Effects of Sauna Alone Versus Postexercise Sauna Baths on Short-term Heart Rate Variability in Patients With Untreated Hypertension

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■ **PURPOSE:** We measured the effects of sauna bathing alone or a 30-minute exercise session followed by sauna bathing on short-term heart rate variability (HRV) in subjects with untreated hypertension.

■ **METHODS:** Ten patients with untreated hypertension (age 59 ± 10 years) were randomly assigned to (1) a control resting session, (2) two 8-minute sauna-only sessions (S), or (3) a 30-minute aerobic exercise session at 75% of maximal heart rate followed by a sauna session (ES). Spectral analysis of HRV was measured with a Polar S810 heart rate monitor at baseline, during the sauna session, and 15 and 120 minutes after the sauna session (T15 and T120). A Fast Fourier Transformation was used to quantify the power spectral density of the low-frequency (LF) and high-frequency (HF) bands.

■ **RESULTS:** For S and ES conditions, LF (NU, normalized unit) and LF/HF were significantly higher ($P < .05$ and $P < .01$) in the first and second sauna sessions, and HF (NU) was significantly lower ($P < .05$, first sauna). At baseline and T15 for S and ES versus control, LF (NU) and LF/HF were significantly higher ($P < .05$), and HF (NU) was significantly lower ($P < .05$), without any effect of the 30-minute exercise session.

■ **CONCLUSIONS:** A single sauna session induced a significant alteration of autonomic cardiovascular control in patients with untreated hypertension, with an increased sympathetic and decreased parasympathetic drive. These alterations were normalized within 15 to 120 minutes after sauna bathing. Additional studies are required to document long-term effects of chronic sauna bathing on autonomic control in patients with hypertension.

K E Y W O R D S

aerobic exercise

autonomic nervous system

hypertension

sauna

short-term heart rate variability

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Hypertension is a well-established risk factor for cardiovascular disease events and outcomes.[1,2] This relation with cardiovascular risk is present independently of gender, age, race, or geographic zone.[1,3,4] There is a continuum between cardiovascular risk and the levels of blood pressure (BP),[1,5] and hypertension has been identified as a major risk factor for myocardial infarction worldwide.[6]

Cardiovascular homeostasis is under control of the autonomic nervous system (ANS), providing fast and short-term regulation of BP and heart rate (HR). The ANS is composed of 2 opposite and complementary components: the sympathetic and parasympathetic systems. Blood pressure is regulated mainly via the arterial baroreflex of the ANS, which maintains equilibrium between heart output, vessels resistance, and renal activity.

Autonomic dysregulation is present in patients with hypertension, and they have greater sympathetic nervous activity.[7] The presence of abnormal cardiac autonomic regulation can be a mechanism for cardiovascular morbidity and has prognosis significance independent of BP lowering.[8,9] Abnormal autonomic regulation has been implicated in the installation and maintenance of hypertension.[10-12] Because of the importance of abnormal autonomic regulation in patients with hypertension, it is important to quantify ANS activity and to see the potential effects of therapeutic interventions.

The analysis of heart rate variability (HRV) enables a noninvasive assessment of cardiac ANS regulation. Previous studies have shown that patients with hypertension have a diminished short-term HRV with a sympathetic predominance.[13] A previous study in patients with chronic heart failure has demonstrated that 2 weeks of daily 15-minute sauna sessions improved long-term HRV (time domain), indicating the potential benefits of sauna bathing on autonomic regulation.[14] Two previous studies in healthy young adults have shown that sauna exposure was responsible for vagal withdrawal and concomitant increases of sympathetic stimulation.[15,16] Sauna bathing is a very popular leisure activity, and sauna baths are generally taken after exercise training by patients in our center. However, little is known on the acute effects of sauna bathing alone or preceded by exercise on the autonomic regulation of the myocardium in patients with hypertension measured during and particularly after sauna bathing. The aim of this study was to measure the effects of sauna bathing alone or preceded by a 30-minute aerobic exercise session on short-term HRV in patients with untreated hypertension.

METHODS

A total of 10 men with untreated hypertension (age 59 ± 10 years; range: 35-71 years) were recruited at the Cardiovascular Prevention and Rehabilitation Centre of the Montreal Heart Institute. All individuals were EPIC Centre members for at least 3 months, 8 patients had *de novo* hypertension (diagnosed at their study entry), and the 2 others had hypertension duration of 1 and 2 years. Our center provides a multiphase, multidisciplinary approach to cardiac rehabilitation as well as exercise and risk factor control programs in the primary prevention setting.[17,18] An ambulatory BP measurement was performed for 24 hours in all the subjects using an automated sphygmomanometer (Spacelabs ABP monitor model 90207, Issaquah, WA). Inclusion criteria were age older than 18 years, no medication for hypertension, and a systolic BP between 130 and 160 mmHg and/or a diastolic BP between 85 and 99 mmHg. The research protocol was approved by the Montreal Heart Institute Ethics Committee, and written informed consent was obtained prior to study entry. Anthropometric and clinical data of the 10 subjects are presented in Table 1.

Study Procedures

At baseline, patients were evaluated with measurement of body mass, height, body composition, fasting lipid profile, resting electrocardiogram, and resting BP. Body composition was measured by bioelectrical impedance analysis (Tanita, model 418C, Tokyo, Japan) to estimate lean body mass and fat mass.[19] Then, patients were exposed to 3 different conditions in an order randomly assigned according to a crossover procedure: (1) a resting control period; (2) sauna intervention (S) consisting of an 8-minute period inside the sauna followed by a 2-minute cold water showering period and a 10-minute rest period outside the sauna, then followed by a second 8-minute period inside the sauna; and (3) an exercise and sauna intervention (ES) consisting of a 30-minute continuous exercise session on ergocycle at an intensity of 75% of maximal HR, followed by the same sauna intervention described in the S condition. Heart rate variability was measured in the 3 conditions during 10 minutes at baseline, during the last 8 minutes of the 2 sauna sessions, and 15 and 120 minutes (for 10 minutes each) after the complete session. The experimental procedure and HRV measurement times are illustrated in Figure 1.

Sauna Baths

The sauna was a standard dry sauna made of cedar wood with dimensions of 4.83 × 2.44 m, height of

Table 1 • General Characteristics of the 10 Patients With Untreated Hypertension

Parameters	Mean ± SD or n (%)
Age, y	59 ± 10
Body mass, kg	97 ± 14
Height, m	1.74 ± 0.05
BMI, kg/m^2	32 ± 5
Fat mass percentage, %	30.2 ± 7.4
Waist circumference, cm	111 ± 15
Lean body mass, kg	67.4 ± 6.1
Resting SBP/DBP, mmHg	141/87 ± 7/7 (median, 143/88)
24-h ABPM, SBP/DBP, mmHg	133/81 ± 9/5 (median, 137/80)
Fasting glycemia, mmol/L	5.86 ± 1.33
Total cholesterol, mmol/L	5.22 ± 1.44
HDL-cholesterol, mmol/L	1.24 ± 0.30
LDL-cholesterol, mmol/L	3.29 ± 1.30
Total cholesterol/HDL	4.37 ± 1.45
Triglycerides, mmol/L	1.51 ± 0.67
Triglycerides/HDL	1.38 ± 0.93
Antiplatelets agents	2 (20%)
Statins	5 (50%)

Abbreviations: ABPM, ambulatory blood pressure measurement; BMI, body mass index; DBP, diastolic blood pressure; HDL, high-density lipoprotein; LDL, low-density lipoprotein; SBP, systolic blood pressure.

2.08 m, and a bench 51 cm above the ground. The sauna temperature was kept constant between 85°C and 90°C (185°F and 194°F) with a relative humidity of 50% to 60%.[20] Patients were studied during the first 8-minute period and the second 8-minute period inside the sauna. After the first 8-minute period in the sauna, the patients showered in cold water for 1 to 2 minutes; after resting for 10 minutes outside the sauna, they returned to the sauna for the second 8-minute period. They were always accompanied by a nurse and a physician. The patients were allowed to leave the sauna at any time if they felt uncomfortable, but all patients remained in the sauna for the 2 sessions. Patients were instructed not to use a sauna or exercise for 3 days before the testing.[20]

Aerobic Exercise Session

The aerobic exercise session was performed on an ergocycle (Startrac, model E.UB, Irvine, CA), after a warm-up of 5 minutes, patients performed a 30-minute continuous exercise session at an intensity of 75% of their maximal HR, which was measured during their last physical stress test evaluation in our center.[18]

Measurement of HRV

Heart rate variability measurements for the 3 conditions were performed at the same hour of the day, in the same semidark room, and in steady state conditions in the last 256 seconds (4.27 minutes) of each analysis period. In the sauna, patients wore shorts and sat upright on the bench, and they were told to stay quiet and not talk during HRV measurement. Respiratory frequency was not controlled during the sauna. Outside the sauna, patients rested comfortably during the recording for at least 10 minutes in a seated position in a quiet and semidark laboratory room, maintained at a temperature of 21°C. To control the respiratory influence on HRV, patients matched their breathing frequency to an auditory metronome set at 0.20 Hz (12 breaths per minute). No attempt was made to control tidal volume. R-R intervals were measured continuously using a Polar HR monitor S810 (Polar Electo Oy, Kempele, Finland) with an accuracy of 1 ms, which has been previously validated.[21] To perform frequency domain analysis, raw R-R intervals were edited to applied visual inspection to the signal, so that artifact and nonsinus beats could be replaced by interpolation from adjacent normal R-R intervals. The signal was then considered to be normal and to provide normal-to-normal intervals. The same segments of 256 seconds (4.27 minutes) were resampled at 2 Hz and detrended for subsequent analysis. As recommended by the Task Force,[22] spectral decomposition of the R-R intervals variability (RRIV) was performed applying a Fast Fourier Transformation and led to typical spectral profiles with 2 main components reflecting 2 main constitutive oscillations in HRV or R-R intervals variability: (1) in the low-frequency (LF) range (0.04–0.15 Hz), which represented both sympathetic and parasympathetic controls of the heart, and (2) in the high-frequency (HF) range (0.15–0.40 Hz) as an index of parasympathetic control of the heart.[23] Power spectral densities were quantified in LF and HF ranges and expressed in both absolute (ms^2) and normalized units (NU, % of LF + HF). Additional calculations included total power (ie, LF + HF), and the LF/HF ratio as an index of ANS balance. All analysis was performed with HRV Analysis Software v1.1 (Biosignal Laboratory, University of Kuopio, Kuopio, Finland).

Statistical Analysis

All data were analyzed using StatView software version 5.0 (SAS Institute Inc, Cary, NC) and are presented as mean ± SD except where otherwise indicated. A 2-way analysis of variance (time × condition) was performed to compare HRV parameters. A

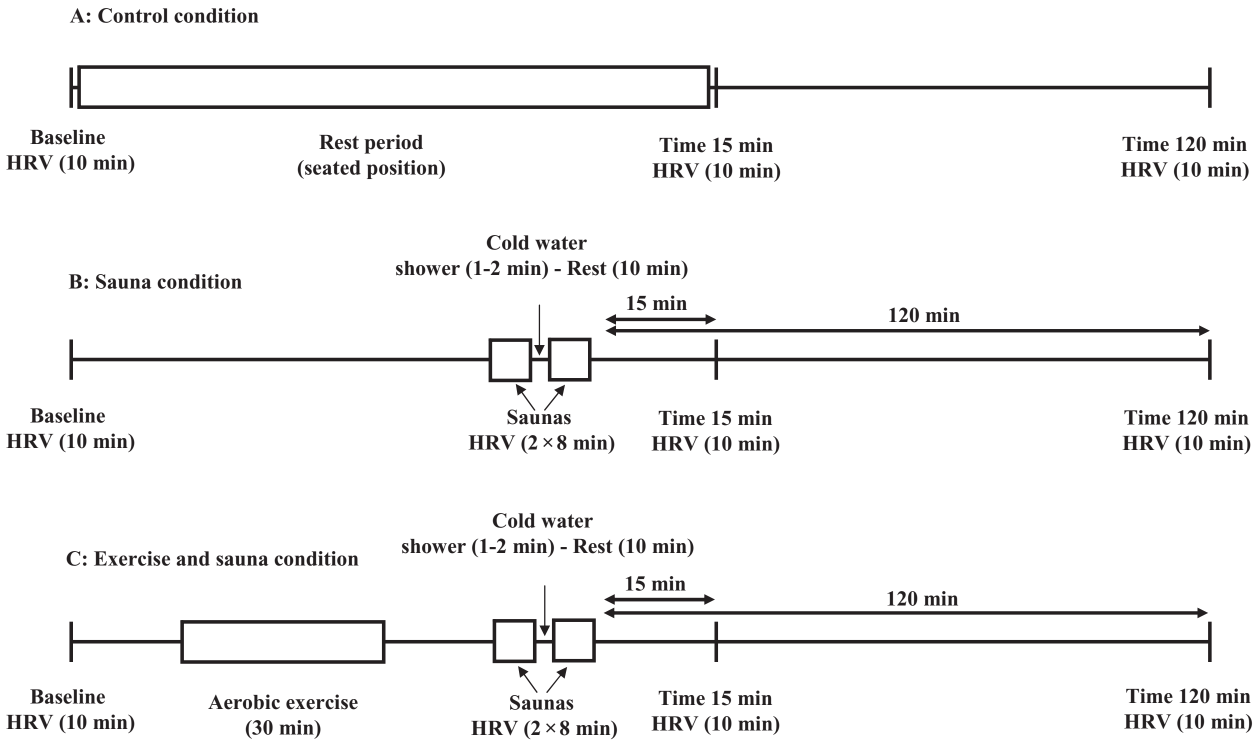

Figure 1. Experimental procedure and heart rate variability (HRV) measurement times.

Bonferroni-Dunn *post hoc* test was used to localize differences. Statistical significance was set at $P < .05$ level for all analysis.

RESULTS

HRV Measurement

Control, S, and ES comparison at baseline, T15, and T120

Heart rate variability parameters for control, S, and ES comparison at baseline, T15, and T120 are presented in Table 2. LFs (NU) were significantly higher for S and ES versus control at baseline and T15 ($P < .05$). HFs (NU) were significantly lower for S and ES versus control at baseline and T15 ($P < .05$). The LF/HF ratio was significantly higher for ES versus control at T15 (effect size: -0.95, $P < .05$).

S and ES comparison at baseline, during the 2 sauna sessions, and at T15 and T120

The HRV variables measured at baseline, during the 2 sauna sessions, and at T15 and T120 for the S and ES conditions in patients with untreated hypertension are presented in Figure 2 (panels A-F). Compared to resting baseline values and for the S and ES conditions, the LF/HF ratio was significantly higher ($P < .01$) during the first and second sauna sessions (Figure 2B), LF (NU) was significantly higher ($P < .05$) during the 2 sauna sessioins (Figure 2D), and HF (NU) was significantly lower ($P < .05$) (Figure 2F). There was no difference between S and ES conditions regarding LF (NU), HF (NU), or the LF/HF ratio.

DISCUSSION

The aim of this study was to measure the effects of sauna bathing alone or preceded by a 30-minute aerobic exercise session on short-term HRV in patients with untreated stage 1 hypertension. Our main finding was that a single sauna bath (preceded, or not, by aerobic continuous exercise) induced a physiological response of autonomic control of the cardiovascular system in these patients, in agreement with previous studies in younger healthy subjects,[16] and that these changes were normalized within 15 to 120 minutes. To our knowledge, this is the first study reporting the effects of a single sauna bathing preceded, or not, by aerobic continuous exercise on short-term HRV in hypertensive patients. Although our study demonstrated an increased sympathetic and decreased parasympathetic control of the heart during the sauna sessions, these changes were not maintained after heat exposure (15 and 120 minutes after), suggesting that 1 sauna session preceded, or not, by moderate exercise has no deleterious effects (sympathetic predominance) on cardiovascular autonomic function in hypertensive patients.

Table 2 • Heart Rate Variability Variables Measured at Baseline and 15 and 120 Minutes After the Second Sauna Time for the 3 Conditions (Control, Sauna, and Exercise and Sauna) in Patients With Untreated Hypertension (Mean ± SD)

	Baseline			T 15 min			T 120 min			
Parameters	Control	Sauna	Exercise Sauna	Control	Sauna	Exercise Sauna	Control	Sauna	Exercise Sauna	ANOVA and *P* Value
LF + HF, ms^2	170 ± 176	140 ± 140	174 ± 149	186 ± 158	142 ± 152	105 ± 110	321 ± 221	250 ± 232	199 ± 319	a: .39 b: .06 c: .87
LF, ms^2	104 ± 140	101 ± 119	109 ± 102	123 ± 114	110 ± 134	82 ± 93	207 ± 245	184 ± 185	132 ± 201	a: .59 b: .09 c: .92
HF, ms^2	65 ± 50	38 ± 27	64 ± 69	62 ± 47	32 ± 23	23 ± 20	114 ± 112	66 ± 55	66 ± 119	a: .10 b: .05 c: .80
LF, NU	57 ± 21	66 ± 16[a]	61 ± 17[a]	58 ± 20	71 ± 11[a]	71 ± 16[a]	65 ± 15	69 ± 14	70 ± 14	a: .04 b: .15 c: .87
HF, NU	47 ± 21	33 ± 16[a]	35 ± 16[a]	41 ± 20	28 ± 11[a]	28 ± 16[a]	34 ± 15	30 ± 14	29 ± 14	a: .03 b: .22 c: .89
LF/HF	1.5 ± 1.2	2.7 ± 1.8	2.1 ± 1.1	1.8 ± 1.0	3.1 ± 1.9	4.9 ± 4.9[a]	2.4 ± 1.5	3.2 ± 2.5	3.5 ± 3.2	a: .02 b: .16 c: .80

Abbreviations: a, condition effect; b, time effect; c, interaction effect (condition × time); LF, low frequencies; HF, high frequencies; NU, normalized unit.
[a]$P < .05$ vs control.

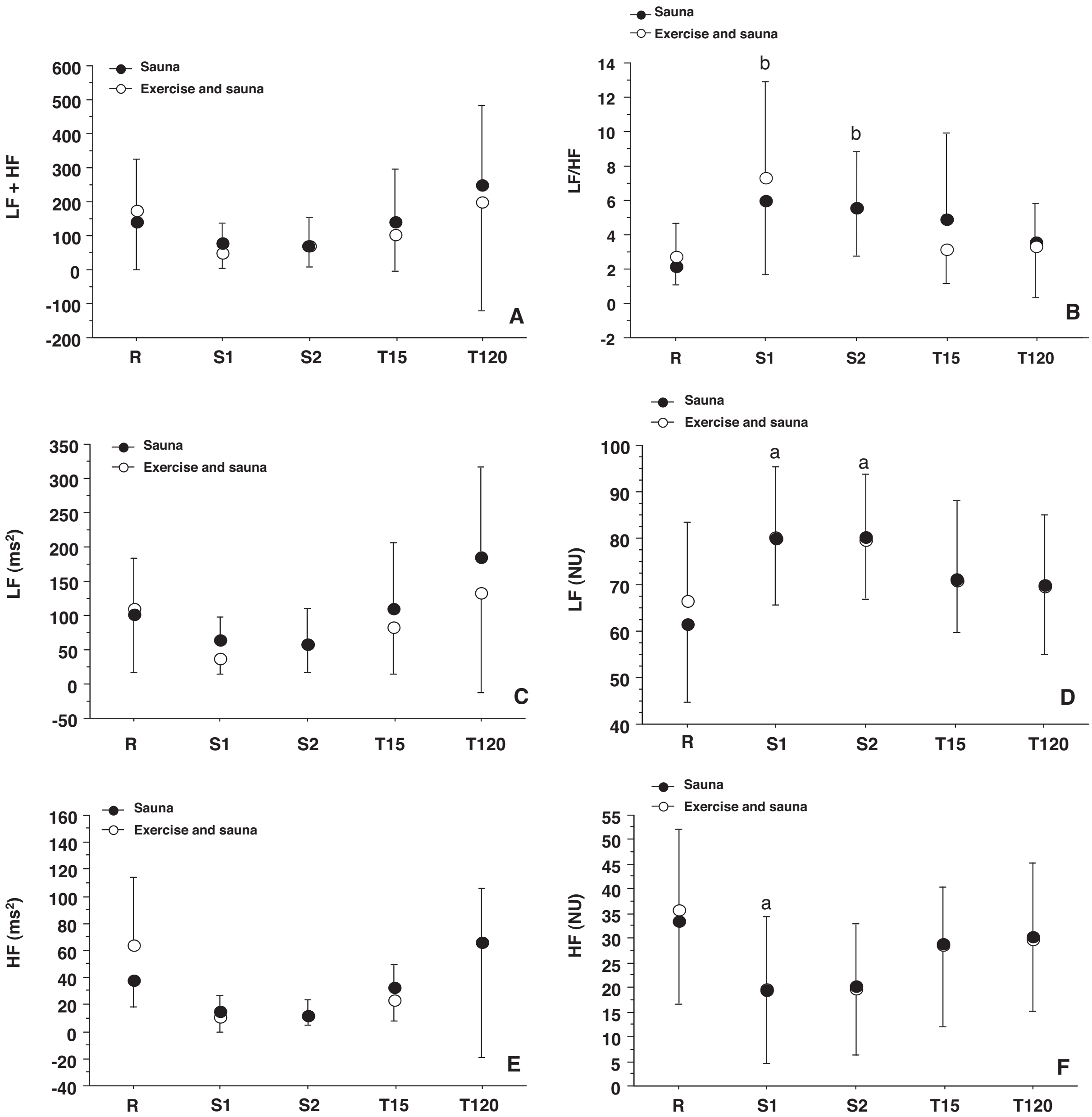

Figure 2. Heart rate variability variables measured at baseline rest (R), during the 1st and 2nd saunas (S1, S2) and 15 and 120 minutes after the last sauna time (T15, T120) for the 2 conditions (sauna, and exercise and sauna) in the patients with untreated hypertension. [a]$P < .05$. [b]$P < .001$ vs. baseline rest values (time effect). Abbreviations: HF = high frequency; LF = low frequency; NU = normalized unit.

The presence of an abnormal cardiac autonomic regulation can be a mechanism for cardiovascular morbidity despite BP lowering.[9] Previous studies have demonstrated that decreased vagal activity, as indexed by HRV, predicts overall cardiac mortality and cardiac events in low- and high-risk patients with cardiovascular risks.[24] In addition, HF power after adjustment with other cardiovascular risk factors was shown to be inversely related to the future development of hypertension, and patients with the lowest quartile of HRV had a 2.44-fold greater risk of developing hypertension compared to the highest HRV quartile.[25] Singh et al[26] also demonstrated that HRV is reduced in men and women with hypertension and that the LF component was predictive of the future development of hypertension in normotensive men. Among lifestyle interventions, exercise training, weight loss, stress management, and dietary changes are nonpharmacological interventions in patients with untreated hypertension that are efficient to positively improve HRV, which could be beneficial in terms of prevention of hypertension and cardiovascular risk.[27] Previous studies showed that regular sauna bathing can lower BP in patients with hypertension[28] and that 2 weeks of sauna bathing improved long-term HRV in patients with chronic heart failure.[14] Whether regular sauna bathing could be an additional lifestyle intervention in patients with untreated hypertension that could potentially improve HRV is still to be demonstrated with additional larger studies. We did not demonstrate different effects of sauna bathing preceded by moderate aerobic exercise on the short-term HRV in our patients during and after sauna bathing. It is well known that aerobic exercise training has beneficial effects on the autonomic function by increasing parasympathetic activity and decreasing sympathetic activity.[29] The addition of moderate aerobic exercise before sauna bathing may not represent a sufficient stimulus to modify the sympathovagal balance in our patients 15 and 120 minutes later.

Our results agree with a previous study[30] performed on healthy young men that showed a normalization of ANS activity (LF NU, HF NU, and LF/HF ratio) 15 to 30 minutes after moderate exercise (50% of oxygen uptake [$\dot{V}O_2$] reserve). In healthy young men, Terziotti et al[31] showed a decrease in HF ($ms^2\ Hz^{-1}$) 15 minutes after acute aerobic exercise performed at 50% and 80% of the anaerobic threshold with a normalization of HRV parameters (HF and LF, $ms^2\ Hz^{-1}$) 60 and 180 minutes after exercise. On the contrary, Heffernan et al[32] reported that autonomic control of the heart (LF NU, HF NU, and LF/HF ratio) was not fully regained after 30 minutes of aerobic exercise (30 minutes at 65% of $\dot{V}O_{2peak}$) in young healthy men. In the same way, Mourot et al[33] reported a significant reduction of total power, LF (ms^2), and an increase of HF (ms^2) and the LF/HF ratio 15 and 60 minutes after continuous exercise (at ventilatory threshold). Those 2 studies[33] concluded that continuous aerobic exercise was responsible for a sympathetic predominance shortly after exercise in young healthy men. Our results may differ because of the age and cardiovascular status of subjects tested (older hypertensive patients vs healthy young men) and the aerobic exercise intensity used in our study. Our study has shown differences in HF (NU) and LF (NU) between control conditions, sauna alone, and postexercise sauna (baseline and T15; Table 2) and between control conditions and postexercise sauna for the LF/HF ratio. Those differences may be due to the physiological interday and diurnal variability in HRV measurement.

Our results demonstrated that sauna bathing alone is responsible for an activation of the sympathetic activity of the heart, with higher normalized LF values (higher LF [NU]: first and second sauna) and higher LF/HF ratio (first and second sauna) observed during heat exposure and with a reduction of parasympathetic activity (reduced HF [NU] during the first sauna). The augmentation of sympathetic regulation of the heart is concomitant with an increase of HR observed during sauna exposure that was largely documented in previous studies.[34] Our results agree with the study of Kinugasa and Hirayanagi[16] that reported a reduction of HF (ms^2) and an increase of the LF/HF ratio during the last 5 minutes of 20 minutes of sauna bathing (at 60°C). In addition, Bruce-Low et al[15] showed a significant decrease in parasympathetic drive with a reduction of HF (NU) ($-82.7 \pm 11.4\%$) and an increase in sympathetic drive through both LF (NU) ($+84.5 \pm 19.4\%$) and the LF/HF ratio ($+10.9 \pm 10.8\%$) during the last 5 minutes of 15 minutes of sauna bathing (at 75°C). In our study, the amplitudes of the variations are different during the last 4 minutes of 8 minutes of the first sauna session (at 85°C), with less reduction in parasympathetic drive (HF [NU]: $-43.2 \pm 10.4\%$) and a higher increase in sympathetic activity (LF [NU]: $+25.2 \pm 13.1\%$; LF/HF: $+158.1 \pm 136.0\%$) (Figure 2, panels B, D, and F). However, in those 2 studies,[34] performed in young healthy adults, HRV parameters were measured during sauna bathing but not after.

The first original finding of this study was that we documented short-term HRV evolution during and especially after sauna exposure alone or preceded by aerobic exercise, which was not studied before in humans. Second, it is the first study of older patients with untreated hypertension compared with previous studies performed mainly in young healthy adults with no cardiovascular risk. Moreover, our patients were their own control (crossover design), which adds some strength to our study methodology. However, our study has some limitations. First, we had a small number of patients. Second, patients with untreated hypertension (including prehypertensive) represent a small proportion of patients with hypertension in our center. Our results may not be applicable to the majority of hypertensive patients, including those receiving treatment and those in other hypertension stages. Another limitation is that we did not control respiratory sinus arrhythmia during sauna sessions and tidal volume during HRV measurement; both of these parameters can influence the spectral component of HRV.[22]

In conclusion, a single sauna bath induced changes of autonomic control of the cardiovascular system in patients with untreated hypertension, as evidenced by increased sympathetic and decreased parasympathetic drive. Compared to previous data observed in younger healthy subjects, the same kinetics of sympathetic and parasympathetic drive were observed, but with differences in the amplitudes of variations, with a much greater increase in sympathetic activities and a greater decrease in parasympathetic activities. However, these changes were normalized within 15 to 120 minutes after sauna bathing, suggesting higher ANS reactivity in hypertensive subjects compared to that found in previous studies of healthy younger subjects. Additional studies are required to document any positive long-term effects of chronic sauna bathing on autonomic control of the heart in patients with hypertension. Moreover, additional studies with different hypertensive patient classification (medically treated and/or other hypertension stages) would be necessary to determine whether similar responses would be obtained.

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