Although our understanding and therapy of chronic heart failure have advanced substantially, acute decompensation of heart failure remains a frequent cause of hospitalization and death (1,2). New effective treatments for acute decompensated heart failure remain elusive despite several recent randomized clinical trials of promising new drugs and interventions (3–5). Many hospitals have created heart failure units with standardized protocols to ensure that staff proactively, and consistently, apply the best current treatments and processes of care to appropriate patients to reduce complications and improve outcomes. Yet length of stay, short-term readmission rates, and mortality remain significant (6), suggesting that our treatment paradigms are not addressing the core problems or precipitants.

The cascade of acute decompensated heart failure requires new understanding and interventions. Its importance and impact are reflected in clinical trials in which a reduction in hospital admission for worsening heart failure is targeted as one of the most common clinical end points. It remains a common reason for hospital admission in patients older than 65 years and consumes many resources. This is despite many large trials that have shown that heart failure can be prevented and that time to first heart failure admission can be reduced, although this may often be a postponement rather than a cure. Upstream, specialized outpatient clinics have been established to stabilize chronic heart failure and reduce decompensation. They have flourished to some degree, but the expertise and care provided are not standardized, although models are available (7–10). These clinics and individual physicians including primary care emphasize evidence-based therapies (11–14), persistence with prescribed treatments, avoidance of a dietary sodium load, and patient recognition of worsening symptoms as a trigger to increase diuretic dose according to previous education and specific directions, including contact with their physician if symptoms do not promptly improve. Many patients are helped in this way, but others still have acute decompensation without recognizing precipitants or warning signs. Weight changes may not be measured regularly or may be a later sign of peripheral edema rather than an early sign of pulmonary congestion. The patient often recognizes the problem late, and a more sensitive indicator of early and persistent accumulation of pulmonary congestion could reduce clinical decompensation with fewer urgent physician visits and hospital admissions.

The emerging emphasis on device therapy in heart failure patients provides additional unique opportunities to study and intervene in the management of this high-risk population. Implantable cardioverter-defibrillators (ICDs) reduce the 5-year mortality rate by 23% among heart failure patients with poor left ventricular function (15,16) and 30% among those with previous cardiac arrest (17–20). Cardiac resynchronization therapy (CRT) (biventricular pacing) coordinates ventricular contraction in a wide QRS rhythm and can reduce heart failure symptoms (21–23), improve quality of life, and reduce hospital admissions for heart failure (24,25). As a result, ICD/CRT therapy for heart failure patients has grown rapidly. It has also provided an exciting opportunity to expand continuous monitoring beyond cardiac rhythm to include novel surrogate measures of lung congestion such as intrathoracic impedance between the lead tip in the right ventricle and the generator in the left subcutaneous infraclavicular region. An increase in lung water is reflected as a decrease in measured impedance, and this may be detected and reported by the device before symptoms of heart failure occur. Impedance monitoring is feasible, and established device algorithms and thresholds correlate reasonably well with invasive measures of venous pressure (26) and have demonstrated modest sensitivity and specificity for clinical events (27). Among patients at high risk of fluid overload and heart failure exacerbations, early warning of impending decompensation may allow treatment adjustment even before symptoms occur and may have an impact on important clinical outcomes such as heart failure hospitalization (28). Although promising, intrathoracic impedance measures are confounded by extrapulmonary
changes in chest impedance, they demonstrate a less than ideal predictive value for heart failure exacerbations, and it is not yet fully clear what independent value they will provide for heart failure management.

In an effort to improve the predictive value of these impedance measures, Whellan et al. (29), in this issue of the Journal, devised an algorithm that combined impedance data with prospectively identified criteria: a fluid index >100 Ω days or any 2 of the following criteria met during 1 evaluation period: long atrial fibrillation duration, rapid ventricular rate during atrial fibrillation, a high (≥60) fluid index, low patient activity, high night heart rate, low heart rate variability, low CRT pacing, or ICD shocks. The use of multiple parameters in this observational study significantly improved the ability to identify patients at risk of heart failure events, beyond the use of intrathoracic impedance alone, in the subsequent 30 days. Although observational studies are important to develop testable hypotheses, they cannot replace randomized clinical trials to confirm the true utility of this strategy. Whether additional parameters beyond atrial fibrillation episodes, patient activity level, and heart rate variability with the impedance fluid index will give increased predictive accuracy can be tested, but the composite score using the available described data is rational and sensible, and this study demonstrates some benefit with this approach.

New prospective studies will expand our current knowledge and refine diagnostic algorithms to predict heart failure exacerbations. Thoracic impedance will be a strategy to consider in appropriate patients who require an ICD/CRT, but it would not be a primary impetus in the decision to implant such device therapy. Yet, it could become a valuable tool to educate patients by correlating their impedance measurements and other algorithm data with diet or medication changes as well as by providing remote monitoring for patients unable to attend regular outpatient visits. Further studies are needed to refine algorithms, to study heart failure with preserved ejection fraction, and to determine cost-effectiveness. Patient selection may evolve from those at highest risk to those at lower risk for whom ICD/CRT may not yet be indicated, and the incremental value of stand-alone impedance measures will need evidence and justification. The addition of other clinical data may add value to the currently described algorithm.

Hospital admissions for acute decompensated heart failure are unplanned but are associated with significant morbidity and mortality. The opportunity to predict what often seems currently unpredictable offers hope for patients, physicians, and payers.

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