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Letter to the Editor

It is likely that the drinking of cold and room temperature water decreases cardiac workload

To the Editor: Recently, we have provided indirect evidence for a decreased cardiac workload in young and healthy subjects in response to ingestion of 500 mL cold (3 °C)- or room (22 °C)-tempered water, but not to body-tempered (37 °C) water. This conclusion was based on the observation of a reduced heart rate and a decreased rate-pressure double product, a marker for myocardial oxygen consumption. Our view has been challenged by the letter to editor written by Dr. McMullen on the basis that ingestion of water is expected to elicit an enhanced sympathetic drive to the heart and thereby increasing cardiac contraction force, which would in turn lead to an increased cardiac workload. However, a careful analysis of our data does not support this argument.

Dr. McMullen challenges our observation and raises two issues about our recent publication (Girona *et al.* 2014). *Firstly*, Dr. McMullen points out that we have not measured the rate of blood pressure changes (d*P*/d*t*). He claims that a possible increase in d*P*/d*t*, elicited by a water-induced enhanced sympathetic drive to the heart, would increase stroke volume and thus blood pressure, which in turn would load baroreceptors to decrease heart rate. This increased d*P*/d*t* would then increase cardiac workload. *Secondly*, Dr. McMullen claims that we had no control condition and raises the issue of stationarity, thereby questioning the reliability of our measurements after the first 30 min post-ingestion.

Before rebutting these two issues, we would like to point out that in the study by McMullen *et al.* (2014) cardiac contraction force was estimated from d*P*/d*t* measured at the finger level via infrared plethysmography, which is not the best method to evaluate cardiac contraction force (Sharman *et al.* 2007). The assessment of cardiac contractility even by measuring ventricular d*P*/d*t* with a high-fidelity catheter should be considered with some caution as it is influenced by preload (Carabello 2002).

In addition, there are many differences between our study (Girona *et al.* 2014) and that by McMullen *et al.* (2014) besides subjects' age (23 vs. 43 years on average) and amount of water ingested (500 vs. 100 mL). Firstly, their study subjects abstained from food and drink, excepting water, for only 2 h before the experimental session, a situation which could add variability in the baseline values. In contrast, our subjects came to the lab after an overnight fasting to ensure standardized conditions. Secondly and most importantly for baseline determination, McMullen's participants '... moved to a sitting position and a 120 s pre-ingestion period was followed by ... the presentation and ingestion of the interventions. ...' (McMullen *et al.* 2014). In contrast, in our study, we had no posture change and we waited 20–30 min after instrumentation before starting a 30 min baseline to ensure stable cardiovascular and metabolic parameters.

As we did not measure d*P*/d*t*, one can merely speculate on a possible augmented d*P*/d*t* in response to drinking water. However, a risen d*P*/d*t* value does not necessarily reflect cardiac sympathetic activation as it is influenced by many other variables (Carabello 2002). An increased preload due to a longer filling time could also increase cardiac contraction force through distension of sarcomeres and contribute to the increased stroke volume. Moreover, our changes in stroke volume were rather small (+5–6% in the peak response) and less pronounced than the changes in heart rate, which explains the trend for a lower cardiac output. We would like to stress that cardiac workload cannot be derived from an averaged single beat (based on an increased stroke volume or an increased d*P*/d*t*), but we should consider instead the total cardiac workload over 1 min. A marked decrease in heart rate would then offset the increased workload of a single beat. The observed decrease in double product, which correlates with the oxygen consumption of the myocardium (Van Vliet & Montani 1999), is thus consistent with a diminished cardiac workload.

We acknowledge that we did not include a no-drink control but the purpose of our study was to compare the responses across water drinks at different temperatures using a randomized cross-over design, which provides sufficient statistical power to draw concrete conclusions. Dr. McMullen also raises the issue of parameter stationarity after 30 min post-drink. Our results were presented as changes from a 30-min stable baseline with baseline values set to 0, explaining the low standard error of the mean (SEM) during the baseline period. An increased SEM after an intervention is expected due to the dynamic of the post-drink situation and that not all study subjects are expected to respond identically, but rather with individual variability in the

response both in amplitude and in time to peak. Furthermore, the decreases in heart rate and double product are evident within the first 15–30 min. We can therefore conclude that it is likely that the drinking of cold and room temperature water decreases cardiac workload in healthy young subjects.

Conflict of interest

None.

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