Reply to Suri *et al.*

From the Authors:

We really want to thank Suri and colleagues for their interest in our study (1) and for indicating that similar observations were made by other studies (2). However, as pointed out by them regarding erroneously citing the paper in which transmission occurred after symptom resolution, we want to clarify. In the cited study, the authors indicated that the transmission between index patients and other patients happened before symptom onset and not after the resolution of the symptoms, which is indeed correct (3). However, in the same study, the transmission was also reported between patient 1 and other patients (3, 4) who had no contact with the index patient. Patient 1 returned to work after the resolution of symptoms but was unaware of his infection with severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2). After confirmation of infection in the index patient upon return to China, patient 1 presented to the Division of Infectious Diseases and Tropical Medicine in Munich for further assessment. Patient 1 was found to be well with no signs of fever but had a very elevated viral load ($>10^8$ copies). Our citation referred to the transmission from patient 1 to patients 3 and 4 rather than from the index patient as was suggested by authors.

Another major point raised by the authors is the comparison between coronavirus and influenza infections. In the ferret model, the infective viral shedding time is limited to 5 days, whereas quantitative PCR (qPCR) positivity remains until Days 11–13. However, there are stark differences between influenza infection and coronavirus disease (COVID-19). Patients succumbing to COVID-19 remain positive for the virus until their death, even if it occurs 30–40 days after symptom onset, suggesting the ability of SARS-CoV-2 to persist in the body for prolonged times (4).

However, we agree with the need to find a better way to ascertain the shedding time in infected patients, as resources have been scarce to test all patients, especially multiple times, before being discharged from hospitals. In these conditions, patients are suggested to remain in isolation to avoid any spread of infection. However, we agree with the authors that it is possible that many of these patients do not shed the active virus despite being positive on qPCR. These findings need to be confirmed by large-scale studies in which throat swabs are indeed tested in a sufficient number of patients to determine if viral positivity on qPCR represents infective virus or just viral remnants that are unable to transmit along with the time kinetics. ■

Author disclosures are available with the text of this letter at www.atsjournals.org.

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Aortic Pulse Wave Velocity Predicts All-Cause Mortality in Chronic Obstructive Pulmonary Disease

To the Editor:

We read with keen interest the results of the SUMMIT (Study to Understand Mortality and Morbidity in COPD) randomized controlled trial of fluticasone furoate/vilanterol in patients with moderate chronic obstructive pulmonary disease (COPD) with a history of cardiovascular disease or at increased cardiovascular risk (1). The trial evaluated both the value of aortic pulse wave velocity (aPWV) to predict all-cause mortality (ACM) in this population and

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