Beneficial effects of sauna bathing for heart failure patients

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Generally, the sauna bathing has been contraindicated for patients with chronic heart failure. However, it has been well tolerated and improved hemodynamics has been shown in patients with chronic heart failure after a single exposure and after a four-week period of sauna bathing (five days per week). Left ventricular ejection fraction increased from 24±7% to 31±9% and left ventricular end-diastolic dimension decreased from 66±6 mm to 62±5 mm after four weeks. In the present review, the mechanisms of action, the clinical data available to date and the possible beneficial effects of sauna bathing for patients with heart failure are discussed, as well as the precautions and the contraindications in this specific group of patients with chronic heart failure.

Key Words: *Congestive heart failure; Endothelial function*

Although sauna bathing causes various acute, transient cardiovascular and hormonal changes, it is well tolerated by most healthy adults and children. Some studies have suggested that long-term sauna bathing may help lower blood pressure in patients with hypertension and improve left ventricular ejection fraction in patients with chronic congestive heart failure (CHF) (1). Contraindications to sauna bathing include unstable angina pectoris, recent myocardial infarction (MI) and severe aortic stenosis. However, sauna bathing is safe for most people with coronary artery disease, stable angina pectoris or old MI (1). Very few acute MIs and sudden deaths occur in saunas, but alcohol consumption during sauna bathing increases the risk of hypotension, arrhythmia and sudden death, and should be avoided.

THE SAUNA (FINNISH BATH)

Unlike the Turkish bath, the sauna (or Finnish bath) has dry air and a high temperature (2). The basic modern sauna is an unpainted, wood-panelled room with wooden platforms and a rock-filled electric heater. The walls are made of spruce or pine, and the benches are made of obeche, spruce or aspen because these types of wood are less hot to sit on. The recommended temperature is 80°C to 100°C at the level of the bather's face and 30°C at floor level (3). The air should have a relative humidity of 10% to 20% (3,4). A good sauna has efficient ventilation; the air should change three to eight times per hour (3).

In a sauna, the skin temperature increases rapidly to approximately 40°C (3,5,6), whereas the increase in rectal temperature depends on heat exposure (7-9). Sweating begins quickly and reaches its maximum at approximately 15 min, with an average total secretion of 0.5 kg (4,5,10). Skin blood flow increases from 5% to 10%, becoming 50% to 70% of the cardiac output, while blood flow to internal organs decreases (11). Cardiac output increases by 60% to 70% in relation to the increase in heart rate (11-13), while cardiac stroke volume does not change (11,13).

Very few sudden deaths take place during or after sauna bathing. Of all sudden deaths in Finland within one year, only 102 (1.7%) occurred within 24 h of the sauna bath (14). One-third of these were accidental, due to consumption of alcohol or drowning; the majority of the non-accidental deaths were due to acute MI in which alcohol intake was an important contributing factor (14). An epidemiological study by Romo (15) showed that very few acute MIs occur during sauna bathing. Of 1631 acute heart attacks or sudden coronary deaths that occurred during a 16-month period in Helsinki, Finland, ischemic symptoms began or death occurred within 3 h of sauna bathing in only 29 patients (1.8%) (15).

In a large prospective study of 12,310 Finnish men and women, 30 to 59 years of age, there were 77 sudden coronary deaths during the six-year follow-up period but only two of these occurred in saunas (16).

Generally, sauna bathing has been contraindicated for patients with chronic heart failure. However, it has been well tolerated, and improved hemodynamics has been shown in patients with chronic heart failure after a single exposure and after a four-week period of sauna bathing (five days per week). Left ventricular ejection fraction increased from 24±7% to 31±9% and left ventricular end-diastolic dimension decreased from 66±6 mm to 62±5 mm after four weeks (17).

Sauna bathing has the potential to be an effective therapeutic option for patients with hypertension or CHF, as well as for patients with known coronary artery disease risk factors. The common mechanism of action is improvement in vascular endothelial function, which reduces cardiac preload and afterload. It would also be interesting to know – provided the improvement is permanent – whether some of the damage on

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the cardiovascular system can be reversed by sauna therapy. Sauna bathing can be risky in patients receiving alcohol, beta blockade and nitrates (18).

Severe aortic stenosis, unstable angina pectoris and recent MI are contraindications to sauna bathing (19). Decompensated heart failure and cardiac arrhythmia are relative contraindications (20). Elderly persons prone to orthostatic hypotension should be cautious in the sauna because a decrease in blood pressure may cause syncope, usually just after sauna bathing (20).

In the present review, we will discuss the mechanisms of action, the clinical data available to date and the possible beneficial effects of sauna bathing for patients with heart failure, as well as the precautions and the contraindications in this specific group of patients with chronic heart failure.

SAUNA IN HEALTHY VOLUNTEERS AND IN PATIENTS WITH CHF

The physiological effects of exposure to Finnish saunas (80°C to 90°C with 30% to 40% relative humidity) were investigated in 60 healthy volunteers (33 men and 27 women, aged 18 to 63 years). Marked physiological changes appeared in the first few minutes in the sauna without any prodromal warning. After 20 min the mean heart rate was 143±25 beats/min, mean rectal temperature was 38.6±0.6°C and skin temperature was 40.4±1°C. In addition, mean systolic blood pressure was 130.5±26.6 mmHg and mean diastolic blood pressure was 66.6±15.9 mmHg. Three subjects experienced syncope, and one developed an anginal attack with electrocardiogram changes suggestive of an acute coronary event (21).

Recently, the temperature of sauna baths worldwide was decreased to 60°C, and a study (22) in Japan evaluated the safety and efficacy of repeated 60°C sauna bathing in patients with chronic systolic CHF. This study included 15 CHF patients with New York Heart Association (NYHA) class III, all in stable condition. Sauna bathing was performed once per day for four weeks. No adverse effect was observed. Symptoms improved in 13 of 15 patients after four weeks. Sauna bathing decreased systolic blood pressure without affecting heart rate, resulting in a significant decrease in the rate-pressure product. Left ventricular ejection fraction, by echocardiography, was significantly increased from 30±11% to 34±11%. Sauna bathing significantly improved exercise tolerance manifested by prolonged 6 min walking distance, increased peak respiratory oxygen uptake and enhanced anaerobic threshold. It also significantly reduced plasma adrenaline and noradrenaline levels, and reduced the number of hospital admissions for CHF.

POSSIBLE MECHANISMS OF ACTION

Animal studies

Vascular endothelial dysfunction is involved in the pathophysiology of CHF. It has been reported that sauna therapy improves vascular endothelial dysfunction in patients with CHF (1).

After four weeks of sauna therapy, endothelial nitric oxide synthase (eNOS) messenger RNA expression in the aortas of cardiomyopathic hamsters was significantly increased compared with those that did not undergo sauna therapy (23). Sauna therapy upregulated aortic eNOS protein expression. Serum nitrate concentrations of the hamsters were increased after four weeks of sauna therapy compared with the level in those that did not undergo sauna therapy. This study (23) has demonstrated that repeated sauna therapy increases eNOS expression and NO production in cardiomyopathic hamsters with heart failure.

In another study (24), an inflammatory arterial lesion was introduced by placement of a polyethylene cuff on femoral arteries of male Sprague-Dawley rats for four weeks. The thermal-treated group underwent daily bathing in hot water (41°C) for 15 min. Neointimal thickening along with immunohistochemical expression of heat shock proteins (HSP), monocyte chemoattractant protein-1 and NADPH oxidase were compared with those from a thermally untreated (control) group. Morphometric analysis demonstrated a significant suppression of neointimal thickening in the thermal-treated group compared with the level in the control group (intimal to medial area ratios of 0.01±0.01 versus 0.31±0.04, $P<0.01$). Expression of HSP was enhanced by thermal treatment, and both NADPH and monocyte chemoattractant protein-1 were augmented in the cuff-injured adventitia in the control but not in the thermal-treated group. This study (24) demonstrated that thermal treatment significantly attenuated infiltration of inflammatory cells in the adventitia and suppressed neointimal thickening in cuff-injured arteries, with the enhancement of HSP expression of oxidative stress.

Other studies (25,26) used cardiomyopathic hamsters with idiopathic dilated cardiomyopathy. Sixty 30-week-old hamsters in the sauna group underwent sauna therapy in an experimental far infrared dry sauna system, at 39°C for 15 min followed by 30°C for 20 min, in which their core temperatures were elevated approximately 1°C.

This protocol was performed once daily, five times a week. During sauna treatment the hamsters were calm, not excited or suffering, and their behaviour was quite similar to that of the controls. No hamsters died during or immediately after sauna treatment throughout the study. These studies (25,26) have shown that repeated sauna therapy improved survival in cardiomyopathic hamsters with CHF.

Human studies

The acute hemodynamic effects of thermal vasodilation were studied in 34 patients with chronic CHF (most of them in NYHA class III and IV with a mean age of 58 years and a mean ejection fraction of 25±9%) (17). All patients had a sauna bath for 15 min at 60°C. There was a mild increase in oxygen consumption, a 1.2°C increase in pulmonary arterial blood temperature and the heart rate increased by 20 to 25 beats/min at the end of the treatment. Systolic blood pressure showed no significant change; however, the diastolic blood pressure decreased significantly during the sauna treatment ($P<0.01$). Cardiac and stroke indexes increased and systemic vascular resistances decreased significantly during and after the treatment ($P<0.01$). This study (17) has demonstrated that sauna baths improve the hemodynamics in patients with CHF.

Sauna treatment has increased cardiac output and peripheral perfusion (27), and improved hemodynamic variables and clinical symptoms (17) in patients with CHF.

Experimental studies (28-31) have demonstrated that CHF impairs endothelial-dependent vasodilation. One of the proposed mechanisms by which this occurs is through decreased peripheral vascular production of endothelium-derived NO in

patients with CHF (32,33). Shear stress is an important stimulus for NO production (34-36) and the expression of eNOS (37,38). Several studies (39,40) have shown that endothelial function in patients with CHF was improved by treatment with L-arginine, angiotensin-converting enzyme inhibitors (41,42), physical training (43), dobutamine (44) or oral vitamin C (45).

In another study (46), 20 CHF patients (30 to 75 years of age) with NYHA class II and III were studied. Mean left ventricular ejection fraction was 38±14%. Ten patients served as the control group. Thermal therapy with a far infrared dry sauna was performed. Patients were placed in a supine position on a bed in a 60°C sauna for 15 min, and once removed, kept on bedrest for another 30 min with a blanket to keep them warm. In the control group, patients were placed in a supine position on a bed in a temperature-controlled (24°C) room for 45 min. Two weeks of sauna therapy improved endothelial function (measured by the noninvasive brachial artery method) and decreased plasma brain natriuretic peptide (BNP) concentrations in patients with CHF. A correlation was found between the degree of improvement in the per cent flow-mediated dilation (%FMD) and plasma BNP concentrations. Repeated 60°C sauna therapy improved peripheral vascular endothelial function, resulting in an improvement in cardiac function in patients with CHF.

This study (46) demonstrated that endothelial function in the brachial artery significantly improved after two weeks of sauna therapy. It was also found that systemic vascular resistance significantly decreased after two weeks of sauna therapy, suggesting an improvement in endothelial function in resistance vessels. Improved endothelial function leads to dilation of vessels by an increase in NO production. The fact that two weeks of sauna therapy significantly decreased systolic blood pressure in this study may reflect the improvement in endothelial function. This results in decreased afterload and, thus, increased cardiac output. These changes improved peripheral circulation, which was probably responsible for the improvement in clinical symptoms. A significant improvement in the %FMD was observed in patients whose clinical symptoms improved, whereas the %FMD did not improve in patients whose clinical symptoms did not change (46). The improvement in endothelial function after long-term repeated sauna therapy was most probably due to improved NO production by eNOS upregulation in patients with CHF. eNOS upregulation was due to a prolonged increase in shear stress; eNOS upregulation in the coronary artery may have directly improved cardiac function due to an increase in coronary perfusion (46).

Another important issue in CHF is ventricular arrhythmias. In a study by Giannetti et al (47), 30 patients (59±3 years of age) with NYHA class II or III and at least 200 premature ventricular complexes in 24 h were studied. They were randomly assigned into sauna-treated (20 patients) or nontreated (10 patients) groups. The sauna-treated group underwent a two-week program of a daily 60°C far infrared dry sauna for 15 min, followed by 30 min bedrest with blankets, five days a week. Patients in the nontreated group had bedrest in a temperature-controlled room (24°C) for 45 min. The total number of premature ventricular complexes per 24 h in the sauna-treated group decreased compared with that of the nontreated group (848 versus 3097, $P<0.01$). Heart rate variability increased and plasma BNP concentrations decreased in the sauna-treated group compared with those in the nontreated group. This study (47) has demonstrated that sauna treatment improved ventricular arrhythmias in patients with CHF.

Fifteen patients with CHF (mean age 64 years with NYHA class II or III) were assigned to six weeks of hydrotherapy or six weeks' restriction in a crossover intervention trial (48). Patients with hydrotherapy had a significant improvement in mood, physical capacity and enjoyment, and a significant reduction in heart failure-related symptoms compared with those of the restricted patients. Patients' heart rates at rest and at 50 W workload were significantly reduced by hydrotherapy. In conclusion, the use of a home-based hydrotherapy sauna was found to improve quality of life, heart failure-related symptoms and heart rate response to exercise in patients with mild chronic heart failure (48).

Another study (49) evaluated the effects of thermal therapy on endothelial function in patients with coronary risk factors. Twenty-five men with at least one coronary risk factor and 10 healthy men without coronary risk factors were enrolled. Patients in the risk group were treated with a 60°C far infrared dry sauna for 15 min and then kept in a bed covered with blankets for 30 min, once a day for two weeks. To assess endothelial function, brachial artery diameter was measured at rest, during reactive hyperemia (%FMD), again at rest and after sublingual nitroglycerin administration (per cent flow independent dilation [%FID]) using high-resolution ultrasound. At the start of the study, the %FMD was significantly impaired in the risk group compared with the level in the control group, while the %FID was similar in both groups. Two weeks of sauna therapy significantly improved the %FMD in the risk group; however, the %FID did not change. Repeated sauna treatment improved impaired vascular endothelial function in the setting of coronary risk factors, suggesting a therapeutic role for sauna treatment in patients with risk factors for atherosclerosis (49).

SUMMARY

Repeated sauna therapy (60°C for 15 min) improved hemodynamic parameters, clinical symptoms, cardiac function and vascular endothelial function in patients with CHF. In patients with CHF, clinical symptoms such as fatigue, heaviness in the limbs, edema, appetite loss and constipation are often observed due to increased peripheral vascular resistance and reduced peripheral perfusion. Sauna therapy improved the cardiac index, mean pulmonary wedge pressure, systemic and pulmonary vascular resistance, and cardiac function. Sauna treatment is considered safe for CHF patients with NYHA classes I, II and III. It seems that sauna treatment may help improve clinical symptoms and hemodynamic parameters secondary to an improvement in the endothelial function of patients with CHF whose endothelial function is impaired.

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