Letters to the Editor

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Impaired left ventricular function in obesity?

We read with interest the findings of Sasso et al.\(^1\) regarding the effects of insulin on left ventricular function during exercise in obese subjects. The authors conclude that their results 'suggest a metabolic pathogenesis for the impaired left ventricular function in obesity'. This may be true, but the data they divulge do not support this conclusion. Most importantly, their interpretation that lower \(\Delta\text{LVEF} \) (changes from rest to submaximal exercise) in obese/overweight subjects represents reduced ventricular performance is contingent upon the assumption that \(\text{LVEF} \) would increase indefinitely. As we all know, this assumption can never be true because mathematically, \(\text{LVEF} \) can never exceed the inviolable maximum of 100%. Moreover, their data show that the \(\text{LVEFs} \) during exercise were also not statistically different between the groups (Table 4). Because of similar exercise values, the size of \(\Delta\text{LVEF} \) is dependent on the resting \(\text{LVEF} \). Their observation of lower \(\Delta\text{LVEF} \) was therefore entirely explained by the higher resting \(\text{LVEFs} \) in the obese/overweight subjects, as shown clearly in Table 4, and had nothing to do with 'reduced ventricular performance'.

Previous work has shown that left ventricular end-systolic volume (LVESV) is a more reliable indicator of cardiac function than LVEF.\(^2\) We have taken the liberty of further analysing the data presented by Sasso et al.\(^1\) and find that there are no apparent differences in LVESV between any of the insulin or saline and lean vs. obese or overweight groups, either at rest or during the submaximal exercise (Figure 1), consistent with previous reports.\(^3\) This further confirms the view that their conclusion is not supported by their own data.

As LVEF is well known to be load- and rate-dependent, and these are likely to be different in the lean vs. overweight cohorts during exercise, it is surprising that no heart rate or blood pressure data were presented in the article, especially because in the Methods section, the authors describe that these variables were actually measured. No doubt, availability of such data would allow readers to gain more insight into what their data convey.

References

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Impaired left ventricular function in obesity?: reply

We read with interest the letter from Barker et al. on our work 'Effects of insulin on left

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