Exercise in Heart Failure: Should Aqua Therapy and Swimming Be Allowed?

KATHARINA MEYER[1] and JÖRN BÜCKING[2]

[1]Swiss Health Observatory, Neuchatel and University of Bern, Bern, SWITZERLAND; and [2]Caspar Heinrich Clinic, Bad Driburg, GERMANY

ABSTRACT

MEYER, K., and J. BÜCKING. Exercise in Heart Failure: Should Aqua Therapy and Swimming Be Allowed? *Med. Sci. Sports Exerc.*, Vol. 36, No. 12, pp. 2017–2023, 2004. **Purpose:** Although exercise training is established as an integrated part of treatment regimes in both patients with transmural myocardial infarction (MI) and chronic congestive heart failure (CHF), there is no consensus yet on the appropriateness of water exercises and swimming. One reason is the lack of information concerning both central hemodynamic volume and pressure responses during immersion in these patients. **Methods:** This paper presents explorative studies on changes in cardiac dimensions and central hemodynamics during graded immersion and swimming in patients with moderate and/or severe MI and in patients with moderate and/or compensated severe CHF. For comparison purposes, healthy subjects were assessed. Measurements were performed by using Swan-Ganz right heart catheterization, subxiphoidal echocardiography, and Doppler-echocardiography. **Results:** The major findings were: 1) Indicators of an increase in preload were seen in patients with moderate and severe MI. In both patient groups, upright immersion to the neck and supine body position at rest in the water resulted in abnormal mean pulmonary artery pressure (PAm) and mean pulmonary capillary pressures (PCPm), respectively. During low-speed swimming (20–25 $m \cdot min^{-1}$), the PAm and/or PCPm were higher than during supine cycle ergometry at a load of 100 W. 2) Left ventricular overload and decrease and/or no change in stroke volume occurred in patients with severe CHF who were immersed up to the neck. 3) Patient's well-being was maintained despite hemodynamic deterioration. **Conclusion:** The acute responses during immersion and swimming suggest the need for additional studies on long-term changes in cardiac dimensions and central hemodynamic in both patients with severe MI and severe CHF who undergo a swimming program, compared with nonswimming patients with MI and CHF of similar etiology and severity of disease. **Key Words:** WATER IMMERSION, CARDIAC PRESSURE, CARDIAC OUTPUT, BALNEOTHERAPY

Aqua exercise and swimming are traditionally recommended for low-risk cardiac patients. In patients with severe myocardial infarction (MI) and compensated severe congestive heart failure (CHF), physicians may have reservations about water exercises and swimming. One reason is the lack of information concerning central hemodynamic volume and pressure responses during immersion in these patients.

During head-out water immersion, a 100-cm column of water exerts a pressure of 76 mm Hg on the body surface. With swimming, depending on the body position, a pressure of 40–60 mm Hg could be assumed. This pressure acts to compress superficial veins, particularly of the lower extremities and abdomen, resulting in a blood volume shift to the thorax and heart. On immersion up to the iliac crest, the blood volume shift is not significant; however, on immersion up to the neck, the central blood volume has been demonstrated to be increased by about 700 mL (1,16); 180–240 mL of this was allotted to the heart volume, with an enlargement of all four chambers (12,16). Planimetry of the diastolic posteroanterior area of the heart showed an average increase in heart size of 30% within 6 s (17). From a central hemodynamic point of view, crucial immersion starts at the diaphragm/xiphoid level. At this level, both the buoyancy effect (8) and the external hydrostatic pressure result in a blood volume shift that amounts: a) up to 85% of the total shift during neck-deep immersion, and b) to a volume shift occurring when moving from an upright to a supine body position out of water (16).

At a water level above the diaphragm and/or xiphoid, the central venous pressure parallels the external hydrostatic pressure; in this condition, the central venous pressure and pressure in the right atria were demonstrated to increase by up to 15–20 mm Hg (6,11,16). Because the intrathoracic vascular compliance tends to remain stable under a variety of conditions, the increase of central venous pressure would reflect an equivalent increase in central blood volume (9). As a consequence, the left ventricular end-diastolic volume, which is considered an indicator of myocardial fiber length, increases. In a single case, left end-diastolic ventricular volume increased by 40–70 mL (2). Along with the preload enhancement, due to the Starling mechanism, the stroke volume increased by 35–45% (4,15). Both the arterial systolic and mean blood pressure were shown to be unchanged (4,18) and/or slightly increased with immersion (6,14,15) .

Address for correspondence: Katharina Meyer, Ph.D., MPH, FACSM, Swiss Health Observatory and University of Bern, Espace de l'Europe, 2010 Neuchâtel, Switzerland; E-mail: meyer.katharina@bluewin.ch.
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The aim of this explorative study was to assess central hemodynamic responses on land and during graded immersion and swimming in various samples of patients with both previous moderate and severe MI, and moderate and compensated severe CHF, respectively. Additionally, for comparison purposes two samples of healthy subjects were assessed. With respect to the hemodynamics in healthy subjects as described in the literature, we hypothesized that in patients with severe MI and/or severe CHF during graded immersion and swimming, there is a critical point at which central hemodynamic alterations change into abnormal cardiac responses. If this hypothesis is confirmed, this study could suggest the need for additional research on the appropriateness of balneotherapy, water exercises, and swimming in such patients.

METHODS

Patients and subjects. The studies were performed in the following patient groups: group A, moderate MI; group B, severe MI; group C, moderate CHF; group D, compensated severe CHF; group E, healthy volunteers; and group F, healthy volunteers (Table 1). Moderate and/or severe MI, and moderate CHF (groups A, B, and C) were defined with respect to findings on echocardiographic wall motion: moderate, no dyskinetic and/or akinetic wall motion; and severe, dyskinetic and/or akinetic wall motion. Severe CHF (group D) was defined according to New York Heart Association class III. During the investigation, all patients were undergoing an in-hospital rehabilitation because of their heart disease. CHF patients had experienced at least one episode of acute heart failure, but were clinically stable for ≥6 wk. No patient demonstrated pathologic arrhythmias in a 24-h Holter electrocardiogram, and none exhibited pulmonary hypertension before examination. Detailed information on patients and healthy subjects are presented in Table 1. The study procedure was approved by the institutional review board. Written informed consent was obtained from all patients and subjects.

Testing procedures. All experiments (graded immersion and swimming) were performed in water with a temperature of 32°C. Graded immersion procedure was performed in upright position as follows: calf, iliac crest, xiphoid, and neck. Swimming was performed as break stroke in a countercurrent device. The countercurrent corresponded to a swimming speed of 20–25 $m \cdot min^{-1}$. To compare hemodynamic measurements from graded immersion and swimming with hemodynamic measurements from cycling out of water, a supine cycle ergometry was performed at 100 W for 5 min.

Right heart catheterization. This procedure was performed in MI patients (groups A and B). An F5 Swan-Ganz catheter was inserted in the jugular vein via a venous lock (Desilet CH 07). The catheter was made airtight by a rubber top and watertight by a foil (3M Tegaderm). The two-luminal Swan-Ganz catheter was positioned using pressure curve analysis. The transducer (Hellige Company, MA 20601001) of the mechanoelectric measuring instrument

TABLE 1. Characteristics of patients with transmural myocardial infarction, compensated congestive chronic heart failure, and healthy subjects (mean; range).

	Moderate Myocardial Infarction (*N* = 16) Group A	Severe Myocardial Infarction (*N* = 4) Group B	Moderate Congestive Chronic Health Failure (*N* = 18) Group C	Severe Congestive Chronic Healthy Failure (*N* = 5) Group D	Healthy Subjects (*N* = 10) Group E	Healthy Subjects (*N* = 45) Group F
Age of patients/ subjects	53 (43–61)	55 (51–58)	54 (25–68)	56 (36–69)	35 (29–48)	53 (30–65)
Age of myocardial infarction (wk)	15 (6–20)	7 (6–10)	8 (6–12)	>1 yr	—	—
Etiology of heart failure						
CHD	—	—	*N* = 16	*N* = 2		
DCM			—	*N* = 3		
Valve disease			*N* = 2	—		
NYHA class						
I	*N* = 3	—	*N* = 4	—	—	—
II	*N* = 9	*N* = 2	*N* = 8	—		
III	*N* = 4	*N* = 2	*N* = 6	*N* = 5		
LV DD mm[a]	45 (42–58)	47 (38–56)	44 (36–54)	65 (61–73)	42 (32–50)	41 (30–55)
LV FS[a]	54 (44–68)	0.31 (0.21–0.39)	0.38 (0.24–0.57)	0.17 (0.15–0.19)	0.36 (0.30–0.45)	0.38 (0.29–0.52)
Medication	ISDN (*N* = 7)	Beta blockers (*N* = 1)	Beta blockers (*N* = 8)	Beta blockers (*N* = 5)		
	Beta blockers (*N* = 3)	Calcium antagonists (*N* = 2)	Calcium antagonists (*N* = 4)	ACE-Inhibitors (*N* = 5)		
	Calcium antagonists (*N* = 5)	Diuretics (*N* = 2)	ISDN (*N* = 5)	Diuretics (*N* = 5)		
	Digitalis (*N* = 2)	Antiarrhythmics (*N* = 1)	Digitalis (*N* = 1)	Digitalis (*N* = 5)		
	ASA (*N* = 10)	ASA (*N* = 1)	ASA (*N* = 12)	ASA (*N* = 5)	—	—

[a] Data from clinical routine echocardiography.
CHD, coronary heart disease; DCM, dilated cardiomyopathy; NYHA, New York Heart Association; LV DD, left ventricular end diastolic diameter; LV FS, left ventricular fraction shortening; ASA, Acetylsalicylic acid.

(Gould Company, Type P23 ID) works by the "floating input" principle, and thus is appropriate for examinations in the water. For the supine position out of water, the zero adjustment was performed by a measuring rod. For measurements during immersion and swimming, the transducer was fixed at the right chest site using an elastic band, and then the zero adjustment was carried out for the sitting position. For the swimming position, the reference point used corresponded to the zero adjustment as obtained for the supine position.

Mean pulmonary artery pressure (PAm), mean pulmonary capillary pressure (PCPm), mixed venous oxygen saturation (Unistat Oxymeter; Scientific Instruments CAT 405806), and heart rate (determined by ECG-water-telemetry, Hellige Company) were measured in the following positions: at rest out of water in an upright position; during graded immersion in an upright position; at rest in the water and after 5 min of swimming in a supine position; and at rest and after 5 min of cycling in a supine position on land. Reference values for PAm and PCPm were given by Ekelund and Holmgren (3).

Echocardiography. All investigations were performed by means of Toshiba equipment (model SS-65A or SSH-140A). For the subxiphoidal studies, an ultrasonic transducer type PSF-25 FT (2.5 MHz) was used, and for transesophageal echocardiography, the phrased array endoscope PEF 511 SA (5 MHz) was used. Doppler-flow data were obtained using a transducer type PSF-25 FT.

Subxiphoidal echocardiography. Echocardiography was carried out in all patients and healthy subjects (groups A–F) in an upright position out of water by using a subxiphoidal window. The echocardiography procedure was repeated after patients and subjects had entered the pool and were in upright positions, immersed neck-deep. The left ventricular cross-sectional diameter was determined for systole (left ventricular end-systolic diameter, LV SD) and diastole (left ventricular end-diastolic diameter, LV DD), and evaluated in real-time and time-motion mode. The data presented are mean values of three measurements obtained in each patient and healthy subject, respectively. The determination of the left ventricular volume was performed by means of the echocardiography computer program, using the Teichholz formula ($V = 7 \times D^3/(2.4 + D)$), where V is volume in milliliters and D is length and cross-sectional diameter in millimeters. Stroke volume (SV, mL) and fraction shortening (FS) were calculated as follows: SV = end-systolic volume (ESV) − end-diastolic volume (EDV); FS = 1 − LVSD/LVDD.

Transesophageal echocardiography. This procedure was performed in both patients with moderate and severe CHF (groups C and D) and in healthy subjects (groups E and F). While patients and healthy subjects were in supine left-side positions on land, an i.v. injection of 5 mg of diazepam was administered, and the endoscope was inserted. Then patients and subjects stood up. After a thorough documentation of the four-chamber view out of water in the upright position, the patients and subjects were lifted into the pool using a hydraulic chair. Measurements were carried out during upright immersion up to the neck.

Doppler-flow imaging. In the upright patients (group D) and healthy subjects (group F) outside of water as well as in the water, blood flow velocities (v) were measured either from the aorta ascendens or, if technically not possible, from the truncus brachiocephalicus. Peak velocities were registered in a pw-mode. Mean velocities were computer calculated. Stroke volume was calculated using the equation:

$$SV = Ao \times \bar{v}_{sys} \times t_{sys},$$

where

$$v_{sys} \times t_{sys} = \int_0^{t_s} v_D(t)dt.$$

(Ao = area of the aortic root, sys = systole).

Statistical analysis. Data are presented as mean values with standard deviation and as range of values. For statistical comparison of dependent sample, a Student's *t*-test was performed. A P value of <0.05 was considered significant.

RESULTS

Pressure measurements during immersion at rest in patients with transmural infarction. In 16 patients who suffered from a moderate MI (group A), upright graded immersion to the xiphoid resulted in PAm that were in the normal range of values (Fig. 1). On immersion up to the neck, PAm increased up to abnormal values on average ($P < 0.01$). The PAm measured during upright neck-deep immersion corresponded to PAm values obtained outside of water for supine position, that is, for supine cycle ergometry as presented below.

Pressure measurements during swimming. In the same patients (group A), central hemodynamic responses

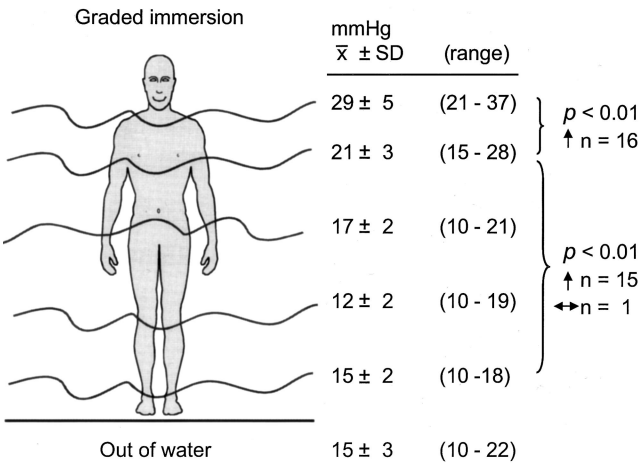

FIGURE 1—Mean pulmonary artery pressure (PAm; mm Hg) (mean ± SD; range) and changes during graded immersion in 16 patients with moderate myocardial infarction (group A); ↑, number of patients with PAm increase; ↔, number of patients with no change of PAm.

were measured during 5 min of slow swimming (20–25 $m \cdot min^{-1}$), which was perceived as comfortable, compared with supine cycle ergometry at a 100-W load. For cycling and swimming, the heart rate was similar (123 ± 11 $beats \cdot min^{-1}$ and 122 ± 15 $beats \cdot min^{-1}$). During swimming, all patients exhibited higher PAm values than during supine cycling. With swimming, the PAm increased from 15 ± 2 mm Hg at rest to 53 ± 13 mm Hg on average ($P < 0.001$) and exceeded pressures measured during cycling ergometry at 100 W (15 ± 3 at rest; 43 ± 8 mm Hg during cycling; $P < 0.001$). In two patients, the PAm exceeded 60 mm Hg; however, both of them reported a feeling of well-being. During swimming, there was a smaller decrease in mixed venous oxygen saturation than during cycling (decreases from 74.4 ± 7.0% to 46.8 ± 7.1% and 34.3 ± 8.3%, respectively; $P < 0.01$).

Figure 2 presents the PCPm of a subgroup of four patients with severe MI (group B) measured during swimming, compared with supine cycle ergometry at a 100-W load. Before starting with cycling in the supine position out of water, all patients exhibited a normal PCPm, but at the supine resting position in the water, they exhibited pathologic pressure values. During swimming, the increase of the PCPm was relatively small compared with cycling. For both swimming and cycling, the mean PCPm reached pathologic values, which were slightly higher for swimming (for a reference on pathologic values, see the legend for Fig. 2).

Left ventricular output response in patients compared to healthy subjects. In 10 healthy subjects studied (group E), both systolic and diastolic left-ventricular cross-sectional diameters were greater on upright immersion up to the neck compared with measures obtained out of water. These changes resulted in an increase of stroke volume (54 ± 21 mL out of water vs 81 ± 19 mL during immersion) (Table 2). Likewise, in immersed patients with moderate CHF ($N = 18$) (group C), both the diastolic and systolic cross-sectional diameters were greater than when out of water and accompanied with an increased stroke volume (61 ± 19 mL out of water vs 86 ± 24 mL during immersion) (Table 2). However, in patients with severe CHF (group D) during immersion, the systolic length diameter increased more pronouncedly than the diastolic length diameter, indicating that the left ventricle became dyskinetic, and the stroke volume either did not change, or decreased (Table 2). In a case study, the echocardiography imaging illustrates that with immersion the left ventricle became dyskinetic (Fig. 3).

Doppler flow imaging of the ascending aorta was used to determine changes in peak blood flow velocities of left ventricular output after moving from out of water to immersion up to the neck in 45 healthy subjects (group F). Using the pw-mean flow for calculation of the stroke volume, the output per heart beat increased from 59 ± 15 mL outside of water to 88 ± 20 mL on immersion ($P < 0.001$), indicating a 29% increase of stroke volume (and pw-mean flow velocity), on average (for a case study see Fig. 4a). The amount of increase corresponded to a stroke volume increase obtained by means of subxiphoid echocardiography (from 60 ± 20 mL outside of water to 85 ± 23 mL on immersion; $P < 0.001$). Conversely, in five patients with severe CHF (group D) assessed during immersion, the pw-mean flow velocity decreased by 7% on average (range 5–10%), implying a decrease in stroke volume (38 ± 3 mL outside of water; 34 ± 3 mL during immersion) (for a case study see Fig. 4b). All patients had a left ventricular end-diastolic diameter of more than 60 mm (see Table 1).

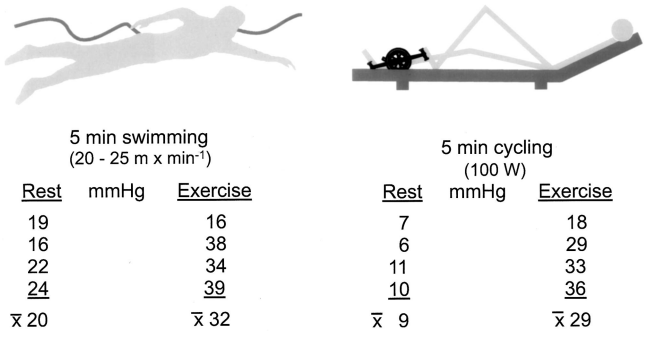

FIGURE 2—Mean pulmonary capillary pressure (PCPm; mm Hg) at rest and during 5 min of low-speed swimming compared with 5 min of supine cycling at a 100-W load in four patients with severe myocardial infarction (group B). Pathologic values of PCPm in supine body position: 13 mm Hg (rest), 23 mm Hg (exercise) (3).

DISCUSSION

Although exercise training is established as an integral part of treatment regimes in both patients with severe MI and CHF, there is no consensus yet on the appropriateness of water exercises and swimming. The results of our explorative studies provide insight concerning central hemodynamic pressure and output responses to water immersion and swimming in patients with severe MI and in compensated severe CHF, with confirmation of the study hypothesis. The major findings are: a) an increase in preload in patients with MI; b) left ventricular overload and decrease in stroke volume in patients with severe CHF; and c) that patient symptoms did not correlate with hemodynamic findings.

Increase in preload in patients with MI. As assessed in patients with moderate and/or severe MI, graded immersion to the xiphoid does not affect PAm (Fig. 1), but on upright immersion up to the neck (Fig. 1) and during the supine resting position in the water (Fig. 2), the hydrostatic pressure on the body surface results in pathologic PAm and/or PCPm, respectively. Because of a parallel increase of PAm and PCPm seen in the patients, and because no patient exhibited a primary pulmonary hypertension, a reflected increase in the resistance of pulmonary vessels during immersion could be excluded. These findings indicate that even under resting conditions, the immersion-induced blood volume shift could result in a marked increase in preload.

For both swimming and supine cycling, the PAm and PCPm, respectively, reached pathologic values, which were slightly higher for swimming (pathologic values of PAm in supine body position: 22 mm Hg (rest), 34 mm Hg (exercise); (3)). Related to baseline pressure with immersion in a

TABLE 2. Left ventricular diameter and stroke volume (mean and standard deviation; range) obtained by subxiphoidal echocardiography in both patients with moderate and severe CHF and in healthy subjects in upright position out of water and immersed neck-deep.

	Moderate Chronic CHF (N = 18) Group C		Severe Chronic CHF (N = 5) Group D		Healthy Subjects (N = 10) Group E	
	Out of Water	Neck-Deep Immersion	Out of Water	Neck-Deep Immersion[a]	Out of Water	Neck-Deep Immersion
LV SD (mm)						
Mean	27.4	33.8**	54	58	26.8	32.3**
SD	6.1	8.2	—	—	5.2	5.3
Range	20–37	25–50	53–62	55–65	20–34	24–26
LV DD (mm)						
Mean	44.3	52.6**	65	68	42.1	50.6**
SD	6.6	7.6	—	—	6.6	7.6
Range	32–54	37–72	61–73	63–75	37–55	40–59
SV (mL)						
Mean	61.3	86.5**	73	70	54.2	81.1**
SD	19.4	24.7	—	—	21.4	19.1
Range	29–95	36–154	52–87	50–83	28–98	53–110
		↑ N = 10; ↔ N = 1; ↓ N = 1		↓ N = 5		↑ N = 10

[a] No statistical comparison was performed.
** $P < 0.001$, changes between out of water vs neck-deep immersion; ↑, No. of patients with increase of SV; ↔, No. of patients with no change in SV; ↓, No. of patients with decrease of SV; CHF, chronic heart failure; LV, left ventricle; SD, systolic cross-sectional diameter; DD, diastolic cross-sectional diameter; SV, stroke volume.

supine position, the increase of PAm and PCPm, respectively, was relatively small during slow swimming, compared with the pressure increase measured during supine cycling at 100 W outside of water (Fig. 2). These findings emphasize the impact of an immersion-induced preload on the increase of left ventricular wall stress.

Left ventricular overload and decrease in stroke volume in CHF. According to current knowledge, abnormal systolic function after myocardial infarction may result in an increased left ventricular end-systolic volume and greater dependence on the Starling mechanism to maintain cardiac output during land exercise. The results of our echocardiography studies on left ventricle diameter and stroke volume in severe CHF patients (group D) (Table 2; Fig. 3; Fig. 4b), compared with patients with moderate CHF and healthy subjects (groups C, E, and F) (Table 2; Fig. 4a), suggest that because of the enhanced preload induced by immersion, the myocardial compliance of the left ventricle could be compromised (10). In other words, according to the Starling law, the preload increase could have shifted the working point of the resting-volume curve far to the right, thus exceeding the crucial point; in consequence, left ventricular overload occurred, and the stroke volume decreased. Because this phenomenon can be interpreted in terms of an overdistension of the A-I-filaments of the myocardial fibers, it entails the risk of further dilatation of a damaged myocardium.

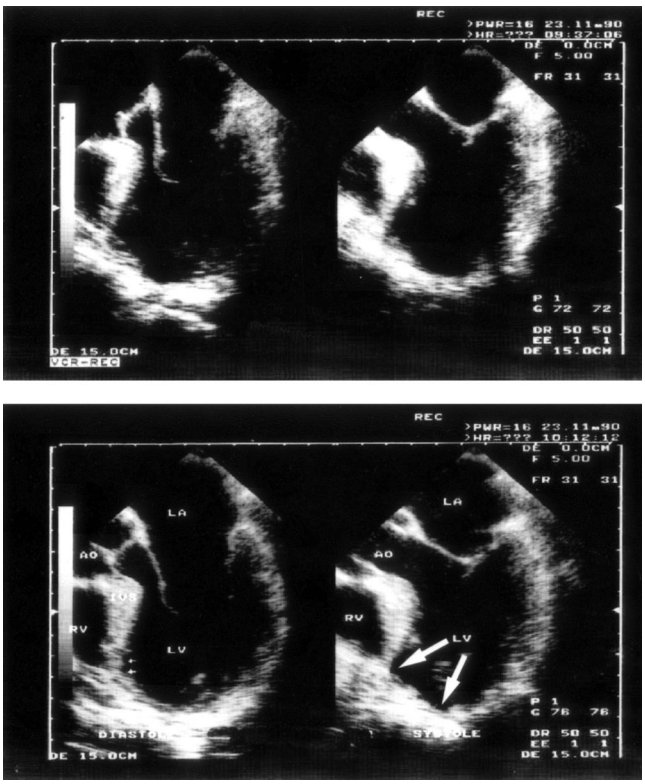

FIGURE 3—Case study: echocardiography imaging of the left ventricle of a patient with severe CHF who suffered from anterior apical MI. The top of the picture shows diastolic and systolic left ventricular measurements obtained from a patient standing out of water; the lower part was measured during immersion up to the neck. The left part indicates end-diastolic and the right part end-systolic phase of contraction.

Patient's well-being is maintained in spite of hemodynamic deterioration. A smaller decrease in mixed venous oxygen saturation was observed during swimming than during cycling. This phenomenon could be caused by a reflex-induced reduced cutaneous blood flow (because of a blunted reflex control of subcutaneous vascular beds (7)), and/or could reflect an enhancement of cardiac output caused by increased preload induced by immersion. A relatively small reduction of mixed venous oxygen saturation might explain why patients felt well in the water even though exhibiting pathologic PAm values. Although swimming even at a slow or moderate speed could cause relatively high central hemodynamic stress, the patients' feelings of well-being deserve attention. In this context, when swimming and cycle ergometry were compared in patients with mild and/or moderate left ventricular dysfunction, the following equivalents were found: with slow swimming (20 $m \cdot min^{-1}$), heart rate, blood lactate, and plasma catecholamines corresponded to values obtained with supine cycling at a 100-W load. Likewise, swimming at 27 $m \cdot min^{-1}$ corresponded to 150 W and 30 $m \cdot min^{-1}$ to 170 W, on average. With these swimming speeds all patients reported a feeling of well-being (13).

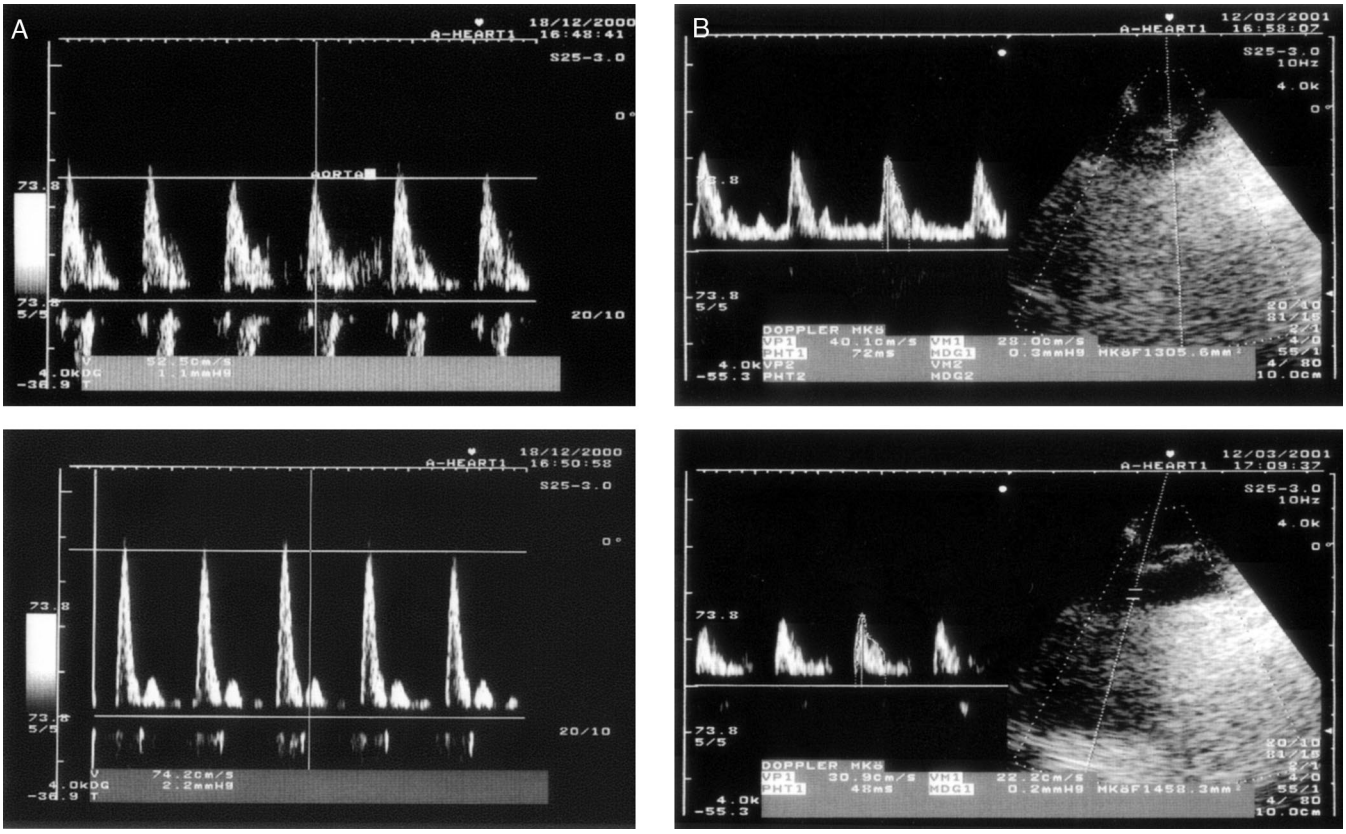

FIGURE 4—Case study: pw-Doppler imaging (brachiocephalic trunk) of a healthy subject (a) and a patient with severe systolic heart failure (b) in upright position outside of water (top part) and on immersion up to the neck (bottom part). In the healthy subject with immersion, the peak velocity increases from 49 to 76 cm·s^{-1}, indicating an increase of stroke volume. In the CHF patient with immersion, the peak velocity diminishes from 40 to 31 cm·s^{-1}, indicating a decrease of the stroke volume.

Study limitations. Our exploratory studies were performed on small samples of patients exhibiting different etiologies and severities of left ventricular dysfunction, and medication. Thus, results do not claim to be representative of all, but provide new information on central hemodynamic responses during immersion and swimming that have to be elaborated in extended studies involving larger samples. These samples should allow an assessment of subgroups of patients with different etiologies and severities of CHF, and how these groups compensate for immersion-induced preload enhancement. Because in compensated CHF patients on immersion up to the xiphoid, release of norepinephrine, renin, and AVP were reported to be suppressed similarly to other types of patients and healthy normals (6), it may be asked whether and/or how neurohumoral regulation of an immersion-induced central volume loading operates in patients under different therapy regimes and in patients with an impaired baroreflex sensitivity (5).

Clinical suggestions and future research need. Additionally, our studies focus on acute responses during immersion and swimming. The explorative studies showed that in patients with severe MI and severe CHF, neck-deep immersion produced abnormal cardiac responses in a temporal manner. These observations do not provide any evidence that repeated immersion, exercises in the water, and swimming would lead to abnormal remodeling of the left ventricle. Nevertheless, from the findings of pathologic PAm and stroke volume decrease in patients with severe MI and severe CHF, respectively, the following preliminary suggestions could be postulated:

1) The patient's feeling of well-being is maintained in spite of hemodynamic deterioration, suggesting that such impressions in the water are no guarantee that the left ventricle tolerates the marked volume loading caused by immersion.

2) Decompensated heart failure is an absolute contraindication for immersion and swimming.

3) Patients with severe MI and/or CHF who can sleep in a flat position can bathe in a tub (e.g., balneotherapeutic baths), taking a half-sitting position (i.e., immersed no deeper than up to the xiphoid).

4) Therapeutic water exercises in a pool (e.g., for orthopedic reasons) can be allowed for patients with severe MI and severe CHF, provided that a patient is in an upright position, and immersed no deeper than up to the xiphoid.

Whether swimming is appropriate (and truly safe in the long term) for patients with severe MI and severe CHF needs to be proven in patients who undergo a swimming program and are compared with nonswimming patients with CHF of similar etiology and severity of disease.

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