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.

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marker of myocardial injury in patients with coronavirus disease 2019 (COVID-19) and on the strong prognostic implications of this simple and easily available biomarker. Unfortunately, troponin is a generic marker of myocardial damage and cannot provide any valuable insight into the pathophysiological mechanism of the damage. We believe that this limitation could have been partly resolved by the systematic evaluation of standard electrocardiogram (ECG). Paradoxically and unexpectedly, 5 months after the beginning of the "COVID-19 era," data on standard ECG as a screening tool for cardiovascular complications are almost completely missing in the literature—1 recently published and 1 in-press paper (2,3)—whereas ECG details are available only for selected patients diagnosed with myocarditis or acute coronary syndrome. The extreme lack of ECG data is all the stranger considering it is a broadly available, low-cost diagnostic test that can be quickly performed without exposing a large number of personnel to the virus. This ECG eclipse has contributed to generate the misconception that "myocardial injury" diagnosed by elevated serum troponin is synonymous with myocarditis or acute coronary syndrome, neglecting the fact, for instance, that acute pressure overload of the right ventricle can also cause an increase of this biomarker. Indeed, compared to troponin, ECG can provide not only a generic diagnosis of myocardial injury or damage but can also orient to the specific pathophysiological mechanism and foster suspicion of pulmonary thromboembolic or in situ thrombosis of the pulmonary circulation, which are being described with increasing frequency (4).

In conclusion, the high frequency and the prognostic implications of increased troponin I reported by Lala et al. support the importance of a systematic screen of the full spectrum of cardiovascular complications of COVID-19 infection, including events threatening the right and not only the left ventricle. Standard ECG is fundamental in this strategy, so systematic studies on this issue are urgently needed.

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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 *JACC* author instructions page.

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REPLY: What Happened to Electrocardiogram as a Screening Test to Recognize Cardiovascular Complications in COVID-19 Patients?

We thank Dr. Bertini and colleagues for their thoughtful response to our paper (1). Specifically, the authors comment on the relative dearth of electrocardiographic (ECG) data in the study of the novel coronavirus disease-2019 (COVID-19). They further suggest that ECG information could have offered incremental insight in determining the underlying pathophysiology of elevated troponin levels, representing myocardial injury.

We agree that ECG data are additive in helping to understand the nature of myocardial involvement and also holds prognostic relevance. Unfortunately, reviewing ECG data in our large cohort of patients (nearly 3,000) was not feasible within a reasonable timeframe to allow the dissemination of information that was of importance during a surge in COVID-19 cases across the globe. Furthermore, data were collected during the peak of the pandemic in New York City (February 27 to April 12, 2020), with variable uses of antiviral therapy, anticoagulation, and hydroxychloroquine, which may have influenced ECG information. Nonetheless, the message of our paper was simple and still holds true: myocardial injury is common, and when present, it portends worse prognosis among patients hospitalized with COVID-19. Myocardial injury is represented by elevated troponin concentrations based on the fourth universal definition of myocardial infarction (2). We acknowledge that

understanding the underlying pathophysiology of myocardial injury in the setting of COVID-19 was beyond the scope of our paper. Indeed, several months after our data were collected, the causal relationship between severe acute respiratory syndrome-coronavirus-2 and cardiac involvement remains elusive and requires further study (3). Systematic study of ECGs among affected patients is of interest.

Another important point raised by Bertini et al. is the frequently inappropriate invocation of severe acute respiratory syndrome-coronavirus-2 myocarditis as a diagnosis based on elevated troponin levels. In fact, as we have seen in recent studies, this diagnosis requires the presence of histologic findings and viral particles within myocytes as well as the exclusion of other cardiotropic viruses within the appropriate clinical setting, and has proven to be quite rare (4).

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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 *JACC* author instructions page.

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