Since January 2020 Elsevier has created a COVID-19 resource centre with free information in English and Mandarin on the novel coronavirus COVID-19. The COVID-19 resource centre is hosted on Elsevier Connect, the company's public news and information website.

comorbidities (which were noted in 31 of the reported patients). Comparing with matched controls rather than predicted values would have been preferable. Yet the patients showed erratic increases in respiratory rate, with early CPET tachypnea as typically seen in PTS. There also was a tendency to decreased peripheral EO_2. This was wrongly calculated as arteriovenous O_2 content differences (DavO2) divided by hemoglobin rather than by arterial O_2 content, but characteristic anyway of deconditioning. Preload failure diagnosed in a subgroup of 7 patients disclosed vagotonic deconditioning as occurs in sedentary overweight subjects. Only 1 patient had upper limit of normal (at 1.97 WU) of pulmonary vascular resistance at exercise, not convincingly diagnostic of exercise-induced pulmonary hypertension.

Long COVID-19 and ME/CFS are patient advocacy-derived entities. Generously funded research to uncover their physiologic or biologic determinants (since 1987 for ME/CFS) has failed until now. Admirable efforts such as those reported by Mancini et al[1] should not distract from adequate attention to their dominant psychological components.

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The authors attest they are in compliance with human studies committees and animal welfare regulations of the authors' institutions and Food and Drug Administration guidelines, including patient consent where appropriate. For more information, visit the Author Center.

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REPLY: CPET for Long COVID-19

We appreciate the comments from Drs Naeije and Caravita and the citation of their work which was not available at the time our report was written.[1] Drs Naeije and Caravita are correct that we performed a detailed analysis of cardiopulmonary stress tests in 41 patients with long coronavirus disease-2019 (COVID-19).[2] Careful review of individual tests should not detract from our overall findings which showed ventilatory abnormalities in 88% of this cohort including 41% with elevated VE/VCO_2, 61% with hypocapnia, and 63% with disordered breathing. Deconditioning is generally not characterized by ventilatory changes. The mechanism underlying these ventilatory abnormalities remain unclear. Anxiety or psychological stress is one potential explanation for the hyperventilation, but altered chemosensitivity, lung thrombotic, or fibrotic changes are also possible. I think we can agree that more research is needed to clarify the significance of our findings and to help find an answer for those patients afflicted with long COVID-19 syndrome.

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