Letters to the Editor
doi:10.1093/eurheartj/ehi572

Online publish-ahead-of-print 5 October 2005

Impaired left ventricular function in obesity?

We read with interest the findings of Sasso et al.\textsuperscript{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 $\Delta$LVF (changes from rest to submaximal exercise) in obese/overweight subjects represents reduced ventricular performance is contingent upon the assumption that LVF would increase indefinitely. As we all know, this assumption can never be true because mathematically, LVF can never exceed the inviolable maximum of 100%. Moreover, their data show that the LVFs during exercise were also not statistically different between the groups (Table 4). Because of similar exercise values, the size of $\Delta$LVF is dependent on the resting LVF. Their observation of lower $\Delta$LVF was therefore entirely explained by the higher resting LVFs 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 LVF.\textsuperscript{2} We have taken the liberty of further analysing the data presented by Sasso et al.\textsuperscript{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.\textsuperscript{3} This further confirms the view that their conclusion is not supported by their own data.

As LVF 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

Diane Barker
The Academic Unit of Molecular Vascular Medicine
Leeds General Infirmary
Great George Street
Leeds LS1 3EX, UK

Simon G. Williams
Cardiac Transplant Unit
South Manchester University Hospital
Manchester, UK

Hilary McLoughlin
The Academic Unit of Molecular Vascular Medicine
Leeds General Infirmary
Great George Street
Leeds LS1 3EX, UK

Lip-Bun Tan
The Academic Unit of Molecular Vascular Medicine
Leeds General Infirmary
Great George Street
Leeds LS1 3EX, UK

© The European Society of Cardiology 2005. All rights reserved. For Permissions, please e-mail: journals.permissions@oxfordjournals.org