomic effects evoked by submersion in water include bradycardia, selective vasoconstriction of the skin and viscera, diminished cardiac output, and either well-maintained or elevated blood pressure.4 In humans, cold-water face immersion (FI) alone is known to produce similar physiological changes and is widely used as a maneuver for stimulating the parasympathetic system.6–8 Clinically, FI has become an important diagnostic tool for detecting and evaluating arrhythmias, especially in patients with long QT syndrome.7,9–11 These physiological changes have been attributed to a reflex involving the trigeminal–brainstem–vagal pathways. Superficial cold receptors that are innervated by the ophthalmic branch of the trigeminal nerve enhance the cardiovagal activity involved in the response.12–14 On the other hand, it is well known that intrapleural pressures15 and changes in chemical mediators, such as arterial carbon dioxide partial pressure and arterial pH, caused by breath holding16,17,18 may also affect the slowing of the heart rate during FI with breath holding.

Power spectral analysis of heart rate variability (HRV) is widely used as a noninvasive method for quantifying cardiac autonomic function in humans.19–20 However, only a few studies have shown the effects of cold-water FI on the measurements of HRV and have used time domain variables and/or traditional frequency domain variables using the Fourier transform, both of which are based on the assumption of steady-state conditions and therefore cannot be applied to the analysis of dynamic changes in autonomic nervous activity such as during FI. To overcome this limitation, wavelet transformation (WT) has recently been introduced for the analysis of nonstationary signals because it enables the analysis of instantaneous changes in autonomic nervous balance caused by physiological interventions, such as FI into cold-water.

The present study is the first to use the WT to test the hypotheses that the bradycardia during cold-water FI is mainly related to stimulation of parasympathetic activity per se and that the contribution of other factors, such as intra-pleural pressure and chemoreceptors, is relatively small. Proving this hypothesis would justify the use of cold-water FI as a maneuver to stimulate cardiac parasympathetic activity.

Methods

Subjects  A total of 8 nonsmoking Japanese volunteers (4 men, 4 women) were recruited. Interviews and examinations (including auscultation) performed by a physician revealed that all the subjects were in good health. The subjects gave their informed consent to participate in the study, which was approved by our institutional review board. The mean age, body weight, and height of the subjects were 33 years (SD 4.9 years), 60 kg (SD 7 kg), and 167 cm (SD 8 cm), respectively. All subjects were moderately active, but none participated in daily exercise at a competitive level.
Experimental Protocol

The experimental protocol comprised 4 procedures.

Protocol 1 (Control: Snorkel Breathing Only) After resting while seated in a chair for at least 10 min, the subject was instructed to bend over and put the entire face, including the forehead and chin, into an empty basin that was placed on a desk in front of him/her, with the chair and basin aligned so that the subject would feel as comfortable as possible during the procedure. The subject breathed through a snorkel (dead space=80 ml) throughout the procedure. The duration of the FI was 30 s.

Protocol 2 (Breath Holding Only) The procedure was the same as for protocol 1 except that the subject was instructed to hold his/her breath at a lung volume of normal inspiration during the procedure.

Protocol 3 (Warm-Water FI With Snorkel Breathing) The procedure was the same as for protocol 1 except that the subject immersed his/her entire face in warm-water (37°C).

Protocol 4 (Warm-Water FI With Breath Holding) The procedure was the same as for protocol 2 except that the subject immersed his/her entire face in warm-water (37°C).

Protocol 5 (Cold-Water FI With Snorkel Breathing) The procedure was the same as for protocol 1 except that the subject immersed his/her entire face in cold-water (4°C).

Protocol 6 (Cold-Water FI With Breath Holding) The procedure was the same as for protocol 2 except that the subject immersed his/her entire face in cold-water (4°C).

We performed the experiments in serial order from protocol 1 to protocol 6, with the interval between each experiment no less than 10 min in order to allow the R-R intervals to return to the resting values.

Recording Procedure

A Holter electrocardiogram (FM-100, Fukuda Denshi, Japan) was recorded continuously throughout the procedures. The data were digitized, and recorded on flash-
memory cards. The beginning and end of each experiment were indicated by pressing an event button.

Blood pressures of the right arm were measured before and during each experiment with an automatic sphygmomanometer (Radarcirc: Dainippon-Sumitomo Pharmaceutical, Osaka, Japan).

Analysis of R-R Intervals

The Holter electrocardiogram system allowed the extraction of the list of R-R intervals with a precision of 1/128 s. The length of each R-R interval was validated manually during this step. The power spectrum was analyzed off-line by continuous WT on a Sony PC (VAIO PCG-R50P/BD) using Fluclet WT (DaiNippon Pharmacy, Osaka, Japan).

Statistical Analysis

The R-R intervals and power spectra before, during, and after each experiment were averaged every 10 s, and are presented here as mean (SD) values. As the distributions of the high-frequency (HF) and low-frequency (LF) power were non-normal, their values were transformed into natural logarithm values (lnHF and lnLF, respectively) and used for statistical analyses. The baseline values were obtained by averaging the measurements over a 90 s period immediately before the beginning of FI. The effects of each experimental protocol on the R-R intervals, measurements of HRV, and blood pressures were expressed by the differences between the measurements obtained by the average of the last 10 s of FI (FI 30) and the baseline values. Comparisons of these effects among experimental protocol were calculated with analysis of variance. If significant difference was detected by the F-test, multiple comparisons of these effects among experimental protocol were made with Dunnett’s method. The correlation between the changes in lnLF power and systolic and diastolic blood pressures were analyzed by simple regression analysis. The statistical analyses were performed with SPSS (version 10, Scientific Packages for Social Sciences, Chicago, IL, USA). A probability value of <0.05 was considered to indicate statistical significance.

Results

Effects of FI on the R-R intervals, lnHF power, lnLF power, and the LF/HF ratio, and blood pressure during protocols 1 through 6 are presented in Fig 1.

Effects on the R-R Interval (Fig 1a)

Significantly longer prolongation of the R-R intervals was observed only with protocol 6 compared with the control procedure (protocol 1). Protocols 2–5 did not elicit significantly longer prolongation of the R-R intervals than protocol 1.

Effects on the HF Power (Fig 1b)

Significantly greater augmentation of the lnHF power was caused only by protocols 5 and 6 compared with the control procedure (protocol 1). Protocols 2–4 did not elicit significantly greater augmentations of the lnHF power than protocol 1.

Effects on the LF Power (Fig 1c)

We did not find statistically significant differences in the changes in the lnLF power among protocols 1–6.

Discussion

In the present study, only cold-water FI with breath holding increased the R-R interval. Only FI into cold-water with and without breath holding resulted in a marked increased in the HF power, whereas FI in warm-water and simply putting the face into an empty basin caused no changes. Moreover, FI into cold-water did not cause elevated systolic and diastolic blood pressures and did not augment the LF power. Furthermore, the effects of body position and breath holding on the R-R interval and its variability were almost negligible. These results indicate that bradycardia elicited by cold-water FI is mainly attributable to stimulation of parasympathetic activity, with only a small contribution from other factors such as intrapleural pressure and chemoreceptors.

The present study shows that the changes in the lnLF power caused by FI did not correlate with the changes in systolic and diastolic blood pressures, which supports the notion that the LF power of the R-R interval is not a pure index of cardiac sympathetic activities.

One unusual result from the present study was the apparent divergence in the dynamics of the R-R interval and the HF power in HRV from the usually rapid activation of the parasympathetic efferent component. The heart rate responses mediated by vagal activity, both when excited and inhibited, are usually very fast whereas in the present study there was a delay of some 10–20 s between the initiation of FI and the onset of bradycardia and the surge in the HF power. This discrepancy may be explained as follows. Diving-induced bradycardia in humans is elicited by chilling of the cold receptors in the forehead and the eyes and conducted via the trigeminal–brainstem–vagal pathway. There will be some delay before the cold receptors on the face are adequately chilled and activated, which also explains why these effects are maintained even after cessation of FI. Therefore, we believe that the delay is not inconsistent with the activation of vagal activity elicited by cold-water FI.

The results of the present study are essentially compatible with the report of Hayashi et al21 that FI while breathing increased the standard deviations and coefficients of variance of the R-R interval, indicating that FI alone increases cardiac vagal activity. However, a major limitation of their study is the use of time-domain indices of HRV, which cannot be used to evaluate sympathetic and parasympathetic activity separately.27 The present study is
the first to use wavelet analysis, which is a frequency-domain method that enables the analysis of nonstationary signals and has revealed increases in the HF power of the R-R interval, an established index of cardiac parasympathetic activity during cold-water FI.

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References