CONFERENCE REPORT

Aquatic therapies in patients with compromised left ventricular function and heart failure

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Summary

With water immersion, gravity is partly eliminated, and the water exerts a pressure on the body surface. Consequently there is a blood volume shift from the periphery to the central circulation, resulting in marked volume loading of the thorax and heart. This paper presents a selection of published literature on water immersion, balneotherapy, aqua exercises, and swimming, in patients with left ventricular dysfunction (LVD) and/or stable chronic heart failure (CHF). Based on exploratory studies, central hemodynamic and neurohumoral responses of aquatic therapies will be illustrated. Major findings are:

1. In LVD and CHF, a positive effect of therapeutic warm-water tub bathing has been observed, which is assumed to be from afterload reduction due to peripheral vasodilatation caused by the warm water.
2. In coronary patients with LVD, at low-level water cycling the heart is working more efficiently than at low-level cycling outside of water.
3. In patients with previous extensive myocardial infarction, upright immersion to the neck resulted in temporary pathological increases in mean pulmonary artery pressure (mPAP) and mean pulmonary capillary pressures (mPCP).
4. Additionally, during slow swimming (20-25m/min) the mPAP and/or PCP were higher than during supine cycling outside water at a 100W load.
5. In CHF patients, neck- deep immersion resulted in a decrease or no change in stroke volume.
6. Although patients are hemodynamically compromised, they usually maintain a feeling of well-being during aquatic therapy.

Based on these findings, clinical indications for aquatic therapies are proposed and ideas are presented to provoke further research.

In patients with left ventricular dysfunction (LVD) and chronic heart failure (CHF), the acute hemodynamic responses to water immersion and swimming have not been thoroughly investigated. This paper presents a selection of published literature on water immersion, balneotherapy, aqua exercises and swimming in patients with compromised cardiac function. On the basis of exemplary exploratory studies, central hemodynamic responses, neurohumoral responses, and a feeling of well-being to aquatic therapy will be illustrated in patients, and in healthy subjects. Finally, an algorithm will be proposed which could support physicians, exercise physiologists and therapists in the decision making process for prescribing aquatic therapy.

Water immersion and central hemodynamic responses in healthy subjects

During head out-of- water immersion, a 100 cm column of water exerts a pressure of 76 mm mercury on the body surface. With swimming, the pressure is assumed to be 40 to 60 mm mercury depending on body position. This pressure compresses the superficial veins, of the lower extremities and abdomen, resulting in a shift of blood volume to the thorax and heart. On immersion up to the iliac crest the blood volume shift was not significant. However, on immersion up to the neck the central blood volume increases by about 700 ml;[1,2] 180 to 240 ml of this was allotted to the heart volume, with an enlargement of all four chambers.[2,3] Planimetry of the diastolic anterior-posterior area of the heart showed an average increase in heart size of 30% within 6 sec.[4] From a central hemodynamic point of view, a critical situation begins on immersion up to the level of the diaphragm and/or xiphoid process. At this level, both the buoyancy effect[5] and the external hydrostatic pressure result in a blood volume shift which amounts to i) up to 85% of the total shift during neck-deep immersion and ii) to a volume shift occurring when moving from an upright to supine body position out of water. [2]

At a water level above the diaphragm and/or xiphoid process, the central venous pressure parallels the external hydrostatic pressure. Central venous pressure (CVP), and pressures in the right atrium increased by up to 15 to 20 mm mercury.[2,6] Since the intrathoracic vascular compliance remains stable under a variety of conditions, the increase of CVP would reflect an equivalent increase in central blood volume ($_{D}V = C \times {}_{D}P$).[7] Consequently, left ventricular end-diastolic volume, which is considered to be an indicator of myocardial fibre length, increases. In a single case, left ventricular end-diastolic volume increased by 40 to 70 ml.[8,9] Together, increase in preload, using Starling's Law, stroke volume increased by 35 to 45%. Since both the systolic and mean arterial blood pressure were unchanged and/or slightly increased with immersion,[8,9] changes in afterload would not play a significant role in enhancing stroke volume.[6]

Neuroendocrine responses to immersion in healthy subjects and heart failure patients

The prompt and sustained increase of central blood volume induced by immersion in thermo-neutral (34.5-35ºC) water of healthy subjects[9,10] was shown to be accompanied by stimulation of the cardiopulmonary and arterial baroreceptors.[12-14] Concomitantly, sympathetic nervous system activity and systemic vascular resistance decreased, and arginine vasopressin release (AVP) and the renin-angiotensin-aldosterone axis were suppressed.[12,14] Since, in many HF patients, the baroreceptor reflex function is impaired, this could alter the modulation of sympathetic nervous activity and release of vasoconstrictor hormones, and decrease the systemic vascular resistance in response to an increase in central blood volume.

Gabrielsen et al.[15] studied HF patients in NYHA class II-III, with a mean left ventricular (LV) ejection fraction of 28% who underwent 30 min of immersion up to the xiphoid process in thermo-neutral (34.7 ± 0.2 ºC) water. For comparison, they studied healthy subjects. During immersion, besides significant increase of central venous pressure and stroke volume index in patients and controls, the systemic vascular resistance decreased similarly in both groups. Forearm subcutaneous vascular resistance decreased in the healthy subjects but did not change in HF patients. During immersion, the heart rate decreased less in HF patients whereas release of norepinephrine, renin, and vasopressin was suppressed in both groups. These findings suggest that reflex control of peripheral subcutaneous vascular beds and heart rate is blunted in compensated HF patients but that baroreflex-mediated systemic vasodilation and neuroendocrine responses to central volume expansion seem to be preserved. These findings help to better understand hemodynamic regulation during immersion in patients.

Immersion of patients with left ventricular dysfunction and chronic heart failure at rest

Pressure response

Patients who suffered an extensive anterior wall myocardial infarction between 6 and 15 weeks earlier with severe hypokinesia of the infarcted area demonstrated hemodynamic responses to upright graded immersion as follows: Immersion to the xiphoid process resulted in a mean pulmonary artery pressure (mPAP) within the normal range. On immersion up to the neck, mPAP increased to abnormal values, of 53±13 mmHg on average. The measurements corresponded to mPAP values obtained outside of water in the supine position.[16]

Output response

In patients with moderate CHF due to myocardial infarction, cardiomyopathy and/or myocarditis, both the systolic and diastolic LV cross-sectional diameters were greater during upright immersion than in an upright position outside of water. Yet, during immersion, the stroke volume was increased (61±19 ml → 86±24 ml). However, in patients with severe CHF during immersion, the systolic diameter increased more profoundly than the diastolic diameter. The patients' stroke volume either did not change, or decreased (Figure 1)[16,17] suggesting that the LV had become dyskinetic. These findings indicate that the blood volume shift due to immersion could result in a marked preload increase – even under resting conditions.

In a study by Bücking et al. (unpublished data), 20 NYHA class II patients with Q-wave myocardial infarction more than 6 weeks earlier underwent subxiphoid echocardiography when not immersed, and then immersed in an upright position. Comparing out of water conditions, with immersion, the LV systolic diameter increased on average by 21% (28±0 ml to 34±8 ml) and the diastolic diameter increased on average by 18% (45±7 ml to 53±7 ml). From these data, increases of 56% in the end-systolic volume and 46%

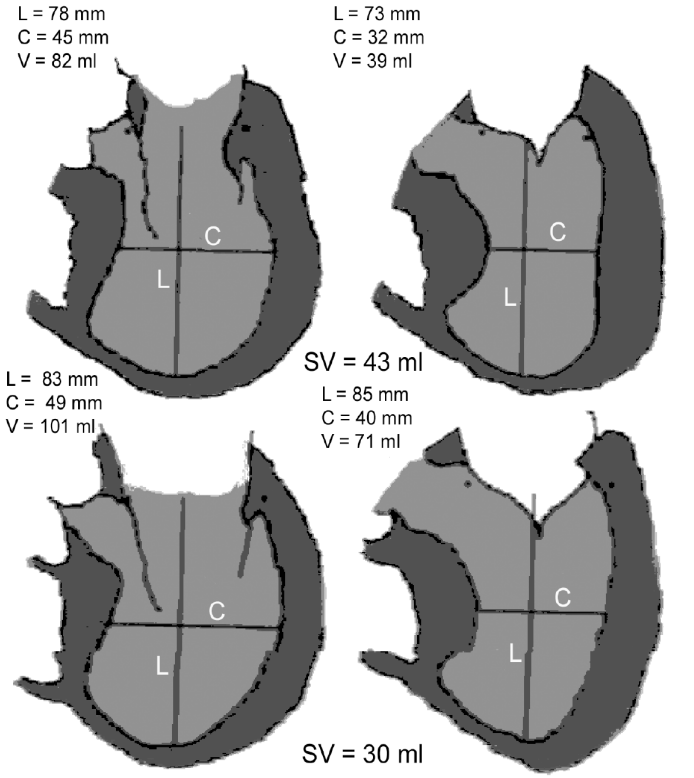

FIGURE 1. Echocardiographic imaging of the left ventricle of a patient with severe chronic heart failure. The top of the picture shows diastolic and systolic left ventricular measurements obtained from the patient standing out of water; the lower part was measured during immersion up to the neck. The left side indicates the end-diastolic and the right side the end-systolic phase of contraction (Abbreviations: L, Left ventricular length diameter; C, Left ventricular cross-sectional diameter; V, Left ventricular volume; SV, Stroke volume).

in the end-diastolic volume were calculated. This represents a 40% increase in stroke volume and a 26% increase in the cardiac index due to immersion. Concurrently, the heart rate decreased by about 10% in each patient. This decrease can be explained as a regulatory response to an increase in stroke volume. Further theories such as "the diving reflex" are not essential.[18]

Doppler flow measurements have been used to determine pulse-waved peak blood flow velocity in order to calculate stroke volume whilst patients with severe stable CHF and healthy subjects were standing upright outside of water and then immersed up to the

neck. When immersed, patients exhibited a decrease of pw-mean flow velocity on average by 7 % (range 5-10%) indicating a corresponding decrease of stroke volume.[17] For comparison, healthy subjects demonstrated an increase of pw-mean flow (34 ± 6.2 to 46.6 ±12.2 cm/s) which equates to a stroke volume increase of 38%. The patients' responses suggest that, according to Starling's Law, the preload increase could have shifted the working point of the rest-volume curve beyond a crucial point of maximum contractility, resulting in a decrease in stroke volume.[19] Because this phenomenon can be interpreted in terms of an over distension of the A-I filaments of the myocardial fibres, it entails the risk of further dilatation of a damaged myocardium.

Balneotherapy and hydrotherapy in a tub

Therapeutic warm-water bathing in a tub has a long tradition in Europe, and its clinical value is used in rehabilitation for cardiovascular patients. The positive effect is assumed to be from peripheral vasodilation which occurs with a warm water environment,[20,21] and the ensuing afterload reduction.

In a study from Tei et al.,[22] NYHA II – IV patients with CHF underwent warm water bathing for 10 min at 41º C water temperature while sitting in a semirecumbent position and immersed up to the subclavicular level which approximately corresponds to the right atrium. Although mPAP, mean pulmonary capillary pressure (mPCP), and mean right atrial pressure increased during warm-water bathing, systemic vascular resistance decreased, and both the cardiac index and stroke index increased significantly. Thirty minutes after bathing, compared with the initial measurements, cardiac dimensions were reduced, and the LV ejection fraction, cardiac index and stroke index maintained their improvement. Similar findings were reported recently by Cidar et al.[23] These findings suggest that warm-water bathing may represent a novel non-pharmacologic therapy for patients with stable CHF, attributable to the reduction of pre- and afterload. Long-term beneficial effects of such intervention need further investigation.

Swimming in patients with left ventricular dysfunction and chronic heart failure: Pressure and output responses

Contrary to immersion under resting condition, during swimming both the horizontal body position and muscular activity act as an additional stress on the heart. When patients who suffered Q-wave infarction 3-4 months earlier were in a supine position outside water they demonstrated an average mPCP of 9 mmHG (range 7-11 mmHg). In water, before swimming, a supine position resulted in highly pathological mPCPs of 19 mmHg (range 16-24 mmHg). During swimming at 20-25 m/min, in all patients the mPCP exceeded the values obtained for supine cycling outside water at 100 W load swimming: mPCP 32 mmHg, (range 16-39 mmHg) versus cycling: mPCP 29 mmHg (range 18-36 mmHg).[17] The findings indicate that, apart from muscular activity, the hydrostatic pressure rather than the supine position is the cause of the increase in preload. Even swimming at a slow speed typically used for cardiovascular training programs, can result in a marked increase of LV wall stress.

The only comparable study in patients with coronary heart disease (CAD), which compared central hemodynamics during swimming with that of cycling outside water, was done almost 30 years ago by Heigenhauser et al.[24] The authors indicated that swimming resulted in a lower cardiac output and a higher heart rate at a given metabolic rate than did cycling.

The effects of early swimming on later scar formation after myocardial infarction in humans is unknown. The only existing study for long-term effects of swimming on scar formation after acute myocardial infarction has been performed in rats. The rats started graded swimming 24 hr after experimental coronary occlusion, and swam up to 45 min daily for 7 days. Compared with a control group without swimming, the swimming rats showed marked scar thinning.[25] It

has not been studied whether, and to which degree, LV ventricular remodelling may occur if patients who are on different drug regimens undergo water exercise early after Q-wave infarction.

Comfortable swimming corresponded to relatively high overall physical stress

In order to support a proper decision on "swimming yes or no" for post-infarction patients with mild to moderate LV dysfunction, Lehmann et al.[26] assessed heart rate, blood pressure, blood lactate, and plasma epinephrine and norepinephrine, during two exercise modes: a) graded cycle ergometry in a sitting position outside of water; b) swimming at comfortable speeds. An integral of measurements obtained for all parameters during swimming were related to an integral of measurements obtained for graded work loads during cycling. By regression analysis, swimming at 20 m/min corresponded to cycling at 100W load. Swimming at 27 m/min corresponded to 150 W, and at 30 m/min to a 170 W load. These findings indicate that swimming even at slow speeds could cause a relatively high cardiovascular and hormonal stress.[26] Similarly, Magdar et al.[27] reported that comfortable swimming for CAD patients in NYHA class I and II resulted in a VO_2 of 54% to 87% of their maximal capacity, depending on their swimming ability. The weaker swimmers were exercising at over 90% of their peak VO2.

Patient's well-being is maintained

Despite hemodynamic deterioration and relatively high overall stress, patients mostly reported maintaining a feeling of well-being.[16,17,28] This phenomenon is not completely understood. However, swimming at 20-25m/min a relatively smaller decrease of mixed venous O_2 saturation (change from 74% to 47%) has been observed than at supine cycling at 100W load (change from 74% to 34%).[17] The smaller mixed-venous O_2 desaturation could be the result of an increase in blood shunting to the central circulation and to the cooling effect of the water. This could be an explanation, among others, for the feeling of well-being in water.

Medical Aqua Cycling

Very recently, MAC- Medical AquaCycling®, using an Aquarider®, was launched for the prevention and rehabilitation of muscular problems and obesity.[29,30] Before using the Medical AquaCycling®, in cardiac patients with impaired LV function it would be beneficial to have a better understanding of the hemodynamic responses during aqua cycling compared to cycling on land. For both conditions, Mc Murry et al.[28] provided information on exercise hemodynamics in 10 patients with CAD, of whom 3 had LV ejection fractions between 37 and 49%. Graded cycling was performed in a sitting position, whilst immersed up to the xiphoid process. There was a trend for the heart rates to be less in water than on land during mild exercise (VO_2 < 1 l/min), but at higher levels of exertion, the values were reversed. At each of the workload levels, cardiac output during immersion trials were observed to be slightly higher than were the land trials. The difference being particularly evident at a VO_2 level of < 1 l/min. Total peripheral resistance was lower throughout graded water exercise than during land exercise. These results suggest that in CAD patients with reduced LV function the heart is working more efficiently with mild cycling in water than cycling outside of water.

General conclusions

There are relatively few studies on the current topic. In these studies, patients' characteristics are not homogeneous. A large variety of aqua exercise modes, and intensities have been assessed. All studies focused on early and/or short-term exposure to water. This implies that cardiovascular responses as measured do not provide any evidence on:

- The optimal time after an acute myocardial infarction to start aqua therapy and swimming;

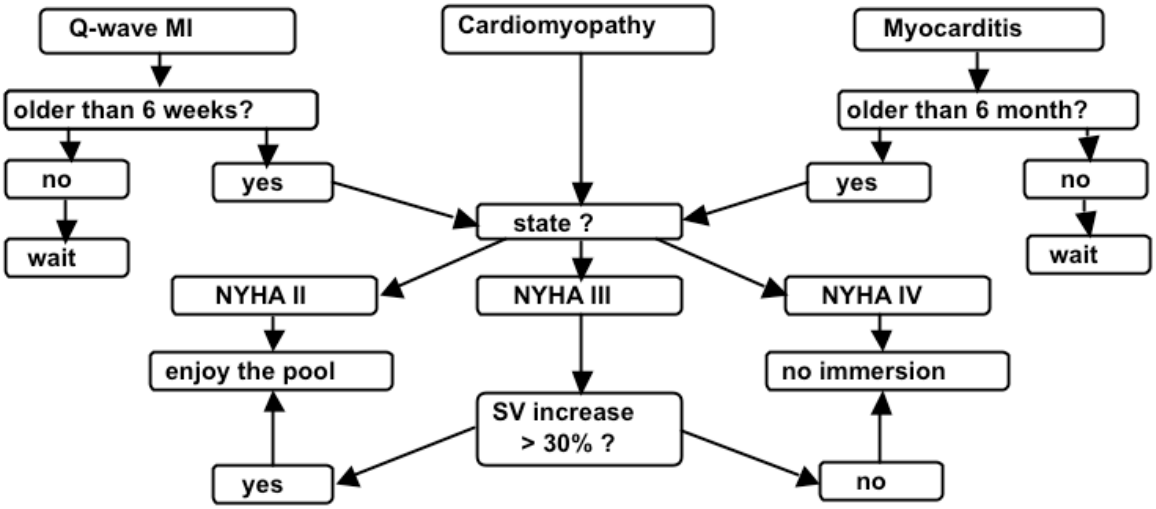

FIGURE 2. Algorithm to support decision making for prescribing swimming to patients with left ventricular dysfunction and/or stable chronic heart failure.

- Whether Diameter or Function of the LV is the more important criterion as an indicator for aqua therapy and swimming;
- Whether repeated water exercises, and swimming would lead to abnormal remodeling, and/or sustained impairment of the LV.

Clinical suggestions for rehabilitation and secondary prevention

According to the exploratory studies presented, we suggest the following clinical implications for patients with LVD and/or CHF:

1. Immersion up to the neck could produce abnormal hemodynamic responses temporarily.
2. Decompensated HF is an absolute contraindication for water therapy.
3. Feeling good in water is no guarantee that the LV tolerates the increased volume loading caused by immersion.
4. Patients with previous severe myocardial infarctions and/or CHF who can sleep supine may bathe in a half sitting position (e.g. balneotherapeutic baths), but immersed no deeper than the xiphoid process.
5. Therapeutic water exercises in a pool, e.g. for orthopaedic reasons, can be allowed for patients with Q-wave myocardial infarctions older than 6 weeks, and/or moderate CHF, provided that patients are in an upright position and immersed no deeper than the xiphoid process.

In order to provide a better rationale for prescribing swimming from the existing literature, we propose a dichotomous approach as shown in Figure 2. Moderate swimming as used for cardiovascular training programs may be allowed exclusively for NYHA class II patients with Q-wave infarction older than 6 weeks, myocarditis suffered more than 6 months ago, and cardiomyopathy. If patients are NYHA class III, they should be able to increase their stroke volume by 30% or more.

Research needs

This presentation of exploratory studies is aimed to stimulate ideas for urgent research on long-term tolerance of different aqua therapies and their effects on the myocardium. For example:

- The answer to the question: when can swimming therapy begin following an infarction event, requires a randomized controlled trial of patients with comparable parameters of: infarct localization, left ventricular diameter & function, and medications taken, etc. Swimming therapy should be of comparable intensity and duration, and starting at different time intervals following the infarction. The evolving changes in measured left ventricular diameter and function are primary outcome determinants.
- The answer to the question: whether long term active water therapy or swimming doesn't lead to cardiac deterioration in patients with serious myocardial infarctions and/or serious CHF requires a randomized, controlled trial or a large case series of patients, with different aetiologies and degrees of LVD and/or CHF, undergoing different water therapies over a long period of time. These procedures should be compared with i) current therapeutic exercise procedures on land, and ii) no therapeutic exercise procedures. The results should be progressively monitored.
- Experimental investigations are necessary, to understand the mechanisms of compensation in immersion induced increased preload, and thereby to better understand the long term adaptation of the left ventricle to water therapy and/or swimming.

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