ventricular function during dynamic exercise in overweight and obese subjects.1 We thank Barker et al. for their comments that need our reply.

First, it is clear from our manuscript that the metabolic hypothesis for cardiac impairments in overweight/obese people is only a suggestion. We observed a significant correlation between insulin resistance and myocardial dysfunction, as we have already described in Type 2 diabetic subjects,2 but we have not investigated the potential biochemical mechanisms. The mismatch of substrate utilization and myocardial flow suggested for diabetic cardiomyopathy could be implicated also in obesity,3 but this remains to be proved.

Secondly, it is obvious that LVEF cannot exceed 100%. We simply reported in the results section, the changes in LVEF in the three groups, but we did not state that we expected that LVEF could increase indefinitely. As can be seen from the title, the aim of our investigation was to study the effect of insulin on ventricular function during exercise and not the reduced ventricular performance of obese subjects. The discussion, as a consequence, is centred on the difference between the two performed tests, thus revealing the effect of insulin. In other words, it does not matter if the size of delta LVEF is partly, totally, or not dependent on rest LVEF, because our aim was to stress the possible action of insulin on the variable, and the repeated measure ANOVA we performed revealed the statistical difference between insulin and saline infusion. This remains even if LVESV is not different. Moreover, we would like to underline that the statement by Barker et al. ‘left ventricular end systolic volume is a more reliable indicator of cardiac function than LVEF’ is referred to an investigation performed on patients with myocardial infarction.4 We remind that our patients had no evidence of pre-existing, structural, or ischaemic heart disease, nor, in particular, were recovering from myocardial infarction.

It is relevant to stress that data from the literature on cardiac function in obese people are scattered and inconclusive. Some authors did not demonstrate impairments, but more recently some others did.5 Our results do not pretend to be conclusive, but certainly support the second.

We did not report the values of heart rate and blood pressure simply because of editorial limitations. However, as stated in the results section, they did not significantly differ between the two tests, so they would add nothing more to what already discussed.

References


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Heart failure in elderly patients with chronic obstructive pulmonary disease

We read with great interest the article by Rutten et al.1 who report a significant association between the presence of chronic obstructive pulmonary disease and unrecognized heart failure in elderly patients. Approximately, 50% of heart failure patients in their study had isolated diastolic heart failure.

The authors suggested that the association between the two disorders may be attributable to the increased prevalence rates of atherosclerosis found in chronic obstructive pulmonary disease patients and the impressive smoking status of these patients. However, there is one other possible explanation for the high prevalence of heart failure in the chronic obstructive pulmonary disease in elderly patients. Some patients may have obstructive sleep apnea (OSA). The possible presence of sleep-related disordered breathing was not ruled out in this study and it has been demonstrated that OSA can originate diastolic dysfunction independent of other factors.2 Indeed, the treatment of OSA among patients with congestive heart failure and left ventricular (LV) systolic dysfunction leads to improvement in cardiac function,3 suggesting that OSA may also have an adverse effect on LV systolic function.

The prevalence of OSA is not greater in chronic obstructive pulmonary disease patients than in the general population, but this association, the so-called overlap syndrome, is not rare, as chronic obstructive pulmonary disease and OSA are both frequently occurring diseases.4,5 In fact, OSA has a two- to three-fold higher prevalence in subjects older than 65 years when compared with those in middle age.6 Overlap patients are at a higher risk of developing respiratory insufficiency than are pure OSA patients. These facts could have contributed to overestimate the real prevalence of heart failure in ‘pure’ chronic obstructive pulmonary disease patients.

References


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Letters to the Editor 2743
Heart failure in elderly patients with chronic obstructive pulmonary disease: reply

We thank the authors for their comments and agree that obstructive sleep apnoea (OSA) could be a risk factor for heart failure in our chronic obstructive pulmonary disease population, just like other known risk factors for heart failure, such as hypertension, diabetes, and atrial fibrillation, which were present in a substantial proportion of our chronic obstructive pulmonary disease patients.

As we mentioned in our discussion paragraph, the outcome panel assessed whether patients had heart failure or chronic obstructive pulmonary disease according to the ESC and GOLD criteria, respectively. These criteria are not influenced by the presence of OSA. We therefore disagree with the authors that (possible) concomitant presence of OSA in some of our chronic obstructive pulmonary disease patients.

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Association between obesity and obstructive sleep apnoea

We read with interest the article by Murphy et al., who report a significant association between obesity and fatal and non-fatal cardiovascular events in a large, population-based cohort of middle-aged obese patients compared with normal weight subjects followed for 20 years.

These comments are focused on the role of obstructive sleep apnoea (OSA) as another possible factor contributing to the described high rates of cardiovascular events and mortality in obese patients, an aspect not evaluated in this study. OSA is characterized by periodic reduction or cessation of breathing due to narrowing of the upper airways during sleep. Prevalence surveys estimate that 2% of women and 4% of men of middle age are affected by this syndrome. In patients with untreated severe OSA, a higher incidence of fatal and non-fatal cardiovascular events has been reported in comparison with that observed in untreated patients with mild–moderate OSA, patients treated with continuous positive airway pressure, and healthy subjects. Importantly, it has been estimated that OSA is present in 40% to 90% of obese subjects. Prevalence of OSA is probably rising as a consequence of increasing obesity, obesity being the most important risk factor for OSA. It may be related to changes in upper airway muscle tone, effects of fat deposition on upper airway anatomic structures, and changes in central mechanisms of breathing control. Obesity and OSA share multiple pathophysiological mechanisms such as endothelial dysfunction, insulin resistance, hyperleptinaemia, systemic inflammation, impaired baroreflex, or sympathetic hyperactivity. As a result, OSA may contribute to the presence of cardiovascular events observed in many obese patients.

In contrast, weight loss in OSA patients is associated to reductions in apnoea index, and chronic application of continuous positive airway pressure, the treatment of choice for OSA, reduces body fat and visceral fat accumulation in OSA patients. Therefore, clinicians must be aware to diagnose and treat obese patients for previously undiagnosed OSA, probably helping to reduce the high rates of cardiovascular events related to both conditions.

References


