



Potential Role of Thermal Therapy as an Adjunct Treatment in Congestive Heart Failure

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Abstract

The clinical and economic importance of heart failure is widely recognized. The incidence of heart failure is on the increase, particularly with the aging of the population around the world. It is time for a paradigm shift in heart failure management. Alternative non-pharmacological strategies to remodel the failing ventricle will shape a major portion of heart failure therapy in the decade ahead.

Exposure to heat is widely used as a traditional therapy in many cultures. In this paper, we will review recent data that suggest thermal therapy may be helpful as an adjunctive non-pharmacological treatment for heart failure.

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Introduction

The clinical and economic importance of heart failure is widely recognized. The incidence of heart failure is increasing (%1 of the population in the western world), particularly with the aging of the population around the world, including developing countries.¹ It has been estimated that approximately 1% of the health care budget in developed countries is spent on the diagnosis and management of patients with heart failure. Heart failure cannot be cured.² Morbidity and mortality from heart failure remain unacceptably high.³ Symptomatic heart failure continues to confer a worse prognosis than the majority of cancers, with one-year mortality of approximately 45 percent.⁴⁻⁶

The most effective treatment approaches are those that correct or reverse underlying pathophysiological mechanisms and metabolic disturbances rather than hemodynamic or peripheral sequels. Use of angiotensin-converting enzyme inhibitors and β -adrenergic receptor blockers in patients with

heart failure remains low despite the results of clinical trials and evidence-based guidelines that support their use.^{7,8} The number of drugs in the contemporary heart failure regimen undoubtedly will grow, especially with promising prospects of new drugs (such as endothelin antagonists and cytokine antagonists) in preliminary clinical studies. Such drugs are often expensive with a substantial side-effect profile and variable treatment effects.⁹ Additionally, not all patients can tolerate treatment with angiotensin-converting enzyme inhibitors or β -blockers. Even if the patient initially tolerates the medication, there is a slow but continuing drop-out rate for these treatments in clinical trials. Finally, even if the treatment is tolerated, patient compliance remains an issue; and non compliance is a common cause for early readmission after hospitalization for heart failure.¹⁰⁻¹²

Surgical approaches and device therapy to improve outcomes in heart failure have been sought for several decades. A growing array of surgical options has been developed to treat patients with pharmacologically-refractory heart failure

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symptoms. Although these techniques have earned some recognition in heart failure, a limited population may benefit from these treatments.

Patients, despite optimal modern therapy, remain symptomatic. In an aging population, chronic heart failure is a frequent cause of disability and a cause of loss of independence. Therefore, any strategy that can aid the self-care of an elderly patient with chronic heart failure would be helpful, not only for the individual and close relatives but also for the community because of the high cost.

Non-pharmacological therapies play an important role in keeping elderly or severe heart failure patients stable and out of hospital. Exercise training, rehabilitation, and dietary and occupational therapy can improve the quality of life and independence of even severely affected patients.¹³

In this paper, we will review recent data that suggest thermal therapy may be helpful as an adjunctive non-pharmacological treatment for heart failure.

Sauna, Its Effects, and Hopes

It is postulated that the physiological reactions caused by a warm environment resemble those used in the modern pharmacological treatment of chronic heart failure.¹⁴⁻¹⁶

Several studies have demonstrated the beneficial acute and chronic hemodynamic and neurohormonal effects of thermal vasodilatation in patients with heart failure.¹⁷⁻²⁶

Description

The basic sauna consists of a wood-paneled room, an unpainted wooden platform, and a heat source. The size of a sauna should also be at least three m² to allow proper heat balance, suitable humidity, and adequate ventilation (3 to 8 times per hour).²⁷

Two kinds of saunas are in common use throughout the world: the Finnish sauna (“dry sauna”), in which the air temperature is usually 80 to 110° C and there is a relative humidity of 30 to 40%, and the “wet sauna” (steam bath, Turkish, or Japanese bath), in which the air temperature is about 45 to 50° C and there is a relative humidity of 100%. The saunas used for experimental research are generally dry and there are not many cases of wet saunas reported.²⁸

Dry Sauna vs. Wet Sauna

In one study, Shoenfeld et al. exposed sixty normal subjects, 33 males and 27 females, from 18 to 63 years of age, to two kinds of saunas. There was a rest period of one week between the experiments. There was a time lag of 10 to 15 minutes until there was a significant increase in rectal

temperature. There were no differences in the skin or rectal temperature of the subjects after 20 minutes of exposure. Comparing the different physiological parameters in both saunas showed significant differences only during the first 10 minutes: a greater drop in diastolic blood pressure ($p < 0.05$), a higher increase in systolic blood pressure ($p < 0.05$), and a greater rise in pulse rate ($p < 0.01$) in the dry sauna. It seems that the cardiovascular strain in the dry sauna during the first 10 minutes is higher than that in the wet one. After 20 minutes, there were no significant differences between any of the above parameters.²⁸

Physiologic Effects of Sauna

Hemodynamic Changes

There is some anecdotal evidence suggesting that sauna bathing is a type of heat exposure, which induces hemodynamic and endocrinological changes in some ways similar to those evoked by physical exercise.^{26,29,30} Many of these responses can be attributed to an increase in the sympathetic tone, which occurs during both heat stress and physical exercise.^{29,31,32} The most striking changes associated with sauna bathing are a rise in heart rate, reflecting an increased sympathetic activity, and increases in serum human growth hormone and plasma renin activity levels.³² The strain on the cardiovascular system in a sauna bath comes from the heat, humidity, and duration of the stay in the hot room, as well as the type of cool-off. The hot phase induces acceleration of the cutaneous circulation, leading to the redistribution of blood. The blood flow to the kidneys and the viscera is reduced, while the muscle blood flow is hardly affected. The skin flow is expanded by 20 to 40 times. Heart rate may accelerate up to and exceeding twice the resting rate.³³ The increase in heart rate may decline in response to frequent sauna bathing, indicating that the pulse generating system is adapted to the heat stimulus. Also, the response of rectal temperature to frequent hyperthermia is decreased. This is probably a consequence of an increased blood flow from deeper parts of the body to the skin, allowing a more effective cooling of the core at the end of repeated heat exposures.³⁴ The effect of sauna bathing on systolic blood pressure has been shown to be quite variable; results depend on the method of measurement (auscultation versus direct), the type and duration of the sauna, and the habituation of the bathers. Investigations using auscultation have demonstrated varying results, including a slight increase,³⁵ no change,³⁶ and a decrease in systolic blood pressure.^{20,37,38} In most studies, diastolic blood pressure has been shown to decline significantly during sauna bathing.^{20,33,35} Furthermore, mean arterial pressure, as measured directly through an intra-arterial technique, has been shown to decrease by 10 mmHg on average, in response to heat.^{20,38} Stroke volume is



unaffected,^{38,39} while cardiac output is increased by 60% to 70% in relation to the increase in heart rate.^{33,38,40} The total peripheral resistance is reduced about 40%. The total work of the heart does not significantly increase in the sauna.³³ In general, heart rate and myocardial oxygen demand during sauna bathing are similar to those observed during moderate to vigorous walking.^{20,38} Nonetheless, the intense vascular dilatation in the skin and the drop in peripheral resistance explain why the work of the heart and its oxygen demand are significantly lower in the sauna than during physical or emotional stress at similar levels of heart rate response.²⁰ There is no evidence that sauna bathing would elicit thrombotic episodes or bleeding tendencies.³³

Neuroendocrine Changes

Sauna bathing activates the sympathetic nervous system, the renin-angiotensin-aldosterone system, and the hypothalamus-pituitary-adrenal hormonal axis. If compared to the daily physiological variation of these hormones, the effects of sauna bathing on the hormonal and metabolic balance of healthy subjects are slight and without any significance.³¹

An interesting question remains. Why is the feeling of well-being and relaxation automatically associated with a sauna bath? In fact, during the sauna bath and immediately afterwards, there is increased sympathetic activity so that the feeling of relaxation, in this sense, would be paradoxical. But it may conceivably be comparable to cigarette smoking, in which although the plasma catecholamines are elevated, there appears to be a sensation of relaxation: perhaps the elevated noradrenaline stimulates the well-being center in the brain.⁴¹

Sweating and Acclimation

In unacclimated persons, maximal sodium excretion may reach 50 mEq/L with little water resorption. The early acclimatization found on day 1 in normal individuals is accompanied by a sweat sodium: potassium ratio of >15 at hour 2 of heat stress (45 °C - 47 °C), while on day 13 it is reduced by 50%. Acclimation to heat is accompanied by increased plasma renin activity and circulating aldosterone levels. These responses appear when these individuals are consuming either a normal or low-salt diet. Over weeks, sweat sodium losses tend to decline.

In patients with chronic heart failure and renin-angiotensin-aldosterone system activation, the sweat sodium/potassium ratio is persistently <1.0 in keeping with marked sodium retention over 2 hours of heat exposure.⁴²

Sauna Effects on Patients with Chronic Heart Failure

Hemodynamic Effects

Tei et al. studied the acute hemodynamic effects of thermal vasodilatation by using a far infrared-ray dry sauna in 34 patients with chronic heart failure. Mean left ventricular ejection fraction was 25% (range 9-44%). The investigators found that warm water immersion for 10 minutes at a temperature of 41 °C or sauna bathing for 15 minutes at 60 °C decreased systemic and pulmonary vascular resistance and increased cardiac index and stroke index following transient thermal exposure as indicated by a mean rise in pulmonary arterial blood temperature of 1.0-1.2 °C ($p < 0.01$). M-mode echocardiograms showed that left ventricular and left atrial dimensions had significantly decreased after thermal vasodilatation ($p < 0.01$). In addition, right atrial, pulmonary arterial, and pulmonary capillary wedge pressures decreased with the improvement in ejection fraction after bathing. Both left and right ventricular functions improved consequent to the reduction in afterload (total peripheral vascular resistance for the left ventricle and pulmonary vascular resistance for the right ventricle) affected by thermal vasodilatation. Mitral regurgitation associated with chronic heart failure decreased significantly, though to varying degrees, during the bath.¹⁶ The redistribution of blood from the intrathoracic compartment toward the peripheral venous system with thermal venous dilatation results in a decrease in pulmonary congestion.^{16,26,43} The increase in venous capacity is important in preload reduction as indicated by a decrease in right atrial pressure and left ventricular dimensions.

In another study, thermal vasodilatation caused a decrease in coronary vascular resistance, an increase in coronary sinus blood flow, and improvement in myocardial metabolism during and after sauna bathing in patients with dilated as well as ischemic cardiomyopathy.²⁶

The long-term effects of repeated warm water immersion and/or sauna bathing (once or twice/day, 5 days/week) were assessed in 56 consecutive patients with moderate or severe chronic heart failure. Thermal vasodilatation for 4 weeks caused a significant improvement in clinical symptoms and ejection fraction and decreased cardiac size. It was observed that repeated thermal vasodilatation had improved the quality of life of patients with heart failure by promoting appetite, sleep quality, and general well-being.²⁵

The TEI index, a combined measurement of systolic and diastolic myocardial performance, is more reflective of overall cardiac function than systolic or diastolic function alone in both ventricles, and provides a conceptually new measure of global cardiac function. The TEI index is simply measured by Doppler-derived time intervals. A significant decrease in the TEI index after sauna bathing indicates an improvement in systolic and diastolic left ventricular

functions. Filling time is prolonged significantly after sauna bathing, although heart rate increases, which is consistent with the improvement in diastolic function. Sauna therapy improves not only left ventricular function but also right ventricular function in patients with right heart failure.²⁴

Dry Sauna vs. Warm Water Immersion

Tei et al. showed that right-sided intracardiac pressures increased significantly during warm water immersion and decreased with the abolition of hydrostatic pressure after bathing. It is mainly attributable to the increase in venous return by hydrostatic pressure.^{14-16,44} Therefore, in the case of warm water immersion for severe chronic heart failure, it may be important to ensure that the water depth be kept below the subclavian level. Right heart pressures were not significantly altered (and in fact, decreased) during dry sauna bathing. Thus, the absence of a hydrostatic component during sauna makes this method of bathing preferable to warm water immersion, particularly for patients in severe chronic heart failure.^{16,26}

Hormonal Effects

Tei et al. demonstrated that epinephrine levels did not change significantly during and after sauna bathing in both healthy control subjects and patients with dilated cardiomyopathy but 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.¹⁶ On the other hand, Miyamoto et al. reported reduced plasma levels of both epinephrine and norepinephrine.⁴⁵ Kihara et al. demonstrated that the plasma concentrations of brain natriuretic peptide after 2 weeks of dry sauna treatment were significantly lower than those at baseline. Since brain natriuretic peptide levels have a direct relation with the severity of heart failure,⁴⁶⁻⁴⁸ these results show the potential benefits of sauna treatment in chronic heart failure patients. However, atrial natriuretic peptide and catecholamine concentrations were similar. The plasma levels of thiobarbituric acid-reactive substances and tumor necrosis factor- α (TNF- α) did not change after two weeks of sauna therapy.²¹

Endothelial Effects

Kihara et al. reported the effects of sauna therapy on the endothelial function of patients afflicted with chronic heart failure. Using high-resolution ultrasound, they measured the diameter of the brachial artery at rest and during reactive hyperemia (percent flow-mediated dilatation,

%FMD: endothelium-dependent dilatation), as well as after sublingual nitroglycerin (%NTG: endothelium-independent dilatation). Two-week sauna treatment significantly increased %FMD ($p=0.0006$). Percent NTG-induced dilatation was similar before and after the two-week sauna treatment. A significant correlation between the change in %FMD and the percent improvement in plasma brain natriuretic peptide concentrations was observed ($p=0.0005$). They concluded that repeated 60 °C sauna therapy improved peripheral vascular endothelial function, resulting in an improvement in the cardiac function of patients with chronic heart failure. The investigators did not clarify the precise mechanisms by which long-term sauna therapy improves the endothelial function of patients with chronic heart failure. However, the fact that two weeks of sauna therapy significantly decreased systolic blood pressure may reflect the reduction in afterload and, thus, increased cardiac output. These changes improve peripheral circulation, which is probably responsible for the improvement in endothelial function and clinical symptoms. They also found that in the patients whose clinical symptoms improved, %FMD improved significantly, whereas in the patients whose clinical symptoms did not change, %FMD did not improve.²¹

Tei et al. demonstrated that a single thermal therapy induced a 50% increase in cardiac output in patients with chronic heart failure.^{16,49} This results in increased peripheral blood flow, which increases shear stress in the vessels. The increase in shear stress leads to an increase in nitric oxide (NO) production by the vessels. Ikeda et al. reported that repeated sauna treatment regulated the endothelial nitric oxide synthase (eNOS) protein in the arterial endothelium.¹⁸ Kihara et al. found that there was no difference in the plasma levels of thiobarbituric acid-reactive substances (TBARS) before and after two weeks of sauna therapy. Therefore, it is likely that the improvement in endothelial function after long-term, repeated sauna therapy is due to improved NO production by eNOS up-regulation in patients with chronic heart failure. Yushi-yuki et al. demonstrated that four weeks of sauna therapy significantly increased eNOS messenger RNA expression in the aortas of TO-2 cardiomyopathic hamsters compared with those that did not undergo sauna therapy. Sauna therapy also up-regulated aortic eNOS protein expression.⁵⁰

Sugahara et al. reported that repeated sauna therapy reduced clinical symptoms and shunt flow in infants with ventricular septal defect ($p<0.05$). Patients with chronic heart failure caused by ventricular septal defect have reduced cardiac output and decreased peripheral blood flow, resulting in a decrease in shear forces. It is believed that these changes decrease NO production. In the study, nitrate and nitrite significantly increased ($p<0.05$) after 4 weeks of repeated sauna therapy. These findings may prove that sauna therapy increases NO production.²³ Ikeda et al. demonstrated that repeated sauna therapy improved survival in cardiomyopathic



hamsters with chronic heart failure ($p < 0.01$).¹⁹ In a clinical study, Masuda et al. found that dry sauna therapy for 2 weeks decreased systolic blood pressure ($p < 0.05$) and increased urinary 8-epi-prostaglandin F_{2α} (8-epi-PGF_{2α}) levels as a marker of oxidative stress ($p < 0.0001$) in patients with at least one coronary risk factor. The investigators suggested that the reduction in urinary 8-epi-PGF_{2α} by repeated sauna therapy might be related to the increase in shear stress and that these results might have a preventive effect in terms of atherosclerosis.⁵¹

For more information on the comparison of acute and chronic effects of dry sauna in patients with chronic heart failure see Table 1.

Clinical Effects

The data reported to date have been promising, that definitive studies are not possible because sauna treatment would be difficult to evaluate in a double-blind, placebo-controlled study.⁵² Kihara et al. reported that all chronic heart failure patients enrolled in the sauna study completed the study. None of the sauna-treated patients experienced dyspnea, angina pectoris, or palpitations. Clinical symptoms were improved in 17 of the 20 patients and were unchanged in 3 patients after the two-week sauna therapy. No patient had worsened clinical symptoms.²¹ Miyamoto et al. demonstrated that sauna bathing significantly improved

exercise tolerance manifested by prolonged 6-minute walking distance, increase peak respiratory oxygen uptake, and enhanced anaerobic threshold in 15 hospitalized heart failure patients. Sauna bathing also reduced the number of hospital admission for congestive heart failure.⁴⁵ Kihara et al. reported that repeated 60 °C sauna treatment improved cardiac arrhythmias in chronic heart failure. The investigators studied on thirty patients with New York Heart Association functional class II or III chronic heart failure and at least 200 premature ventricular contractions (PVCs)/24h assessed by 24-h Holter recordings. They were randomized into sauna-treated (n=20) or non-treated (n=10) groups. The sauna-treated group underwent a 2-week program of a daily 60 °C far infrared-ray dry sauna for 15 minutes, followed by 30 minutes of bed rest with blankets, for 5 days per week. The total numbers of PVCs/24h in the sauna-treated group decreased compared with the non-treated group. Heart rate variability increased and plasma brain natriuretic peptide concentrations decreased in the sauna-treated group compared with the non-treated group.⁵³ Increased heart rate variability is a sign of improved cardiac rhythm.⁵⁴ Based on various studies, it has been speculated that the decreased incidence of ventricular arrhythmias following thermal therapy may be attributed to decreased ventricular stretch.⁵³ Several studies have shown that patients with coronary heart disease tolerate sauna well.²⁰ No occurrences of sudden death, re-infarction, or serious arrhythmia attributed to sauna bathing in post-myocardial infarction patients have been reported in either

Table 1. Acute and chronic effects of dry sauna therapy in patients with chronic heart failure

	Acute Effects			Chronic Effects		
	Control	During	After 30min	Baseline	After 2 weeks	p value (n=20)
Heart rate (beats/min)	77±18	97±22**	81±20**a	71±13	70±11	0.61
SBP (mmHg)	115±18	116±19	110±18 ^a	107±22	97±17	0.019
DBP (mmHg)	78±10	70±12 [†]	67±11**a	63±13	61±10	0.40
CTR (%)	-	-	-	58.2±7.1	55.9±7.9	0.002**
Norepinephrine (pg/ml)	464±141	580±184**	493±130 ^b	426±234	432±333	0.91
Epinephrine (pg/ml)	45±28	48±24	43±16 ^b	31±36	32±34	0.94
Dopamine (pg/ml)	-	-	-	12±30	11±35	0.31
ANP (pg/ml)	-	-	-	107±101	90±94	0.34
BNP (pg/ml)	-	-	-	441±444	293±302	0.005**
TBARS (pg/ml)	-	-	-	2.6±1.1	2.6±1.3	0.89
TNF-alpha (pg/ml)	-	-	-	1.4±0.8	1.6±1.7	0.65
FMD (%)	-	-	-	4.4±2.5	5.7±2.5	0.0006**
NTG-induced dilation (%)	-	-	-	19.2±6.5	18.7±6.9	0.61
LVDd (mm)	71.1±9.0	-	69.0±9.1**c	59±8	57±9	0.047 [†]
LVSd (mm)	63.2±10.2	-	59.6±9.1 ^c	-	-	-
LAD (mm)	41.4±10.7	-	39.9±10.6**c	46±9	45±10	0.17
EF (%)	24.1±8.2	-	28.5±8.6**c	38±14	39±12	0.78

*All values are given as mean±SD

n values for a=32, b=13 and c=28. ** P<0.01, † P<0.05 vs. control

Data at baseline and after sauna treatment were compared by using the paired t- test

SBP, Systolic blood pressure; DBP, Diastolic blood pressure; CTR, Cardiothoracic ratio; ANP, Atrial natriuretic peptide; BNP, Brain natriuretic peptide; TBARS, Thiobarbituric acid-reactive substances; TNF-Alpha, Tumor necrosis factor-alpha; FMD, Flow-mediated dilation; NTG, Nitroglycerin; LVDd, Left ventricular end diastolic dimension; LVSd, Left ventricular end systolic dimension; LAD, Left atrial dimension; EF, Ejection fraction

Adapted from Tei et al.²⁴, and Kihara et al.³²

Norwegian or German studies of sauna.²⁰ The sauna effect on acclimatized patients to tropical climate is controversial, although one result showed that the physiological responses of individuals living in tropical regions were similar to the ones found in the literature.²⁷

Quality of Life

Heart failure is a debilitating disease; patients usually suffer from low exercise capacity in addition to many restrictive symptoms such as leg edema and dyspnea. Besides the physical manifestations, depression and sleep disorders are not infrequent in such patients. The impact of thermal therapy on the quality of life (QOL) of heart failure patients has been the subject of various studies. A German study on home-based hydrotherapy conducted QOL studies during a 6-week course of thermal therapy in patients with NYHA class II or III heart failure.²² QOL, which was assessed using a validated questionnaire, was significantly improved in terms of physical capacity, enjoyment, and relaxation. In a Swedish research, 15 patients with NYHA class II or III heart failure were instructed to perform exercises in warm water (30-34 °C) for 45 minutes, 3 times a week for 8 weeks. Two different questionnaires, the Short Form-36 Health Survey Questionnaire and the Minnesota Living with Heart Failure Questionnaire, were used. The results demonstrated that exercise capacity improvement was also associated with improvements in QOL for these patients. Two weeks of dry thermal therapy in 20 patients with dilated and ischemic cardiomyopathy was found through a self-assessment QOL questionnaire to improve a composite of symptoms including dyspnea, fatigue, edema, sleeplessness, appetite loss, and constipation in 17 out of 20 patients.^{53,55}

Heat-Shock Proteins and Cardiovascular System

The knowledge about the potential of stress-mediated up-regulation of heat-shock proteins (Hsps) synthesis is mostly from the experimental animals and in the human heart is almost nonexistent.⁵⁶ The powerful adaptive phenomenon, called preconditioning, is illustrated by the fact that a sub-lethal stress such as ischemia or heat stress (HS) applied to the myocardium enhances its tolerance to a subsequent ischemic stress.^{57,58} The delayed transient cardio protection, occurring 24-48 h after HS, results in a significant myocardial salvage following coronary occlusion and reperfusion.⁵⁷ HS induces an increase in the expression of various Hsps (Hsp110, Hsp90, Hsp70, and small molecular mass Hsps) that could all be responsible for protection against myocardial ischemia. In particular, members of the Hsp70 family have been shown to repair or remove denatured proteins within the cell,

leading to the restoration of cell function during recovery from stress.⁵⁹ Local heating of the heart is associated with elevated levels of Hsp72 and improved myocardial salvage.

Ischemic preconditioning seems to induce two distinct phases of protection: an early phase (lasting for 2-3 h), followed by a delayed one (after 24-96 h).⁵⁸ As for HS, delayed ischemic preconditioning protects the myocardium against infarction,⁶⁰ stunning,⁶¹ arrhythmias,⁶² and endothelial dysfunction.⁶³ There is now convincing evidence that HS preconditioning only induces delayed cardio protection, suggesting that it is exclusively dependent on new protein synthesis, a characteristic which could ultimately represent an advantage for clinical use.⁵⁷

Cardiovascular Contraindications

Severe aortic stenosis, unstable angina pectoris, and recent myocardial infarction are contraindications to sauna bathing. Decompensated heart failure, cardiac arrhythmia, and uncontrolled hypertension are relative contraindications.^{64,65} Although sauna bathing by patients with a history of stroke or transient ischemic attacks has not been studied, it should be avoided until the condition stabilizes. 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.⁶³

However, sauna bathing is safe for most patients with coronary artery disease, stable angina pectoris, or old myocardial infarction. Only 2% reported chest pain during sauna bathing, whereas 60% had chest pain during normal daily life.³⁹ Very few acute myocardial infarctions and sudden deaths occur in saunas, but alcohol consumption during sauna bathing increases the risk of hypotension, arrhythmia, and sudden death, and thus should be avoided.⁶⁶

Environmental temperature was found to be a risk factor for topiramate-related hyperthermia;⁶⁷ therefore, thermal therapy is not recommended for patients undergoing treatment with topiramate.⁵⁵

Patients with pulmonary hypertension should also be cautioned. While no specific mention of pulmonary hypertension was found in the literature reviewed, the monograph for epoprostenol sodium, a vasodilator used in the treatment of primary pulmonary hypertension, indicates that patients should avoid situations that promote vasodilatation such as saunas, hot baths, and sunbathing. Severe hypotension has been seen in patients treated with chronic epoprostenol infusions under such circumstances.⁵⁵

Another risk factor could be pregnancy. Although there is evidence that sauna bathing is safe during pregnancy,³⁹ sauna bathing for pregnant women in early pregnancy is suspected to increase the risk of neural tube defects.^{55,68}



Sauna and Drugs

Vanakoski et al. reviewed the effects of heat exposure on the pharmacokinetics of drugs.⁶⁹ The effects of hyperthermia on the absorption, distribution, and elimination of orally administered drugs were shown to be minor in studies with propranolol,⁷⁰ captopril,⁷⁰ midazolam,⁷¹ ephedrine,⁷¹ and tetracycline.⁶⁹ Increased skin blood flow during sauna bathing, however, enhances the systemic absorption of transdermally administered nitroglycerin⁷² and nicotine.⁷³ The absorption of subcutaneously administered rapid-acting soluble insulin was improved in men with insulin-dependent diabetes,⁷⁴ whereas the absorption of intermediate-acting amorphous insulin was not changed during sauna bathing.⁷⁵

Conclusion

Recent studies have shown that non-pharmacological peripheral vasodilatation with thermal therapy by means of sauna bathing has beneficial effects in chronic heart failure. Thermal therapy through sauna bathing increases cardiac output and peripheral perfusion in patients with chronic heart failure and improves hemodynamic variables and clinical symptoms in many of these patients.

Many scientific hypotheses and theories have been introduced in favor of the mechanisms involved in thermal therapy. The knowledge about the potential of stress-mediated up-regulation of heat stress protein synthesis, nitric oxide alterations, and endothelial dysfunctions has provided proof of the advantage of heat application in chronic heart failure patients.

Compared to pharmacological vasodilatation therapy and physical exercise training, there are several advantages of thermal vasodilatation therapy for congestive heart failure. First, it is non-pharmacological, devoid of adverse effects, and easily available and repeatable. Second, unlike exercise training, patients who are aged or have severe congestive heart failure and orthopedic limitations are not exempt from undergoing thermal vasodilatation therapy. Third, this intervention promotes mental and physical relaxation and promises to be a very economical means of therapy even for severe heart failure.

Despite positive clinical, hemodynamic, and neurohormonal effects, this type of therapy is still at its early stages of development and requires extensive research work before it becomes a standard method of therapy worldwide. Areas of study should include effective duration of treatment per patient, frequency of treatment, indigenous trends like habits/culture, practicality of treatment for a sizable number of patients in various geographical locations, etc.

Thermal vasodilatation therapy may be a valuable adjunct to pharmacological manipulation in the management of

congestive heart failure despite limitations described above.

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