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Blood Pressure Regulation During Bathing: Is There a Cardiovascular Risk?

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1. Introduction

Water immersion-induced augmentations of external hydrostatic pressure increase central venous and atrial volume and pressure (Arborelius et al., 1972; Gabrielsen et al., 1993; Onodera et al., 2001). The increased left ventricular preload results in the elevation of stroke volume and cardiac output (Arborelius et al., 1972; Ueno et al., 2005). In Poiseuille's law, an elevated stroke volume should increase blood pressure. Therefore, it is reasonable to predict that blood pressure increases during bathing. However, previous studies have reported contradictory results. Ohnaka et al. (1995) have demonstrated that 10-min bathing (40 ^{o}C) decreased systolic blood pressure (SBP) in young and middle-aged subjects. Additionally, Nagasawa et al. (2001) have reported that SBP decreased during hot-water immersion (40 ^{o}C, 10 min) in young subjects, but increased at the onset of immersion and recovered during the latter phase of immersion in older subjects. Furthermore, Allison & Reger (1998) have reported that SBP in young and middle-aged men showed initial decrease, followed by a gradual increase back toward the baseline during 21-min bathing (40.0 ^{o}C). Taken together, the vasopressor effects of bathing are less likely to be marked. Blood pressure during bathing may be maintained by some facilitated regulatory mechanisms.

Blood pressure is regulated mainly by autonomic nervous, endocrine, and renal body-fluid modulation systems. Particularly, the autonomic nervous system rapidly responds to changes in blood pressure. The cardiovagal baroreflex is one of the autonomic functions that regulate blood pressure. Arterial baroreceptors, stretch receptors in the carotid arterial and aortic bodies, detect any blood pressure-associated strain of arterial walls and send afferent signal to increase or decrease heart rate (HR). In this way, the cardiovagal baroreflex regulates blood pressure. Since the baroreceptor firing rate is proportional to changes in arterial circumference at a physiological blood pressure (Aars, 1969), cardiovagal baroreflex sensitivity (BRS) is considered to be affected by carotid arterial or aortic stiffness (Kingwell et al., 1995; Monahan et al., 2001; Rowe, 1987). Additionally, *in vitro* diameter-pressure curves at different temperatures, illustrated using harvested human common carotid arteries, have suggested that hyperthermia may have softening effects on the carotid artery (Guinea et al., 2005). Therefore, bathing-induced hyperthermia may reduce carotid arterial stiffness and consequently increase cardiovagal BRS. Evidence demonstrating that short-term heat stress (5 min, 46.0-48.0 ^{o}C) increases skeletal muscle sympathetic BRS (Keller et al., 2006), may support this hypothesis. However, it has remained unclear whether cardiovagal BRS increases during bathing.

We hypothesized that cardiovagal BRS increases during bathing to attenuate bathing-induced blood pressure changes. To test this hypothesis, cardiovagal BRS and hemodynamics in women were measured before, during, and after bathing. Cardiovagal BRS was evaluated using the sequence method (Bertinieri et al., 1988; Hayashi et al., 2006). As a control experiment, a showering session was performed according to the protocol for the bathing session. Body temperature before and after showering/bathing and carotid arterial pulse wave velocity (PWV; an index of arterial stiffness) at rest were also investigated.

2. Methods

2.1 Subjects

Twelve women [age, 35.5±6.6 (19-69) years; height, 157±2 (147-169) cm; weight, 52.4±2.1 (39.3-62.7) kg; and BMI, 21.2±0.7 (16.2-25.4) kg/m^2] volunteered to participate in this study. All subjects were free of signs and symptoms of cardiac diseases, diabetes, and hypertension. None of the participants were currently taking any medications. Premenopausal women were tested at least 3 days before or 3 days after menses. All subjects provided written informed consent before inclusion in the study. This study conformed to the principles outlined in the Helsinki Declaration.

2.2 Experimental protocol

Two testing sessions, showering and bathing sessions, were performed in a randomized order on the same day, at least 2 h apart, in a quiet and temperature-controlled room. First, carotid arterial PWV was measured at supine position after a rest period (formPWV/ABI; Omron Colin, Tokyo, Japan). Second, radial arterial pulse waveforms were continuously recorded for 5 min by applanation tonometry in a seated position (BP-508SD, Omron Colin). At the onset of waveforms recording, brachial arterial pressure was measured by oscillometry (BP-508SD, Omron Colin). Skin temperature was measure at the end of the rest period using an electronic axillary thermometer (CT513, Citizen Systems, Tokyo, Japan). Also, tympanic temperature was measured via an infrared ear thermometer (CT820, Citizen Systems). Third, subjects were showered with hot water (40 ^{o}C, 12.5 L/min) or immersed in hot water (40 ^{o}C) up to the axillae for 10 min in a sitting position. The radial arterial waveforms and brachial blood pressure waveforms were investigated as previously mentioned. Finally, subjects underwent a 10-min recovery period after the cessation of showering/bathing in the same position as in the pre-showering/bathing period. Again, radial pressure waveforms, brachial blood pressure, and body temperatures were measured in this period.

2.3 Carotid arterial PWV

Carotid arterial PWV was measured using a device to investigate PWV (formPWV/ABI; Omron Colin), as previously described with minor modifications (Kimoto et al., 2003; Kobayashi et al., 2004). Briefly, subjects assumed a supine position with a cardiac sound microphone placed at the right sternal margin at the second intercostal level, with an applanation tonometer on the common carotid artery. The pulse wave transit time between the aortic valve and carotid artery was determined based on the time delay between the

beginning of the second heart sound and notch of the carotid artery pulse wave. The distance traveled by the pulse waves was estimated according to the height of subjects using the following equation: 0.2437 x height - 18.999 (Kimoto et al., 2003; Kobayashi et al., 2004). Carotid arterial PWV was calculated as the distance divided by the transit time.

2.4 Cardiovagal BRS

Cardiovagal BRS was assessed according to previous studies with minor modifications (Bertinieri et al., 1988; Hayashi et al., 2006). Under all recording conditions, an applanation tonometer (BP-508SD, Omron Colin) was placed at the forth intercostal level. The applanation tonometer sampled the radial arterial pressure waveforms and stored data in a computer. HR was calculated from pulse intervals. We identified baroreflex sequences (three or more beats relating to pulse intervals and progressively and spontaneously changing SBP at the same detection points). Next, we determined the slope of the linear relationship between the pulse intervals and SBP at these points. The minimum changes observed were 1 mmHg for SBP and 1 ms for the pulse interval. Linear regressions relating SBP to the pulse interval were plotted for each sequence; only those sequences with linear r values > 0.85 were accepted. For the latter 5-min of respective period, the results were averaged to provide a single data set.

2.5 Statistical analysis

Data are expressed as means±SE. Statistical analysis was carried out using repeated-measures two-way ANOVA followed by Scheffe's test for multiple comparisons. Linear correlation analysis was used to examine the relationship between carotid arterial PWV and cardiovagal BRS. $P<0.05$ was regarded as significant.

3. Results

Respective temperatures of shower/bath water were 40.6±0.2/40.6±0.1 ^{o}C. Bathroom temperature in showering and bathing sessions was 27.3±0.1 and 27.8±0.1 ^{o}C, and room humidity was 66.7±0.9 and 62.3±1.7 %, respectively.

Cardiovagal BRS before, during, and after showering/bathing is indicated in Fig. 1. Two-way ANOVA revealed an interaction between session (showering or bathing) and time in cardiovagal BRS. In multiple comparisons, cardiovagal BRS did not change during showering but increased during bathing compared to the baseline. Also, cardiovagal BRS was greater during bathing than during showering. The elevated cardiovagal BRS recovered to the baseline after the cessation of bathing.

Hemodynamics during sessions are summarized in Table 1. We did not identify interactions (session x time) in all measures. Again, there were no main effects of session in all indices. Main effects of time were detected in SBP, mean blood pressure (MBP), HR, and double-product (DP, *i.e.*, product of SBP and HR). In details, SBP was higher during and after showering and bathing than the baseline. MBP increased after showering and bathing compared to at rest. HR was higher during showering and bathing in comparison to before showering and bathing. Increased DP was observed during and after showering and bathing. Time was not associated with diastolic blood pressure and pulse pressure.

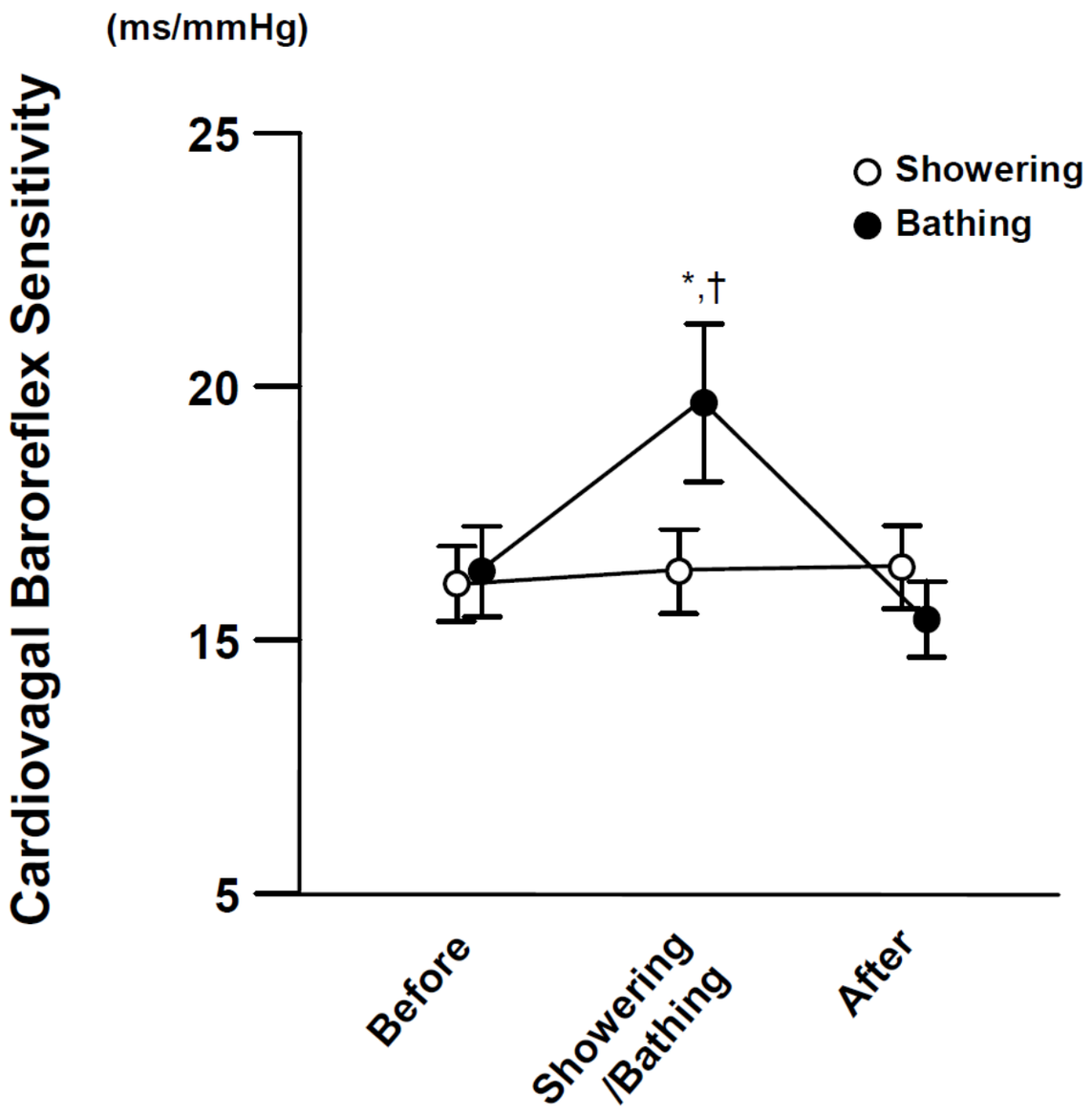


Fig. 1. Cardiovagal baroreflex sensitivity (BRS) before, during, and after showering/bathing. Data are expressed as means±SE. Repeated-measures two-way ANOVA (session x time) revealed an interaction in cardiovagal BRS (F=6.4, P<0.01). On multiple comparisons, showering was not associated with cardiovagal BRS, but bathing increased it compared to the baseline. The increased BRS during bathing recovered to the baseline after the cessation of bathing. *P<0.05 vs. before bathing; †P<0.05 vs. showering.

		Before			Showering /Bathing			After			Interaction
SBP, mmHg	Showering	100	±	2	103	±	2	105	±	2	*F*=0.3
	Bathing	99	±	3	103	±	2	102	±	3	*P*=0.73
MBP, mmHg	Showering	67	±	2	71	±	2	73	±	3	*F*=0.7
	Bathing	70	±	2	71	±	2	72	±	2	*P*=0.49
DBP, mmHg	Showering	55	±	2	55	±	2	56	±	2	*F*=1.3
	Bathing	52	±	3	56	±	4	58	±	3	*P*=0.28
PP, mmHg	Showering	45	±	2	48	±	2	49	±	3	*F*=1.4
	Bathing	46	±	2	47	±	4	44	±	2	*P*=0.27
HR, bpm	Showering	68	±	1	70	±	2	68	±	2	*F*=2.2
	Bathing	68	±	2	72	±	3	71	±	2	*P*=0.12
DP, AU	Showering	68.4	±	1.7	71.5	±	1.6	71.3	±	1.4	*F*=1.6
	Bathing	66.5	±	2.7	73.7	±	3.3	72.0	±	3.0	*P*=0.20

Table 1. Hemodynamics before, during, and after showering/bathing. Values are means±SE. SBP, systolic blood pressure; MBP, mean blood pressure; DBP, diastolic blood pressure; PP, pulse pressure; HR, heart rate; DP, double product; AU, arbitrary unit.

The relationship between carotid arterial PWV and cardiovagal BRS at rest is demonstrated in Fig. 2. Carotid arterial PWV was correlated with cardiovagal BRS.

Table 2 shows body temperatures before and after showering/bathing. There was no interaction between session and time in skin temperature. However, two-way ANOVA identified the main effect of time; skin temperature increased after showering and bathing. No differences in skin temperature between showering and bathing were present. On the other hand, we observed an interaction (session x time) in tympanic temperature. In multiple comparisons, showering did not change tympanic temperature but bathing increased it.

4. Discussion

Spontaneous cardiovagal BRS, hemodynamics, and body temperature in women were measured before, during, and after showering/bathing. Carotid arterial PWV, an index of arterial stiffness, was also measured at rest. We demonstrated for the first time that spontaneous cardiovagal BRS increased during bathing compared to the baseline. There were no differences in the changes from the baseline in blood pressure, HR, and DP between showering and bathing sessions. Tympanic temperature did not change after showering but increased after bathing in comparison to the baseline. Cardiovagal BRS was negatively correlated with carotid arterial PWV. We concluded that cardiovagal BRS increases during bathing. Increased cardiovagal BRS may attenuate the blood pressure changes during

bathing, resulting in comparable hemodynamics responses between showering and bathing. We would like to propose that it may be unreasonable to withhold bathing by reason of cardiovascular risk. Taking into consideration the slight but significant hyperthermic effects, bathing may be more beneficial to humans in comparison to showering, although caution may be required for humans with stiffened carotid arteries.

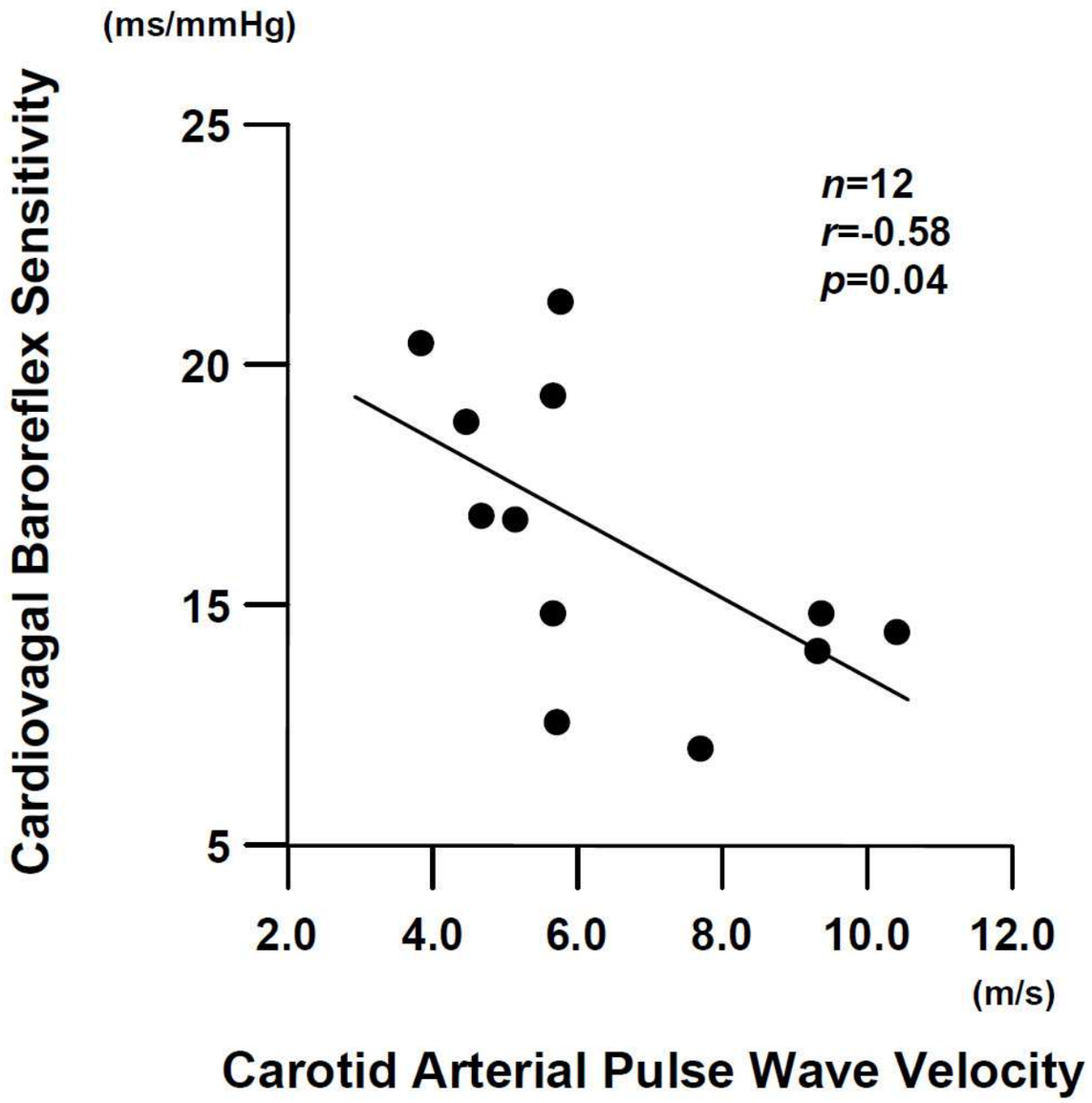


Fig. 2. Relationship between carotid arterial pulse wave velocity (PWV), an index of arterial stiffness, and cardiovagal baroreflex sensitivity (BRS) at rest. Carotid arterial PWV was correlated with cardiovagal BRS.

		Before			After			Interaction
Skin temperature, °C	Showering	36.4	±	0.1	36.6	±	0.2	*F*=0.3
	Bathing	36.3	±	0.2	36.6	±	0.1	*P*=0.57
Tympanic temperature, °C	Showering	37.0	±	0.1	36.9	±	0.1	*F*=7.4
	Bathing	37.0	±	0.1	37.1	±	0.1*	*P*=0.01

Values are means±SE. *P<0.05 vs.

Table 2. Body temperature before and after showering/bathing.

Water immersion increases venous return and ventricular preload (Arborelius et al., 1972; Gabrielsen et al., 1993; Onodera et al., 2001). Consequently, stroke volume and cardiac output increases in the water-immersed subjects (Arborelius et al., 1972; Ueno et al., 2005). Therefore, theoretically, arterial SBP and pulse pressure are speculated to be elevated during bathing. However, the reported effects of bathing on blood pressure are not consistent (Allison & Reger, 1998; Nagasawa et al., 2001; Ohnaka et al., 1995). Also in this study, we did not observe differences in blood pressure changes between showering and bathing periods. It is reasonable to consider that the effects of bathing on blood pressure are not marked. The mechanisms underlying the paradox (*i.e.*, bathing increases blood flow but not arterial blood pressure) have, up to now, remained unclear. This study demonstrated that cardiovagal BRS increases during bathing. This enhanced cardiovagal BRS may participate in the regulation of blood pressure and reduce cardiovascular risks during bathing.

Cardiovagal BRS is regulated via arterial baroreceptors, stretch receptors in arterial tissue. The baroreceptors are located in carotid arterial and aortic bodies. Baroreceptors sense arterial wall strain caused by blood pressure changes and send afferent signals corresponding to the strain level to the cardiovascular center. The stiffness of baroreceptor-containing arteries such as the carotid artery has been hypothesized to be one of the key factors determining cardiovagal BRS (Kingwell et al., 1995; Monahan et al., 2001; Rowe, 1987). Indeed, Monahan et al. (2001) have demonstrated that age-related carotid arterial stiffening, evaluated using echo tracking, was closely associated with decreased cardiovagal BRS, investigated using the Oxford technique. Again, we demonstrated a linear relationship between carotid arterial PWV and cardiovagal BRS. It is well-established that arterial stiffness increases with advancing age (Avolio et al., 1985; Otsuki et al., 2006a; Otsuki et al., 2006b; Vaitkevicius et al., 1993). Central arterial stiffness are also associated with daily physical activities (Iemitsu et al., 2006; Sugawara et al., 2006). Although the present subjects did not experience a marked blood pressure increase during bathing [*e.g.*, maximal SBP increase was 16 mmHg (from 98 at rest to 114 mmHg)], humans with markedly stiff arteries may need to take precautions.

Body temperature was measured before and after showering/bathing at two sites. The skin temperature increased after both showering and bathing. On the other hand, the effects on tympanic temperature were different between showering and bathing. Showering did not change the tympanic temperature, bathing slightly but significantly increased it. The tympanic temperature is closer to the core temperature than skin temperature. It is possible that bathing elevated not only the surface but also internal temperature. At least, the hyperthermic effects are likely to be greater in bathing compared to showering.

We showed that cardiovagal BRS increased on short-term (*i.e.*, 10 min, 40.6 °C) bathing. Also, Keller et al. (2006) have demonstrated that skeletal muscle sympathetic BRS is enhanced by short-term heat stress (5 min, 46.0-48.0 °C). On the other hand, prolonged bathing may be counterproductive. Lee et al. (2003) have reported that hot-water (44.0 °C) immersion of the lower legs for 30 min reduced spontaneous cardiovagal BRS. Prolonged hot-water bathing should be refrained from.

The mechanisms responsible for bathing-induced increases in cardiovagal BRS remain unclear. One explanation for this response may be hyperthermia-related reduction of arterial stiffness. Guinea et al. (2005) have reported that hyperthermia shifts the carotid arterial diameter-pressure curve to the right, suggesting that hyperthermia reduces carotid arterial stiffness. Carotid arterial baroreceptors are stretch receptors. A compliant carotid artery is easily stretched and would be sensitive to changes in blood pressure. Indeed, the present and previous (Monahan et al., 2001) studies have showed a negative relation between cardiovagal BRS and carotid arterial stiffness. We observed that showering increased only the skin temperature, but bathing increased both the skin and tympanic temperature. Collectively, it may be possible that a hyperthermia-induced reduction of carotid arterial stiffness is associated with increased cardiovagal BRS during bathing. Another possible explanation for the changes in cardiovagal BRS is the direct effect of an elevated core temperature on central baroreflex pathways. Previous findings have demonstrated that an increased temperature enhances the neuronal firing rate of thermosensitive neurons (Boulant, 1998). While these findings were limited to the hypothalamic region of the brain, it is a possibility that neurons involved in the pathways of baroreflex control are also thermosensitive and are responsible for the changes in cardiovagal BRS with bathing.

In conclusion, cardiovagal BRS appears to increase during bathing. It may attenuate the bathing-induced blood pressure changes.

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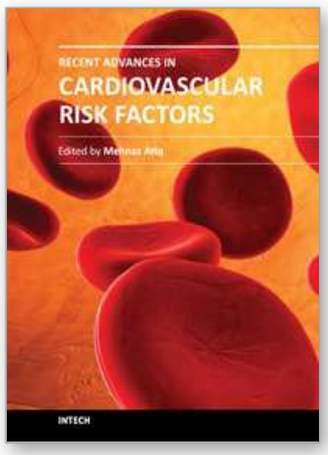
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Recent Advances in Cardiovascular Risk Factors

Edited by Prof. Mehnaz Atiq

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Among the non-communicable diseases, cardiovascular disorders are the leading cause of morbidity and mortality in both the developed and the developing countries. The spectrum of risk factors is wide and their understanding is imperative to prevent the first and recurrent episodes of myocardial infarction, stroke or peripheral vascular disease which may prove fatal or disabling. This book has tried to present an update on risk factors incorporating new research which has thrown more light on the existing knowledge. It has also tried to highlight regional diversity addressing such issues. It will hopefully be resourceful to the cardiologists, general practitioners, family physicians, researchers, graduate students committed to cardiovascular risk prevention.

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