

Acute Hemodynamic Improvement by Thermal Vasodilation in Congestive Heart Failure

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Abstract

Background A warm-water bath (WWB) or sauna bath (SB) has generally been considered inappropriate for patients with severe congestive heart failure (CHF). However, a comprehensive investigation of the hemodynamic effects of thermal vasodilation in CHF has not been previously undertaken.

Methods and Results To investigate the acute hemodynamic effects of thermal vasodilation in CHF, we studied 34 patients with chronic CHF (mean age, 58±14 years). Clinical stages were New York Heart Association functional class II in 2, III in 19, and IV in 13 patients. Mean ejection fraction was 25±9%. After a Swan-Ganz catheter was inserted via the right jugular vein, the patient had a WWB for 10 minutes at 41°C or an SB for 15 minutes at 60°C. Blood pressure, ECG, echo-Doppler, expiration gas, and intracardiac pressures were recorded before, during, and 30 minutes after each bath. Oxygen consumption increased mildly, pulmonary arterial blood temperature increased by 1.2°C, and heart rate increased by 20 to 25 beats per minute on average at the end of WWB or SB. Systolic blood pressure showed no significant change. Diastolic blood pressure decreased significantly during SB ($P<.01$). Cardiac and stroke indexes increased and systemic vascular resistances decreased significantly during and after WWB and SB ($P<.01$). Mean pulmonary artery, mean pulmonary capillary wedge, and mean right atrial pressures increased significantly during WWB ($P<.05$) but decreased significantly during SB ($P<.05$). These pressures decreased significantly from the control level after each bath ($P<.01$). Mitral regurgitation associated with CHF decreased during and 30 minutes after each bath. Cardiac dimensions decreased and left ventricular ejection fraction increased significantly after WWB and SB. In an additional study, plasma norepinephrine increased significantly during SB in healthy control subjects and in patients with CHF and returned to control levels by 30 minutes after SB.

Conclusions Hemodynamics improve after WWB or SB in patients with chronic CHF. This is attributable to the reduction in cardiac preload and afterload. Thus, thermal vasodilation can be applied with little risk if appropriately performed and may provide a new nonpharmacological therapy for CHF.

In congestive heart failure (CHF), peripheral vascular resistance is increased by sympathetic hypertonia to compensate for reduced cardiac output. For this reason, vasodilators were once believed to be harmful to patients with CHF. However, since Majid et al reported good results in CHF, afterload reduction has been shown to improve hemodynamic function, symptoms, and survival in

patients with CHF. Peripheral vasodilation also occurs with a warm environment, but there have been little data concerning the hemodynamic effects of warming on patients with CHF. Our previous study suggests that bathing at a temperature of 41°C in a semirecumbent position decreases cardiac afterload. Increased venous return, however, is associated with external hydrostatic pressure and results in increased preload during water immersion. To determine if warm-water bathing has clinical value in CHF, a comprehensive assessment of the hemodynamic effects of warm-water immersion is necessary. For comparison, the effect of sauna, a method of body warming without increased external hydrostatic pressure, on cardiac hemodynamics in patients with CHF would be of value to determine the extent to which changes in preload influence overall circulatory function during heat exposure.

The purpose of the present study was to evaluate the acute hemodynamic effects of warm-water and sauna bathing and the potential benefits of thermal vasodilation therapy in patients with chronic CHF.

Methods

The study subjects were 34 patients with chronic CHF, of whom 26 (19 men and 7 women) had idiopathic dilated cardiomyopathy and 8 (7 men and 1 woman) had coronary artery disease. The patients' ages ranged from 17 to 83 years (mean, 58±14 years). Symptoms at hospitalization were New York Heart Association (NYHA) functional class II in 2, class III in 19, and class IV in 13 patients. The mean left ventricular ejection fraction was 25±9% (range, 9% to 44%).

Clinical Findings for 34 Patients With Congestive Heart Failure

Of the 34 patients, 24 had a warm-water bath first so we could monitor hemodynamic changes. After a 1- to 2-day interval, they had a sauna with hemodynamic monitoring. Of the remaining 10 patients, 2 had a warm-water bath only and 8 had a sauna only. Thus, the acute hemodynamic effects of warm-water bathing were investigated in 26 patients and of sauna in 32 patients. The study was conducted without alterations in current medications. Informed consent was obtained from each patient after they fully understood the purpose and methodology of the study.

Changes in blood pressure, heart rate, and rhythm were monitored with an automatic sphygmomanometer and ECG monitor, and changes in core body temperature (pulmonary arterial blood temperature), pulmonary arterial pressure, pulmonary capillary wedge pressure, and right atrial pressure were monitored with a Swan-Ganz catheter. Cardiac output was measured by the thermodilution method. Determination of peripheral resistance was calculated using the following formulas: total systemic vascular resistance=[mean systemic arterial pressure (mm Hg)–mean systemic venous pressure (mm Hg)]/cardiac output (L/min) and pulmonary vascular resistance=[mean pulmonary arterial pressure (mm Hg)–mean pulmonary artery wedge pressure (mm Hg)]/cardiac output (L/min). We also recorded two-dimensional and M-mode echocardiograms using an ultrasound diagnostic system (SSD 870, Aloka Co, Ltd). The left ventricular and left atrial dimensions were measured on the M-mode echocardiograms, and the ejection fraction was calculated by the method of Teichholz et al before and after each bath. In patients with mitral regurgitation, regurgitant volume changes were monitored by the color Doppler method during and after the bath. Semiquantitative assessment of mitral regurgitation was performed using the area of the maximum regurgitant jet. Changes in oxygen consumption were monitored with an expiratory gas meter (MG-390, Minato Co).

In an additional study, 13 healthy control subjects (mean age, 52±13 years) and 13 patients with dilated cardiomyopathy (mean age, 56±15 years; 9 idiopathic and 4 ischemic; NYHA functional class II in 7, III in 5, and IV in 1 patient; mean left ventricular ejection fraction, 29±10%) were studied to measure changes in circulating catecholamines before, during, and 30 minutes after sauna bathing.

Before applying thermal vasodilation to patients with CHF, we conducted studies using healthy volunteers to determine the most appropriate temperature and duration of warm-water and sauna bathing. Based on these trials, we developed the methods used in the present study.

Warm-Water Bath

Monitoring of the warm-water baths was performed with a Swan-Ganz catheter inserted via the right jugular vein, an automatic sphygmomanometer, an ECG monitor, and continuous expiratory gas analysis. Room temperature was maintained at $25\pm 2^{\circ}\text{C}$. Each patient, who was wearing only underwear but was kept sufficiently warm with two blankets, reclined on a stretcher at an angle of 45 degrees. After stable baseline measurements were obtained, a bathtub permitting automatic vertical motion (Elevele; Sakai Co) was raised, and the patient was immersed up to subclavicular level (approximately the level of the right atrium). The patient was kept at rest for 10 minutes in a semirecumbent position with the bath temperature regulated at 41°C . Cardiac hemodynamic changes were monitored before, during, and after the bath. After completion of warm-water bathing, the bathtub was automatically lowered, and the patient was dried thoroughly with a towel. Hemodynamic changes were monitored for 30 minutes after completion of the bath, while the subject was kept warm wrapped in blankets on a stretcher.

Sauna Bath

Hemodynamic changes during a sauna bath were monitored using an experimental far infrared-ray dry sauna that had a window for communication and permitted monitoring in exactly the same manner as for the warm-water bath study. First, cardiac hemodynamic baseline values were obtained at room temperature with each patient in a supine position on a movable rail-bed. The patient then had a sauna for 15 minutes in the same position, while the sauna temperature was kept at 60°C (as measured where the patient was positioned). Oxygen consumption during the sauna was measured by moving the patient's head out of the sauna via the window with the subject still lying on the rail-bed. After the sauna, measurements were recorded for 30 minutes while the patient was kept at rest in a supine position with sufficient warmth provided by blankets.

All pressure and M-mode echo measurements were averaged from five heartbeats, and cardiac output was averaged from three measurements at baseline and after intervention (during and after the bath or sauna).

Results

No patients experienced dyspnea, angina pectoris, or arrhythmia during the warm-water or sauna bathing; examination was safely completed in all patients.

Oxygen Consumption

Oxygen consumption increased significantly and to nearly the same extent during both warm-water and sauna bathing, but the increase from the resting value was 0.3 metabolic equivalents (METs) on average. Oxygen consumption returned to near-control level 30 minutes after the bath, with no significant differences between warm-water and sauna bathing.

Core Body Temperature

Core body temperature, in terms of pulmonary arterial blood temperature, rose significantly to $38.2\pm 0.4^{\circ}\text{C}$ after a 10-minute warm-water bath and to $38.1\pm 0.4^{\circ}\text{C}$ after a 15-minute sauna. In both cases, the pulmonary arterial blood temperature remained significantly higher than the control level, even 30 minutes after completing the bath.

Heart Rate

Heart rate increased significantly from the control level during the warm-water bath and sauna. Although the heart rate decreased 30 minutes after warm-water bathing, it remained significantly higher than the control level.

Blood Pressure

Systolic blood pressure showed no significant change during or after the warm-water bath or sauna. With respect to diastolic pressure, however, a significant decline occurred 30 minutes after warm-water bathing, although there was no significant change during the bath itself. Diastolic pressure declined significantly during the sauna and then declined further 30 minutes after the sauna.

Cardiac Index and Stroke Index

Cardiac index increased markedly from the control level during the warm-water bath and sauna; it remained significantly higher than the control level even 30 minutes after the bath or sauna. Stroke index also increased significantly during and after the bath and sauna.

Bar graphs of changes in cardiac index during warm-water and sauna bathing. Note that the cardiac index increased significantly both during and 30 minutes after warm-water bath and sauna. Bar graphs of changes in stroke index during warm-water and sauna bathing. Note that the stroke index increased significantly both during and 30 minutes after warm-water bath and sauna.

Peripheral Vascular Resistance

Both systemic and pulmonary vascular resistances decreased significantly from the control level during the warm-water and sauna bath; this significant change persisted 30 minutes after bathing.

Bar graphs of changes in systemic vascular resistance during and after warm-water bath and sauna. Note the significant decrease in systemic vascular resistance during warm-water bath and sauna compared with control and the persistent decrease 30 minutes after each bath. Bar graphs of changes in pulmonary vascular resistance during and after warm-water bath and sauna. Note the significant decrease in pulmonary vascular resistance both during and 30 minutes after warm-water bath and sauna compared with control.

Intracardiac Pressures

Mean pulmonary arterial, pulmonary capillary wedge, and right atrial pressures increased significantly during the warm-water bath. After the bath, these parameters decreased significantly compared with baseline values. During sauna bathing, however, all of these parameters decreased significantly, with even lower values at 30 minutes after the sauna.

Plots of changes in intracardiac pressures during and after warm-water bath and sauna. Mean pulmonary artery pressure (mPAP), mean pulmonary capillary wedge pressure (mPCWP), and mean right atrial pressure (mRAP) increased significantly during warm-water bath but decreased significantly 30 minutes after the bath compared with control. In the case of sauna, these pressures decreased significantly even during the bath, with the maximum effect observed 30 minutes after the sauna.

Mitral Regurgitation

Mitral regurgitation was detected in 26 of the 34 patients at baseline. It decreased significantly in 20 of 26 patients, although to varying degrees, during the bath. No patients showed an increase in mitral regurgitation. Although the degree of regurgitation tended to return to the control level after the bath, mitral regurgitation remained significantly less than at baseline, even 30 minutes after the bath.

Color Doppler echocardiographs of the same patient with dilated cardiomyopathy showing change in functional mitral regurgitation during warm-water bath and sauna. Note that mitral regurgitation is significantly decreased during warm-water bath and sauna compared with control.

Cardiac Dimensions

In 20 of 26 patients who underwent warm-water bathing and in 28 of 32 patients having a sauna, measurements of left ventricular and left atrial dimensions could be made on M-mode echocardiograms acquired before and 30 minutes after thermal vasodilation. In the remaining patients, M-mode tracings were considered unsuitable for further analysis because the cursor could not be directed perpendicular to the short-axis image of the left ventricle. Both left ventricular and left atrial dimensions significantly decreased after thermal vasodilation.

Ejection Fraction

Ejection fraction increased significantly after warm-water and sauna bathing compared with control values.

Arrhythmias

Isolated ventricular extrasystoles occurred sporadically in 2 patients (6 and 31) during warm-water bathing. In contrast, coupled or multiform extrasystoles, which occurred frequently before sauna bathing, decreased during sauna and almost disappeared after sauna in 2 patients (15 and 33).

Plasma Catecholamines

Epinephrine levels did not change significantly during and after sauna bathing in both healthy control subjects and patients with dilated cardiomyopathy. On the other hand, plasma norepinephrine increased significantly during sauna in healthy control subjects and in patients with dilated cardiomyopathy and returned to control levels 30 minutes later in both groups.

Discussion

The present study represents the first investigation into the hemodynamic effects of thermal vasodilation in patients with CHF. We confirmed that a warm-water bath or sauna decreases systemic and pulmonary vascular resistances and increases cardiac and stroke indexes after transient thermal exposure as indicated by the rise in pulmonary arterial blood temperature. In addition, right atrial, pulmonary arterial, and pulmonary capillary wedge pressures decrease with improvement in ejection fraction after bathing. The key findings appear to be improvement in left and right ventricular functions associated with reduction in afterload (total peripheral vascular resistance for the left ventricle and pulmonary vascular resistance for the right ventricle) on thermal vasodilation. Warming is also expected to dilate the venous system. The redistribution of blood from the intrathoracic compartment toward the peripheral venous system with thermal venous dilatation results in a decrease in pulmonary congestion. The increase in venous capacity is important in preload reduction as indicated by the decrease in right atrial pressure and left atrial and left ventricular dimensions. The increase in heart rate may be associated with a vasodilation-related reflex and the direct cardiac effect of warming or stimulation of the sympathetic nervous system; however, the increase in stroke volume suggests improved cardiac function by afterload reduction.

A major reason why warm-water or sauna bathing has been considered inappropriate for patients with CHF is the concern that cardiac work increases. Therefore, in the present study, the effects of thermally induced sympathetic hypertonia and various bath-related activities were minimized; the warm-water bath temperature was set at a comfortable 41°C, and a bathtub with automatic motion was used. None of our patients complained of excessive heat stimulation. In the sauna experiment, a far infrared-ray sauna was used at a room temperature of 60°C to prevent thermal stimulation due to high-temperature air inhalation. This temperature is lower than that of a conventional sauna. Physical activity was minimized by providing aid to the patients when entering or leaving the sauna. As a result, both warm-water and sauna bathing were safely accomplished without arrhythmias, dyspnea, or angina occurring, and there was an increase in oxygen consumption of only 0.3 METs.

Attention should be paid, however, to the effects of hydrostatic pressure during warm-water bathing. With the water level below the subclavicular region and patients in semirecumbency with the lower limbs extended, as in the present study, right atrial, pulmonary arterial, and pulmonary capillary wedge pressures increased significantly during the bath; these findings are consistent with those of previous studies. All pressure parameters decreased with abolition of hydrostatic pressure after the bath and as anticipated were not significantly increased during the sauna. Thus, the increase in intracardiac pressures during warm-water bathing is mainly attributable to the increase in venous return by hydrostatic pressure. Although none of our patients experienced dyspnea during warm-water bathing, deeper immersion and thermal stress with higher bath temperatures may further increase intracardiac pressures. In studies of healthy volunteers, right atrial pressures increased to approximately 15 to 20 mm Hg during immersion up to the neck. It has been suggested that water immersion to the neck causes a greater blood shift into the thorax. In this regard, the sauna, where hydrostatic pressure is nonexistent, appears safer than warm-water bathing. Therefore, when warm-water bathing is performed for severe CHF, it may be important to ensure that the water depth is below subclavian level and to keep any exposed skin warm with a towel.

Concerning arrhythmias, Luurila has reported a 10% to 20% incidence of extrasystole induction or tachycardia during sauna bathing at 90°C in the post-myocardial infarction setting. The same report also states that extrasystoles disappeared from approximately 10% of the subjects. On the other hand, extrasystoles were sometimes observed in some healthy persons having a sauna and in healthy subjects during the first few minutes of warm-water immersion to the neck level. It is speculated that an acute strain of the right heart may elicit extrasystoles. In the present study of patients with CHF, however, only 2 patients showed sporadic extrasystoles during warm-water bathing, whereas multiform or coupled extrasystoles disappeared in 2 patients after the 60°C sauna. It appears unlikely that warm-water and sauna bathing aggravate arrhythmias, provided that due care is paid to providing correct bath temperature, to limiting patient exertion, and to ensuring correct depth of water immersion.

A warm-water bath or sauna reportedly affects sympathetic nervous system activity and norepinephrine, renin, and thyroid hormone secretions in healthy subjects. According to the study by Kukkonen-Harjula et al of healthy men exposed for 19 to 32 minutes to a 80° to 100°C dry sauna until subjective discomfort was felt, plasma norepinephrine level increased approximately 100% to 160%. However, the increase in plasma norepinephrine level was considerably less during 15 minutes of dry sauna at 60°C in patients with dilated cardiomyopathy (25%) as well as in healthy subjects (32%). With higher temperatures and prolonged exposure until subjective discomfort, sympathetic nervous system stimulation was even more pronounced and resulted in a greater increase in plasma norepinephrine level. Thus, we believe that use of comfortable temperatures and optimization of the duration of sauna bathing results in less sympathetic nervous system stimulation by heat stress.

In the present study, the acute hemodynamic changes in CHF were examined for one warm-water bath or sauna. The long-term effect of repeated baths remains a subject of interest. A number of patients have since shown good responses to several months of daily warm-water or sauna bathing (unpublished data). We are impressed that repeated thermal vasodilation improves the quality of life of patients by permitting an increase in daily activities and improving appetite, sleep quality, and general well-being. However, further investigations are needed regarding the clinical applicability of warm-water and sauna bathing as a nonpharmacological, vasodilation therapy for patients with CHF. In the present study, we did not demonstrate the effect on the myocardial circulation of thermal vasodilation. By placing a Webster multithermistor catheter into the coronary sinus during and after sauna in patients with idiopathic dilated as well as ischemic cardiomyopathy, we have recently found in a limited number of patients that thermal vasodilation causes a decrease in coronary vascular resistance, an increase in coronary sinus flow, and improvement of myocardial metabolism. These data suggest that thermal vasodilation may be responsible for increased myocardial blood flow and clinical improvement in these patients.

The method of warm-water or sauna bathing we describe should be safe in patients with CHF. We have not observed patients with CHF to deteriorate symptomatically during and after the bath or sauna using this method; all patients in the present study remained comfortable during the procedure. However, a few caveats must be emphasized. First, although we have previously shown an improvement in myocardial blood flow with sauna bathing, there remains the potential danger of an increase in heart rate in patients with ischemic cardiomyopathy during thermal exposure. Second, more exhaustive neurohumoral studies are required to establish the safety of thermal vasodilation therapy; although the transient rise in norepinephrine levels during bathing is of uncertain significance, other studies have demonstrated an association between high catecholamine levels and a poorer prognosis in patients with CHF.

In conclusion, cardiovascular hemodynamics improve after a warm-water bath or sauna in patients with chronic CHF due to ischemic or idiopathic dilated cardiomyopathy. This is presumably due to a reduction in cardiac preload and afterload by thermal systemic arterial, pulmonary arterial, and venous vasodilation. The results of the present study suggest that thermal vasodilation has salutary effects even for patients with severe heart failure and may represent a novel nonpharmacological therapy for patients with CHF. The long-term benefits of these interventions warrant further investigation.

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