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LETTER TO THE EDITOR

Rethinking $\dot{V}O_{2\max}$: right problem, wrong solution (Letter to the Editor regarding Poole and Jones' "Measurement of the maximum oxygen uptake $\dot{V}O_{2\max}$: $\dot{V}O_{2\text{peak}}$ is no longer acceptable")

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TO THE EDITOR: I applaud Poole and Jones (3) for their provocative review noting that current tests to measure $\dot{V}O_{2\text{peak}}$ and $\dot{V}O_{2\max}$ are "no longer acceptable." Unfortunately, their solution for finding the elusive true $\dot{V}O_{2\max}$ does not address the fundamental problem with traditional cardiopulmonary exercise testing (CPET). Adding yet another difficult procedure to the already challenging task of pushing participants to exercise "to the limit of tolerance" at unnatural and uncomfortable exercise intensities is unlikely to succeed. In one sense, CPET is a biological application of nondestructive evaluation and testing (NDT). NDT is used to predict mechanical failure in many other complex systems [e.g., bridges (2)], in which a known perturbation is applied to the system at a level far lower than would actually damage the structure but sufficiently high to elicit a robust response variable that has predictive utility. No better example of this can be found than in cardiac exercise stress testing, in which ECG signals (abnormalities of ST segment and T-wave) can indicate the presence of coronary artery disease at submaximal exercise intensity levels without actually causing deleterious cardiac ischemia. Early 20th century scientists were intrigued by identifying the inability (failure) to increase $\dot{V}O_2$ with increasing work intensities that did not appear to harm healthy volunteers. The compelling nature of this marker of physiological system failure ($\dot{V}O_{2\max}$) shaped much of exercise physiology research for the past 100 years and led to profound advances in our understanding of exercise.

But more recently, adverse consequences of the "gold standard" status of $\dot{V}O_{2\max}$ have emerged. Poole and Jones (3) highlighted one of the most salient of these: the inability to easily measure $\dot{V}O_{2\max}$ in many (arguably, the majority) of human subjects. It has been recognized for years that submaximal CPET variables are themselves highly predictive of $\dot{V}O_{2\max}$ (1). But rather than focus on the actual variables obtained from submaximal exercise tests (such as heart rate or distance traveled, which are much more suitable to large- sample studies than specialized laboratory-based CPET), enormous efforts have been made to convert the measured values to estimates of $\dot{V}O_{2\max}$ by complex and assumption-ridden formulas. Many clinicians and researchers are concerned about excessively cajoling their subjects, particularly those with chronic disease and even otherwise healthy people with conditions like obesity, during heavy exercise, accompanied as it is by acidosis and large increases in inflammatory and neuroadrenergic mediators. Many researchers [including Poole and Jones (4)] are already pioneering alternative approaches like, for example, potentially predictive exercise biomarkers, such as exercise onset and recovery kinetic gas exchange, and heart rate variables. Enhanced computing and data analytics (machine learning) and innovative (even wearable) technologies to noninvasively capture a wide variety of physiological signals are increasingly available. It is time to rethink the biological value of maximal exercise testing, apply in novel ways the theoretical construct of NDT, and develop strategies to test fitness that are cost-effective, participant-friendly, easily linked to the burgeoning advances in genomics, proteomics, and metabolomics, and readily translated to the needs of clinicians and researchers alike.

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DISCLOSURES

No conflicts of interest, financial or otherwise, are declared by the author.

AUTHOR CONTRIBUTIONS

D.M.C. conceived and designed research; D.M.C. drafted manuscript;
D.M.C. edited and revised manuscript; D.M.C. approved final version of manuscript.

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