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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: Insights From HeartLogic Multisensor Monitoring During the COVID-19 Pandemic in New York City

We recently described effects of the coronavirus disease-2019 (COVID-19) pandemic on health care delivery for patients with heart failure as well as the intersections between COVID-19 infection and heart failure (1). In light of increased noncontact care delivery methods for ambulatory care, telemedicine combined with the use of pulmonary artery pressure monitoring and biosensing devices can help guide heart failure management.

Dr. Mitter and colleagues describe their institutional experience with remote monitoring using HeartLogic multisensor monitoring during the COVID-19 pandemic. The HeartLogic algorithm captures information regarding heart sounds, respiration, thoracic impedance, heart rate, and activity data (2). They retrospectively reviewed data from 38 patients and found a significant decrease in activity level with only small increases in thoracic impedance. As the authors note, sedentary behavior is thought to contribute to worsening heart failure syndromes. They postulate that changes in diet and decreased autonomic tone may have contributed to their findings. While thought provoking, these data are reported from a very small sample population and are hypothesis generating. As the COVID-19 pandemic continues, larger such studies in populations of patients with heart failure and cardiac implantable electronic devices are warranted.

The COVID-19 pandemic is an opportunity for the heart failure community to incorporate more data generated by implantable monitors into routine care. Many of our patients, particularly those with systolic heart failure, have cardiac implantable electronic devices. Although HeartLogic is specific to Boston Scientific (Marlborough, Massachusetts) devices, Medtronic (Minneapolis, Minnesota) similarly has a CareLink remote monitoring network, and St. Jude Medical (St. Paul, Minnesota) has the Merlin network in addition to the implantable CardioMEMS device. These tools can provide valuable insight into patients' activity levels, volume status, and arrhythmia burden, which could also be a trigger for worsening heart failure. These devices have limitations, and the use of intrathoracic impedance monitoring or heart rate variability from cardiac implantable electronic devices has not been demonstrated to improve clinical outcomes in large trials (3). Yet, integrating these data from remote monitoring may constructively supplement our care of patients with heart failure during the COVID-19 pandemic and beyond.

We thank Dr. Mitter and colleagues for their application of important insights from our work. Moving forward, we must continue to stay vigilant and creative to find new and effective strategies for caring for our patients with heart failure.

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When and for Whom Should We Use SGLT2 Inhibitors in HFrEF?

Many new therapies for heart failure with reduced ejection fraction (HFrEF), such as sacubitril/valsartan and sodium-glucose cotransporter-2 (SGLT2) inhibitors, have been shown to reduce morbidity and mortality in patients with HFrEF (1,2). Interestingly, SGLT2 inhibitor therapy was associated with improved morbidity and mortality rates regardless of type 2 diabetes (T2D) status (2), raising the importance of glucose-lowering drugs in HFrEF therapy. Thus, it is highly needed to reconstruct the clinical positioning of SGLT2 inhibitors in HFrEF therapy and to consider proper use and combination of those medications (3).

In a recent issue *JACC: Heart Failure*, Solomon et al. (4) compared the efficacy and safety of dapagliflozin among HFrEF patients who were and were not taking sacubitril/valsartan at baseline in the DAPA-HF (Dapagliflozin And Prevention of Adverse-outcomes in Heart Failure) trial. They found that dapagliflozin carried a similar safety profile in both subgroups and that use of both agents was additively efficacious in patients with HFrEF. Mortality among patients who were taking sacubitril/valsartan was slightly lower compared with among those who were not, while the risk of worsening heart failure in patients who were taking sacubitril/valsartan was still higher. This may at least partly result from the fact that patients who were originally taking sacubitril/valsartan were sicker, with a higher clinical severity of HFrEF, such as lower left ventricular ejection fraction and higher incident cardiac device use, as shown in the paper. Importantly, the use of dapagliflozin reduced the risk of worsening heart failure and deaths even in refractory HFrEF patients taking sacubitril/valsartan, suggesting that dapagliflozin has great potency to be incorporated into the algorithm for HFrEF treatment, irrespective of T2D.

Currently, SGLT2 inhibitor therapy is recommended to reduce the risk of worsening heart failure, particularly in T2D patients in whom heart failure predominates (5), while there is no treatment guideline for HFrEF listing an SGLT2 inhibitor in its therapeutic algorithm. Given the striking results by Solomon et al. (4), even the combined use of a SGLT2 inhibitor and sacubitril/valsartan would further benefit patients with HFrEF. Ongoing clinical trials and future research should further elucidate the appropriate clinical setting for SGLT2 inhibitor therapy in the algorithm for HFrEF treatment.

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