Peak oxygen uptake and exercise capacity: a reliable predictor of quality of life?

With great interest we read the manuscript published by Gratz et al.1 addressing the important issue of quality-of-life (QoL) and its possible correlation to exercise capacity in patients with congenital heart disease (CHD). The majority of surprisingly high QoL scores were not correlated to exercise capacity as quantified by peak oxygen uptake (peakVO2).

However, several questions have to be addressed concerning the manuscript. Gratz et al. showed unreliably high peakVO2 results with single values of more than 70 mL/kg/min. Applying our own recently published reference equations on these values would result in a peakVO2 of 170% as personally predicted (for a male subject of 25 years, 180 cm, 70 kg). Even the mean peakVO2 of their population partly exceeds the age, sex, and body size-adjusted fifth percentile for subjects of the above mentioned demographics. In addition, the cited reference values by Cooper et al. are not corrected for body weight; calculating a reference value for a dummy subject as described above results in about 3400, very likely not ml/kg/min. Furthermore, the applied reference equations provide unusually high reference values for peakVO2 when compared with ours and others.2–4

Besides methodological inconsistencies, it remains disputable to what extent peakVO2 contains reliable potencies to quantify QoL in a severely diseased population. QoL in patients with cardiac pathologies is well reflected by measures other than peak exercise values. For patients with congestive heart failure as well as pulmonary hypertension parameters of ventilatory inefficiency have been shown as reliable predictors of dyspnoea,5–7 a leading cause of exercise limitation in patients with CHD as well. Our own data in 25 adults with congenital cyanotic heart disease have shown that simple scores of the ability to accomplish daily life as well as dyspnoea8 are best reflected by measures of ventilatory inefficiency.9 For the slope of the regression of ventilation to carbon dioxide output (VE vs. VCO2 slope), these differences were: ABILITY II 43 ± 14; III 56 ± 25; IV 87 ± 37 (P < 0.001). Oxygen uptake at peak exercise and anaerobic threshold were less significantly correlated to dyspnoea and daily life symptoms.

PeakVO2, as assessed within a symptom-limited ramp-wise incremental exercise test, is probably hampered because of its limited reflection of capacity for daily activities. In severely diseased subjects it has been shown that peakVO2 values are higher if assessed within a six-minute walk test in comparison with incremental tests.10 This surprising difference possibly explains the limited value of the applied test design to correlate peakVO2 to QoL. Patients with severe cardiac abnormalities do very likely avoid exercising up to the extremes resulting in diminished peakVO2 but irrelevant for the daily life.

Gratz and colleagues do have an excellent material in their hand to further clarify which functional correlates do best reflect the underlying pathophysiology influencing QoL in this specific patient population. Focusing on sub-maximal parameters as well as values describing gas exchange may provide such explanations. The missing link of QoL to peakVO2 is, however, not surprising.

References