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<https://doi.org/10.1016/j.jacep.2021.07.001>

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Dr. Cauti has received support from Abbott and Boston Scientific for EP and CRM proctoring. All other authors have reported that they have no relationships relevant to the contents of this paper to disclose.

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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TO THE EDITOR

The 12-Lead ECG in COVID-19

QT Prolongation Predicts Outcome



We read with the interest the recent paper by Raad et al (1) describing the association between right heart strain (RHS) pattern on the electrocardiogram (ECG) and poor outcome in coronavirus disease-2019 (COVID-19). Patients developing a new RHS pattern in their series were more likely to require mechanical ventilation and less likely to survive, in comparison with patients without this ECG finding. This finding may provide an index of acute respiratory distress: compared with their counterparts, patients with the RHS pattern had a lower peripheral capillary

oxygen saturation relative to the fraction of inspired oxygen.

The RHS pattern is seen as a hallmark of pulmonary embolus, and thromboembolism is one of the characteristics of severe COVID-19 illness (2). Raad et al (1) were able to confirm or exclude this crucial diagnosis in only a small minority of their cohort: only 2 computed tomography pulmonary angiography scans were completed in their study population. The lack of a clear time frame also weakens the argument; it is not clear from the paper whether pre-COVID ECGs were available for all subjects, and the time from the reference ECG to the time of presentation with COVID is not stated. It therefore cannot be determined whether the RHS pattern was truly “new” in these patients or was a reflection of prior respiratory pathology that had remained undetected until the COVID admission.

Raad et al (1) present data showing a longer QTc interval in the cohort of patients with RHS compared with others (449 ms vs 435 ms; $P < 0.001$) and a greater proportion of patients with a prolonged QTc interval (53% vs 23%; $P < 0.001$). These fascinating findings are presented in Table 1 of the paper but are not mentioned elsewhere. There is no account of the methodology of QTc calculation; perhaps it was overlooked, as the QT interval was not their focus. This link between COVID and QT interval has been reported previously (3) but is unappreciated.

The QTc interval has been a consistent marker of poor outcome in COVID-19, independent of drug effects (3,4). The most significant prolongation occurs in patients with worst outcome; the QTc interval has been shown to predict prognosis (3). Compared with a categorical marker like the RSR' pattern, a continuous variable like the QT interval has the advantage of potentially providing a gradation of risk. As a single numerical measurement, the QTc interval may be easier to standardize for inexperienced readers of the ECG. It is also a variable that can be measured automatically, albeit with a need for some overreading by an experienced clinician.

The simplicity and availability of the 12-lead ECG makes this a potentially valuable tool in the risk stratification and management of COVID patients. Although an abnormal ECG may be a marker of patient outcome, no single ECG abnormality is pathognomonic of the SARS-CoV-2 (severe acute respiratory syndrome-coronavirus-2) infection (5). The findings of Raad et al (1) allow us to add the RHS pattern to the list of ECG features that should

heighten concern for a patient with COVID. QT prolongation should head that list.

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<https://doi.org/10.1016/j.jacep.2021.05.017>

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Dr. Akhtar has received funding for a research fellowship from Abbott Medical. Dr. Kontogiannis has reported that he has no relationships relevant to the contents of this paper to disclose. Dr. Sharma has received educational grants and served as a paid speaker for Boston Scientific, Bayer, Abbott Medical, Pfizer,

and Bristol-Myers Squibb. Dr. Gallagher has received research funding from Attune Medical; and served as a consultant and a paid speaker for Boston Scientific and Cook Medical.

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