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Is Swimming Safe in Heart Failure? A Systematic Review

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ABSTRACT

It is not clear whether swimming is safe in patients with chronic heart failure. Ten studies examining the hemodynamic effects of acute water immersion (WI) (155 patients; average age 60 years; 86% male; mean left ventricular ejection fraction (LVEF) 29%) and 6 randomized controlled trials of rehabilitation comparing swimming with either medical treatment only (n=3) or cycling (n=1) or aerobic exercise (n=2), (136 patients, average age 59 years; 84% male, mean LVEF 31%) were considered. In 7 studies of warm WI (30 -35 °C): heart rate (HR) fell (2-15%), and both cardiac output (CO) (7-37%) and stroke volume (SV) increased (13-41%). In 1 study of hot WI (41°C), systemic vascular resistance (SVR) fell (41%) and HR increased (33%). In 2 studies of cold WI (12-22 °C), there were no consistent effects on HR and CO. Compared with medical management, swimming led to a greater increase in peak VO_2 (7-14%) and 6 minute walk test (6 MWT) (7-13%). Compared with cycle training, combined swimming and cycle training led to a greater reduction in resting HR (16%), a greater increase in resting SV (23%) and SVR (15%), but no changes in resting CO and a lesser increase in peak VO_2 (6%). Compared with aerobic training, combined swimming and aerobic training lead to a reduction in resting HR (19%) and SVR (54%) and a greater increase in SV (34%), resting CO (28%), LVEF (9%) and 6MWT (70%). Although swimming appears to be safe, the studies conducted have been small, very heterogeneous and inconclusive.

Key words: heart failure, water immersion, swimming

All current guidelines recommend regular exercise as an integral part of the management of patients with chronic heart failure (CHF).¹⁻⁴ The sorts of exercise that have been shown to be helpful include aerobic exercise on a treadmill or a cycle at 3-5 times per week at a heart rate of 60-70% of heart rate reserve; and resistance training of peripheral muscles and calisthenics.⁵⁻⁷ In two meta-analyses, exercise training was associated with a reduction in mortality and hospital admissions, as well as improvements in maximum oxygen consumption (peak VO₂), walking distance and quality of life compared to standard medical care.^{6,7} A subsequent trial was conducted of more than 2000 patients randomized to supervised exercise training followed by unsupervised exercise training at home (3 sessions per week for 30-35 mins at 70% of heart rate reserve, total of 36 sessions) or usual care. Exercise was associated with an absolute risk reduction of 4% in death or hospitalization from any cause at 3 years.⁵

However, whether swimming is safe or beneficial is not clear. Most guidelines steer clear of discussing swimming. Only the Scottish guidelines mention swimming in order to warn against it in patients with New York Heart Association (NYHA) class III and IV symptoms.³ Potentially, the pressure exerted by water immersion (WI) can result in the shift of extracellular fluid and peripheral blood back into the venous circulation. This would, in turn, increase venous return to the failing heart and precipitate pulmonary oedema in susceptible patients.

Patients with many different conditions do swim regularly, especially patients with musculoskeletal conditions.⁸ Whether it is an appropriate treatment for people with heart failure is not clear: therefore, we conducted a systematic review on the effects of WI and swimming in patients with CHF.

METHODS

Search Strategy and Study Selection

We systematically searched for publications on swimming and heart failure on Pub Med until January 2016. The words “heart failure”, “ventricular dysfunction” or “cardiomyopathy” in combination with “swimming”, “water immersion”, “hydrotherapy”, “aquatic exercise”, “water exercise” and “water based exercise therapy” were used as search criteria. Randomized controlled trials, controlled trials, and single arm studies were included.

Outcome Measures

Hemodynamic variables measured during WI to the neck and swimming rehabilitation were: heart rate (HR), stroke volume (SV), cardiac output (CO), systemic vascular resistance (SVR), peak VO_2 , left ventricular end diastolic diameter (LVEDD), left ventricular end systolic diameter (LVESD) and left ventricular ejection fraction (LVEF). Another two outcomes reported for swimming rehabilitation were: quality of life (QOL) and 6 minute walk test (MWT) distance.

RESULTS

We identified 41 publications in English. Studies on animals (n=10), in conditions other than heart failure (n=4), reviews, case reports or letters to editors (n=12) were excluded. Fifteen eligible publications were thus considered.(Figure 1)

Acute Hemodynamic Effects of Water Immersion

There were 10 studies examining the acute hemodynamic effects of WI, with a total of 155 patients (average age 60 years; 86% male; mean LVEF 29%).⁹⁻¹⁸ Seven studies were in warm water (32-35°C) with a total of 97 patients (average age of 61

years; 85% male; mean LVEF 30%). Four of these 7 studies compared the responses of patients with normal subjects, and the remaining 3 were in patients alone.⁹⁻¹⁵ Two studies were in cold water (12-22°C) with a total of 24 patients (average age of 60 years; mean LVEF 31%).^{17,18} One study compared the responses of patients with normal subjects and 1 study was in patients alone. There was a single study of hot water immersion (41°C) with 34 patients (average age 58 years; 76% male; mean LVEF 25%).¹⁶ (Figure 2)

Warm Water Immersion

In the 7 studies which compared haemodynamics at baseline to warm WI: 6 studies measured HR, which was reduced by a mean of 7% (range: 2 to - 15%).¹⁰⁻¹⁵ Five studies measured SV, which was increased in most patients by a mean of 32% (range: 13 to 41%),^{9,11,13-15} apart from 1 study of a sub-population of patients with severe heart failure in whom SV decreased by 4%.⁹ Five studies measured CO, which increased by a mean of 22% (range 7 to 37%).¹¹⁻¹⁵ Three studies measured SVR, which decreased by a mean of 9% (range 3 to 21%).^{11,14,15} (Figure 2)

In 3 studies which compared echocardiographic changes at baseline to warm WI: 2 studies measured LVED volume (LVEDV), which increased by a mean of 16% (range 6 to 24%) and LVEF, which increased by a mean of 13% (range 12 to 13%).^{11,13} One study showed a greater increase in LVEDD in patients with moderate heart failure (19%) compared to severe heart failure (5%).⁹ (Figure 2)

Four studies examined the change in haemodynamics from baseline during warm WI in patients with CHF compared with healthy subjects: the changes were of similar magnitude in both groups. In 4 studies, HR decreased in both groups by a

mean of 7% (range 2 to -15%) in CHF, and by a mean of 11% (range -1 to -22) in healthy subjects.^{10,12-14} In 2 studies, SV increased in both groups by a mean of 28% (range 17 to 37%) in CHF and by a mean of 50% (range 30 to 65%) in healthy subjects.^{13,14} In 3 studies, CO increased in both groups by a mean of 20% (range 14 to 37%) in CHF and by a mean of 26% (range 19 to 31%) in healthy subjects.¹²⁻¹⁴ In 1 study, SVR decreased in both groups by a mean of 21% in CHF and by a mean of 28% in healthy individuals.¹⁴ (Figure 2)

Hot Water Immersion:

In the single study of hot WI, HR increased (33%), SV increased (16%), CO increased (50%) and SVR decreased (41%).¹⁶ On echocardiography, LVEDV and LVESD both decreased (2.4% and 5.4% respectively), but LVEF increased (23%).¹⁶ (Figure 2)

Cold Water Immersion

In the 2 studies which compared haemodynamics at baseline to cold WI: both studies measured CO which increased, both measured HR with inconsistent results and 1 study measured SVR, which fell.^{17,18} (Figure 2)

Swimming as a Form of Rehabilitation in Patients with Heart Failure

There were 6 trials of exercise training with patients. Three studies randomized patients to either swimming or continued medical therapy (67 patients, mean age of 70, 73% male, mean LVEF of 32%).^{11,19,20} Two studies randomized patients to either a combination of swimming and aerobic exercise or aerobic training only (45 patients, mean age of 60, mean LVEF of 31%).^{21,22} One study randomized patients to either gymnastics on land or in water in combination with cycle training in

both groups (24 patients, mean age of 54, mean LVEF 30%).²³ All patients had to be clinically stable (mostly for at least 3 months) prior to inclusion. (**Table 1, Figure 1**)

Swimming Training Compared to Medical Management Only:

Three studies compared swimming training with medical management only.^{11,19,20} At the end of follow up (8 weeks), there were no changes in HR, SV, CO or SVR at rest in 1 study.¹¹ In 2 studies, there was an increase in peak VO₂ by a mean 10% (range: 7 to 14%) in swimming training vs. a reduction by a mean -10% (range -12 to -7% in medical management), while there was no change in peak VO₂ in the third study.^{11, 19,20} In 2 studies, there was a greater increase in 6 MWT with swimming, by a mean of 10% (range: 7 to 13%) in swimming training vs. increase by a mean of 1% (range 0 to 2%) in medical management.^{19,20} There was no consistent pattern in QoL in the 2 studies where it was measured.^{19,20} (Table 1)

Cycle Training Compared to a Combination of Swimming and Cycling Training

In a study which compared haemodynamics and peak VO₂ between cycle training only or a combination of swimming and cycle training, there was a greater reduction in resting HR (-16% swimming and cycling vs. -8% cycling only), greater increase in resting SV (23% swimming and cycling vs. 10% cycling only), no change in CO at rest (0% swimming and cycling vs. 7% cycling only), an increase in resting SVR (15% swimming and cycling vs. -15% cycling only) and an increased peak VO₂ (6% swimming and cycling vs. 11% cycling).²³ (Table 1)

Aerobic Training Compared to a Combination of Swimming and Aerobic Training

In 2 studies which compared haemodynamics between aerobic exercise training only or a combination of swimming and aerobic exercise, at the end of follow

up (range 3-24 weeks) there was a greater reduction HR (-19% swimming and aerobic exercise vs. +5% aerobic exercise only) in 1 study, but no difference in the other; a greater increase in SV (34% swimming and aerobic exercise vs. 15% aerobic exercise only) in 1 study, but no difference in the other.^{21, 22} In one study there was a greater increased CO at rest (28% swimming and aerobic exercise vs. 9% aerobic exercise only) and a greater reduction in SVR (54% swimming and aerobic exercise vs. 19% aerobic exercise only).²² (Table 1)

The echocardiographic findings were: LVEDD was reduced in 1 study (3% swimming and aerobic exercise vs. 5% aerobic exercise only), but no difference was seen in the second; LVESD was reduced (9% swimming and aerobic exercise vs. 2% aerobic exercise only) in one study, with no change seen in the second; a similar increase in resting LVEF was seen in both studies (9% swimming and aerobic exercise vs. 6% aerobic exercise).^{21,22} (Table 1)

One study reported 6 MWT distance, and found that there was a greater increase following swimming and aerobic exercise (70%) than following aerobic exercise alone (32%).²² (Table 1)

DISCUSSION

We have found that WI to the neck is well tolerated in stable patients with CHF, particularly when the water is warm. Both cold and hot WI caused adverse hemodynamic effects, whereas warm WI appeared helpful. The most consistent effects were a fall in resting HR and SVR. Exercise in water has effects similar to other forms of exercise training, with a similar improvement in exercise capacity.

Exercise in water in stable patients with NYHA class II-III symptoms was well tolerated with no adverse effects.

The mechanical properties of water are considerably different to air; there are several changes in haemodynamics and fluid shift even when healthy subjects are immersed in water. During WI to the neck, approximately 700 ml of peripheral blood pools centrally (of which 180-240 ml accumulates in the heart) due to the hydrostatic pressure exerted on the body.^{24,25} As a consequence, in healthy subjects, there is an increase in LV volume and an increase in LVEF, resulting in increased SV and blood pressure.^{26,27} In healthy subjects, cycling in water leads to a greater increase in CO than cycling in air by an average of 0.7 l/min.²⁸ SV increases by 49% in air compared to 34% in water.²⁷

Guidelines for management of patients with heart failure are cautious about WI and swimming in patients with heart failure because of the potential to precipitate pulmonary oedema secondary to the mobilization of peripheral fluid into the central circulation. Reports of swimming-induced pulmonary oedema (SIPE) are rare, and confined to athletes usually undertaking severe exercise. The pathophysiology of SIPE is not well understood, but factors such as the central shift of fluid during WI, vasoconstriction due to cold water, and over hydration may increase right-sided cardiac pressures leading to pulmonary oedema.^{29,30}

In a study by Weiler-Ravell and colleagues on 30 young men in a military fitness program,³¹ swimming in water at 23°C caused pronounced shortness of breath in 8 subjects. Five subjects stopped swimming early, requiring oxygen supplementation, and in the more severe cases, intravenous diuretics. Subjects with a history of SIPE have higher pulmonary artery pressure (previous SIPE = 34 mmHg vs

control = 22 mmHg, $p=0.004$) and pulmonary artery wedge pressure (previous SIPE = 19 mmHg vs control = 11 mmHg, $p=0.028$) when exercised in cold water on a cycle than those who do not.³¹

None of the studies of WI or swimming rehabilitation in patients with heart failure reported SIPE. However, all the studies we found were small, involving at most 34 patients. The majority of patients were male and were young compared to the general population of patients with heart failure. Most of the studies which looked at swimming rehabilitation did not prescribe swimming on its own, but in conjunction with aerobic exercise on land, therefore it is difficult to assess the effects of swimming training alone on cardiac function and exercise performance. ***The patients all had to be “stable” to be included in the studies, but other than meeting a minimum time for stability, no further information is available.***

What is perhaps surprising is how few patients have been included in studies of swimming. In the United Kingdom, swimming is the most popular sport with over 3 million people taking part in at least a session per week. This is a million more people per week than play football (which is often considered to be the UK's national sport).³⁰ Swimming may be more popular than other forms of exercise because it is a low impact activity suitable for people with disability, poor mobility or frailty.²⁷ Given how common swimming is, it must be the case that patients with heart failure are swimming regularly. The data we have found (and perhaps the lack of reports in the literature) suggest that swimming is probably safe in patients with heart failure. Given that patients with heart failure are often frail and have other co-morbidities (which reduce mobility), swimming is perhaps the ideal way to encourage exercise without the difficulty of weight-bearing exercise on land. However, much larger

studies are needed to assess the safety and potential benefits of swimming compared to conventional heart failure rehabilitation.

CONCLUSION

Although exercise in water appears to be safe, the studies conducted have been small, very heterogeneous and inconclusive. Further investigation is required to establish the effects of WI and swimming, so as to be able give accurate advice to patients with heart failure.

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Figure 1: Breakdown of eligible studies. WI: water immersion

Figure 2: Percentage change in hemodynamic, respiratory and echocardiographic findings from rest to water immersion. VO₂: Maximum volume of oxygen; LV: Left ventricular

| Study | Pts | Age yrs | LVEF % | Sex % male | NYH A class | Stability | Swimming/ control | Percentage change from baseline to end of rehabilitation / % | | | | | | | | |
|------------------------|-----|------------|-----------|---------------|-------------------|-----------|-----------------------|--|-----------|-----------|-----------|-----------------|-----------|-----------|------|------|
| | | | | | | | | HR | SV | CO | SVR | VO ₂ | LVEDD | LVESD | LVEF | 6MWT |
| Cider ²⁰ | 24 | 67 | 34 | 80 | II-III | 3 months | Swimming | | | | | ↑ 14 | | | | ↑13 |
| | | | | | | | Medical management | | | | | ↓7 | | | | 0 |
| Cider ¹⁹ | 25 | 72 | 31 | 68 | II – III | 3 months | Swimming | | | | | ↑7 | | | | ↑7 |
| | | | | | | | Medical management | | | | | ↓12 | | | | ↑2 |
| Svealv ¹¹ | 18 | 69 | 31 | 72 | II – III | 2 months | Swimming | No change | No change | | No change | No change | ↑ | | | |
| | | | | | | | Medical management | No change | No change | | No change | No change | | | | |
| Teffaha ²¹ | 24 | 53 | 30 | 100% | II - III | 2 weeks | Swimming and aerobics | ↓ | ↑ | | | | No change | No change | ↑ | |
| | | | | | | | Aerobics | ↓ | ↑ | | | | No change | No change | ↑ | |
| Caminiti ²² | 21 | 68 | 32 | N/A | II - III | 3 months | Swimming and aerobics | ↓19 | ↑34 | ↑28 | ↓54 | | ↓3 | ↓9 | ↑9 | ↑70 |
| | | | | | | | Aerobics | ↑5 | ↑15 | ↑9 | ↓19 | | ↓5 | ↓2 | ↑6 | ↑32 |
| Laurent ²³ | 24 | 54 | 30 | 100% | N/A | 3 weeks | Swimming and cycling | ↓16 | ↑23 | No change | ↑15 | ↑6 | | | | |
| | | | | | | | Cycling | ↓8 | ↑10 | ↑7 | ↓15 | ↑11 | | | | |

Table 1: Randomized Controlled Trials of Rehabilitation Comparing Swimming with Either Medical Treatment Only or Cycling or Aerobic Exercise. Pts: patients, yrs: years, LVEF: left ventricular ejection fraction, NYHA: New York Heart Association, HR: heart rate, SV: stroke volume, CO: cardiac output, SVR: systemic vascular resistance, VO₂: Maximum volume of oxygen, LVEDD: left ventricular end diastolic diameter, LVESD: left ventricular end systolic diameter, 6MWT: 6 minute walk test. N/A: not available.

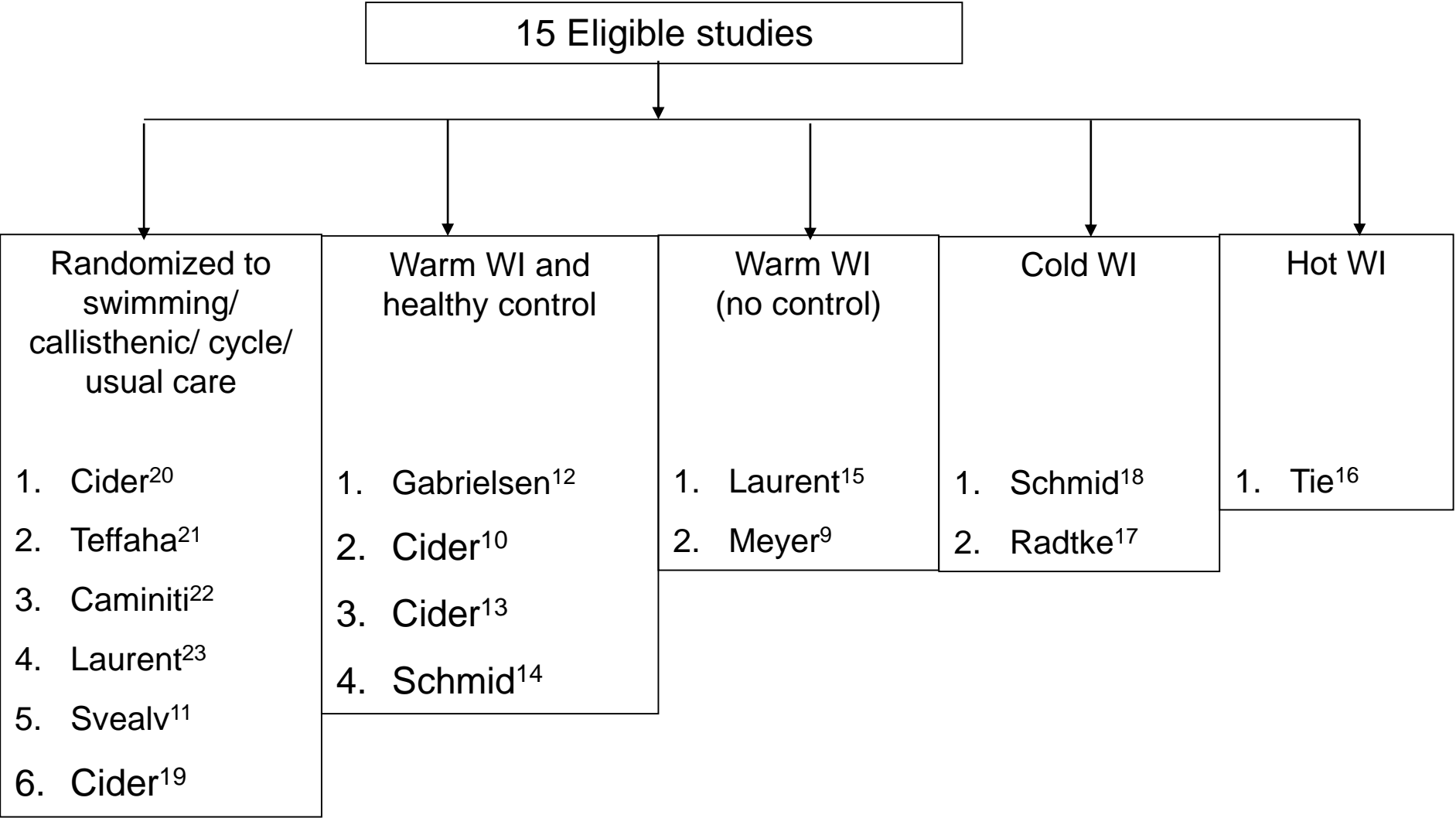


Figure 1

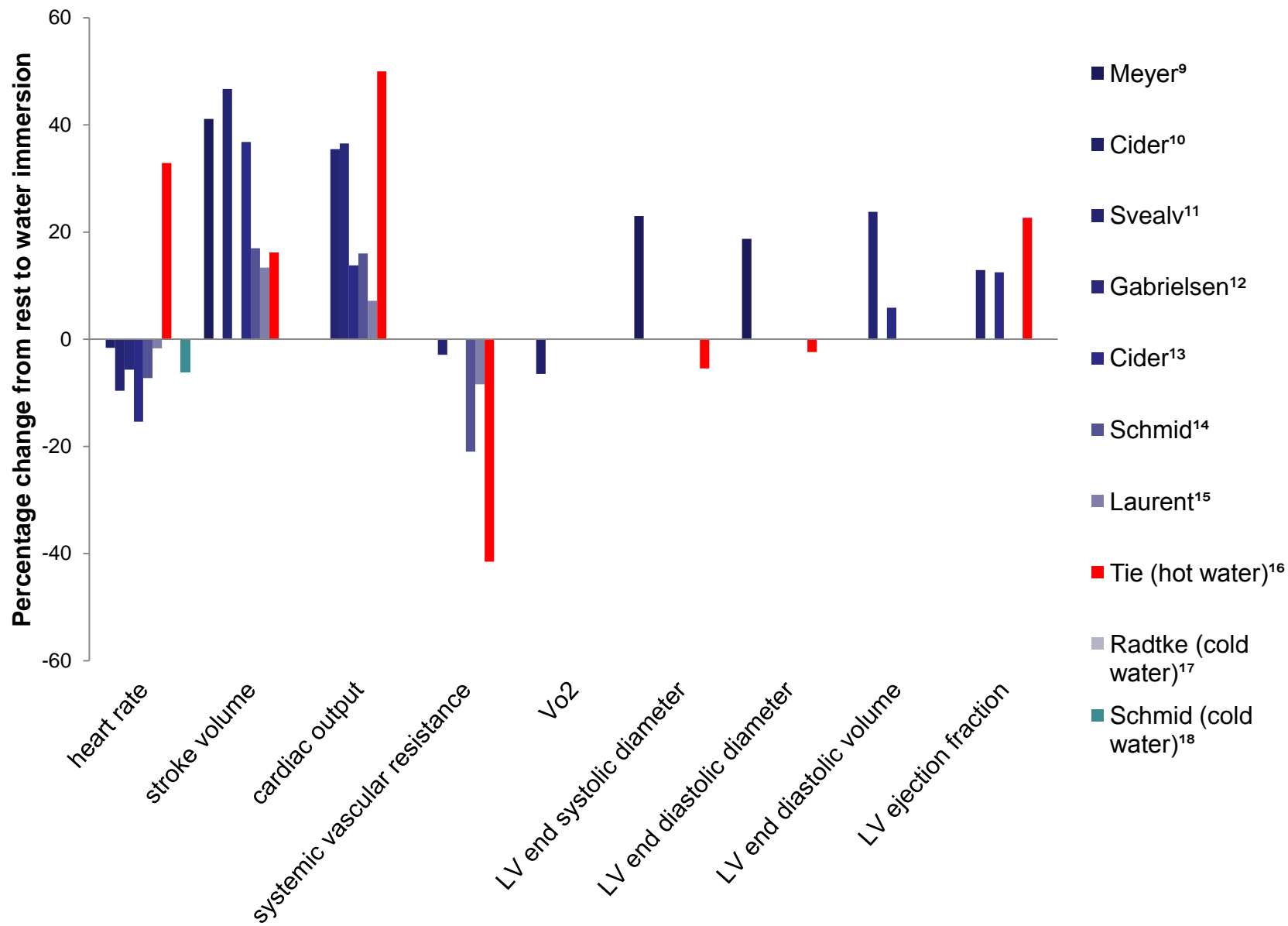


Figure 2