Letter Regarding Article by O’Neill et al, “Peak Oxygen Consumption as a Predictor of Death in Patients With Heart Failure Receiving β-Blockers”

To the Editor:

A recent article1 published in Circulation investigated cardiopulmonary exercise testing with a primary aim of comparing patients who were or were not taking β-blockers at the time of the exercise test. The authors’ limitations did not mention the tracking of medication profiles during follow-up. It is likely, especially at a tertiary care center and during the early β-blocker era, that patients who were assigned to the no-β-blocker group at the time of testing were prescribed β-blockers shortly thereafter.

There are plenty of data that show that ventilatory efficiency measurement is a more powerful mortality predictor than peak oxygen consumption, and these measures must be explored further.2,3 Data suggest that the peak value of V˙E/V˙CO2 for ventilatory efficiency is probably not the best choice. The V˙E/V˙CO2 slope to peak exercise appears to be the best individual predictor of mortality obtained from cardiopulmonary exercise testing.2,3 Investigators should be wary of reporting any data that are automatically reported in final reports from computerized metabolic carts, such as a peak V˙E/V˙CO2 value. The authors should address the discrepancy in the peak V˙E/V˙CO2 value because the reported value is significantly lower (upper 20s versus ≈40) than those reported in the literature and those reported by the same group of authors4 a few years before. In 1999, this group reported a peak V˙E/V˙CO2 value of 40 in 470 subjects,4 subjects who may likely be included in the O’Neill data set.

Calculation of V˙E/V˙CO2 slope in O’Neill’s large cohort of patients within a multivariable model would add valuable prognostic information with relatively little additional effort. These data would be helpful in attempts to standardize reporting of cardiopulmonary exercise testing.

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Response

We agree with Mr Bard that lack of information about medication use after stress testing is a potential limitation of our study. If, as he suggests, patients not taking β-blockers were later treated with them, these patients would come to resemble those patients who were taking β-blockers initially. This greater similarity over time would be expected to diminish any differences between the groups, probably resulting in our underestimating the enhanced prognostic value of peak VO2 in β-blocker-treated patients.

We also agree with Mr Bard that the V˙E/V˙CO2 slope is a better measure than peak V˙E/V˙CO2. Unfortunately, we have been unable, despite a number of attempts, to retrieve from the metabolic carts the data needed to calculate the slope. We thank Mr Bard for pointing out a transcription error. As noted in our previous publication stemming from these data,1 the mean value for peak V˙E/V˙CO2 was 33±10, not 29±10 as we reported. We have confirmed the accuracy of all of the other data in our article, including the poor prognostic value of peak V˙E/V˙CO2 as compared with peak VO2. Still, as Mr Bard notes, this value is less than what we reported earlier on a much smaller sample of non-β-blockers with patients who underwent testing nearly 10 years ago.2 We suspect that the decline in V˙E/V˙CO2 noted over time may be caused by our changing practice of stressing maximal compensation of congestion before stress testing. Evidence of this is the higher rate of diuretic use in the more contemporary cohort of non-β-blockers patients (87% versus 79%).

Although we are in complete agreement with Mr Bard that it would have been ideal to have had detailed posttesting data on β-blocker use as well as actual V˙E/V˙CO2 slopes as opposed to only peak values, neither of these issues have an impact on the main finding of our article. β-Blocker use does not adversely affect the ability of peak VO2 to predict mortality in patients with severe systolic heart failure.

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