Chronic heart failure

Left ventricular dysfunction and chronic heart failure: should aqua therapy and swimming be allowed?

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Aqua therapy for patients with chronic heart failure may be dangerous

Aqua exercise and swimming are traditionally recommended for low risk cardiac patients. In patients with severe myocardial infarction (MI) and compensated severe chronic heart failure (CHF), physicians may have reservations about water exercises and swimming. Concerns become understandable when central haemodynamic responses in healthy subjects during immersion and swimming are considered.

During neck deep water immersion, a 100 cm column of water exerts a pressure of 76 mm Hg on the body surface. During swimming, a pressure of 40–60 mm Hg can be assumed. This pressure compresses 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, but, on immersion up to the neck, the central blood volume has been shown to be increased by about 700 ml; 180–240 ml of this was allotted to the heart volume, with enlargement of all four chambers.¹ Planimetry of the diastolic poster-anterior area of the heart has shown an average increase in heart size of 30% within six seconds.² From a central haemodynamic point of view, crucial immersion starts at the diaphragm/xiphoid level. At this level, both the buoyancy effect³ and the external hydrostatic pressure result in a blood volume shift, which (a) corresponds to up to 85% of the total shift during neck-deep immersion and (b) amounts to a volume shift occurring when moving from an upright to supine body position out of water.¹

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 have been shown to increase by up to 15–20 mm Hg.^{1,4} As the intrathoracic vascular compliance tends to remain stable under a variety of

conditions, the increase in central venous pressure would reflect an equivalent increase in central blood volume.⁵ As a consequence, the left ventricular end diastolic volume, which is considered to be an indicator of myocardial fibre length, rises. In a single case, left end diastolic ventricular volume increased by 40–70 ml.¹ Along with the preload enhancement, due to the Starling mechanism, the stroke volume increases by 35–45%.⁶ Both the arterial systolic and mean blood pressure have been shown to be unchanged⁶ and/or slightly increased⁴ with immersion.

How do patients who suffered a prior MI (6–10 weeks) and exhibiting marked hypokinesias in the infarction area respond to upright graded immersion? Immersion to the xiphoid results in a mean pulmonary artery pressure (PAm) in the normal range of values. On upright immersion up to the neck, PAm increases to abnormal values—that is, 53 (13) mm Hg on average—which correspond to PAm values obtained outside of water for the supine position.⁷ These findings indicate that, even under resting conditions, the immersion induced blood volume shift may result in a considerable increase in preload.

When five minutes of slow swimming (20–25 m/min) perceived as comfortable was compared with supine cycle ergometry at a 100 W load, heart rate was similar for both exercises. During swimming, PAm exceeded the pressure values generated during cycling in all patients,⁸ which emphasises the impact of an immersion induced preload on the increase in left ventricular wall stress. In two patients, the PAm exceeded 60 mm Hg; however, both reported that they felt fine.

What about left ventricular output response in the patients? Recently it was reported that, in upright immersed patients with moderate CHF, both the diastolic and systolic cross sectional diameters were shown to be greater

than when out of water, and accompanied by a significantly increased stroke volume. However, in patients with severe CHF 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 did not change, or decreased.⁸ The same authors used echocardiographically measured peak blood flow velocities from the ascending aorta to determine stroke volume in patients with severe CHF after moving from out of the water to immersion up to neck. All patients exhibited a left ventricular end diastolic diameter of more than 60 mm. During immersion, pulsed wave-mean flow velocity decreased by 7% on average (range 5–10%) implying a decrease in stroke volume. These findings suggest that, because of the enhanced preload induced by immersion, the myocardial compliance of the left ventricle may be compromised.⁹ In other words, according to Starling's law, the preload increase may have shifted the working point of the resting volume curve far to the right, thus exceeding the crucial point and, in consequence, left ventricular overload occurs and the stroke volume decreases. As 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.

Despite the relatively high central haemodynamic stress and in spite of haemodynamic deterioration during immersion and slow swimming, the patient's feeling of wellbeing is maintained.⁸ One reason may be a smaller decrease in mixed venous oxygen saturation observed during water exercise than during cycling outside of water.⁸ This phenomenon may be due to a reflex induced reduced cutaneous blood flow (because of a blunted reflex control of subcutaneous vascular beds¹⁰), and/or may reflect an enhancement of cardiac output due to increased preload induced by immersion and thus may explain why patients felt well in the water even though exhibiting pathological PAm values. At any rate, the above observation suggests that feeling well in the water is no guarantee that the left ventricle tolerates the considerable volume loading caused by immersion.

What can we learn from these findings? According to current available research, patients with severe MI and severe CHF immersed up to the neck may produce abnormal cardiac responses in a temporal manner. This finding does not provide any evidence that repeated immersion, exercise in the

water, and swimming lead to abnormal remodelling of the left ventricle, but it does indicate that care should be taken when prescribing aqua exercises and swimming.

- (1) Decompensated heart failure is an absolute contraindication to immersion and swimming.
- (2) Patients with severe MI and/or CHF who tolerate sleeping in a flat position can bathe in a tub—that is, balneotherapeutic baths—in a half sitting position—that is, immersed no deeper than up to the xiphoid.
- (3) Therapeutic water exercises in a pool—for example, for orthopaedic reasons—can be allowed for patients with severe MI and severe CHF provided that they are in an upright position and immersed no deeper than the xiphoid.
- (4) Whether swimming is appropriate, and truly safe in the long term, in

patients with severe MI and severe CHF needs to be investigated in a study of patients in a swimming programme and compared with non-swimming patients with CHF of similar aetiology and severity of disease.

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