

experience of each individual institution. If advanced therapies are unavailable, thrombolytics may remain a primary treatment modality. Even though people may have survived prolonged cardiopulmonary resuscitation while waiting for thrombolytics to work, we feel that such a scenario should be avoided if possible. Consideration should be given to transfer to a facility with more advanced capabilities, if the patient can be stabilized for transport.

Dr. Dai and colleagues highlight issues of using invasive hemodynamics as proxies for RV function in the setting of SMPE. By definition, patients with SMPE have objective evidence of RV strain and are hemodynamically stable. Within the SMPE category, there is a broad spectrum of patients, ranging from truly asymptomatic, stable patients to “stable” patients on the verge of decompensating. Supposedly “stable” patients may show signs of imminent decompensation with minimal challenge, such as minor movement. Thus, the European Society of Cardiology (ESC) subdivides SMPE into intermediate-low and -high risk categories, with intermediate-high risk having some clinical features predictive of decompensation (5). Yet, regarding SMPE, ESC guidelines note “...no individual clinical, imaging, or laboratory finding has been shown to predict the risk of an adverse in-hospital outcome.” Determining which “stable” patients are on the verge of decompensation relies largely on the astute assessment of the treating clinician. The SMPE in our series only included intermediate-high risk patients who had clinical features predictive of decompensation, such as recurrent syncope/near syncope or exacerbation of symptoms with minor movement. All patients with SMPE in our study received typical management for RV failure, including volume restriction. Our intraoperative management entailed intubation after insertion of invasive monitoring lines. Thus, confounding factors, such as volume overload and mechanical ventilation, did not have an impact on the preoperative central venous pressure (CVP) values in our series. We believe that the mean CVP in our SMPE population of 22.1 mm Hg, which immediately improved to 10.6 mm Hg post-operatively, reflects the severity of RV dysfunction and the efficacy of treatment. It is important to emphasize that we do not reflexively intervene on all intermediate-high risk SMPE. Not mentioned in our paper are 3 patients with SMPE who were taken off the operating table due to normal pre-operative CVP and were successfully managed with heparin alone. Perhaps patients with normal CVP fall within the lower-risk spectrum of SMPE and can be managed with heparin alone.

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



We read with great interest the paper from Lala et al. (1). The authors must be congratulated for focusing attention on the clinical relevance of troponin I as a

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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## 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