editor, we feel that some of them miss the point—namely the primary endpoint (pun intended) of the study: colchicine was not associated with any improvement in symptom severity. Having said that, we find it hard to believe that using cardiopulmonary stress testing would change the essence of the study results in any meaningful way. In regard to the degree of inflammation abatement, we agree that the levels of on-treatment C-reactive protein indicate significant residual inflammation; however, more potent anti-inflammatory action would probably require higher colchicine doses, which would entail higher frequency of adverse effects (from our experience, doses higher than 0.5 mg twice daily are associated with markedly increased—mostly gastrointestinal—side effects, which lead to significant declines in patient compliance). Whether other anti-inflammatory agents can offer full inhibition of inflammatory processes, without significant toxicity, is a matter of future research—but, again, any such effect must be accompanied by significant improvements in symptoms or, even better, in hard endpoints, including heart failure-related hospitalizations and mortality (unfortunately, clinical study results have been less than compelling in that respect).

Finally, we find the idea that more severely afflicted heart failure patients might benefit more from anti-inflammatory treatment quite intriguing and believe it is indeed worthy of further investigation.

Pseudobendopnea?

The well-performed and presented study on the characterization of bendopnea in patients with advanced heart failure by Thibodeau et al. (1) merits additional clinical commentary. It is unclear as to why the patients were referred for right heart catheterization. Was this performed in concert with a left heart catheterization? It would appear that it was not. Equally so, it would appear that it was not done solely for the purpose of the study but as a secondary foundation for pathophysiological characterization of the symptom of dyspnea on bending. As a diagnostic tool, the data indicate a low sensitivity for that purpose because all patients with bendopnea (100%) had dyspnea on exertion, and of those without bendopnea, 80% had dyspnea on exertion, whereas bendopnea was present in only 29% of the patients studied.

The excellent clinical commentary by Nohria and Stevenson (2) that observation is never obsolete, and its correlation with signs and symptoms appropriately noted, may be overzealous concerning the utilization of bendopnea in the routine management of cardiac patients. The elucidation of bendopnea either voluntarily by the patient or extracted by the attending physician is also limited as a sign or symptom when compared with the standard and acceptable clinical measures of cardiac decompensation via the history and physical examination, as noted in Thibodeau et al. and Nohria and Stevenson (1,2).

This study now brings to the clinical forefront of medical practice the following question: what is the significance of shortness of breath on bending forward (standing or sitting) in those patients who do not have heart failure as a diagnosis? This is not an uncommon complaint in the early-elderly and mid-elderly patient populations. They may have the usual comorbid diagnoses (excluding obesity) of hypertension, diabetes, asymptomatic coronary heart disease, or mild pulmonary disease of various categories; contrariwise, they may not have any of the aforementioned conditions other than age, which in itself may be associated with “stiffness” of the lungs and/or myocardium. In these patients, do we now invoke the term pseudobendopnea? It seems inappropriate to do so, for they still complain of shortness of breath on bending. Unfortunately, we have no studies or observations that assist in the elucidation and characterization of the pathological physiology in those patients when compared with those in heart failure as studied by Thibodeau et al. (1). It is possible that we may be dealing with impaired systolic function with preserved ejection fraction as studied by

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Kraigher-Krainer et al. (3) or pre-clinical diastolic dysfunction as reviewed in the state-of-the-art paper by Wan et al. (4). Perhaps a combination of both may share responsibility in the production of bendopnea in these patients, thus placing them in a state of unfortunate prognostication. I have noticed that these particular patients may be performing a modified Valsalva maneuver upon bending forward, and the symptom of shortness of breath can be eliminated by exhaling when going downward and inhaling when coming up, thus bypassing the physiology produced by a Valsalva-type maneuver.

Perhaps it is appropriate to conclude by quoting the words of the protagonist at the end of Ernest Hemingway’s magnificent novel The Sun Also Rises: “Well, where do we go from here?”

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Characterization of a Novel Symptom of Advanced Heart Failure: Bendopnea

The article by Thibodeau et al. (1) characterizing the invasive hemodynamics of heart failure patients complaining of shortness of breath while bending corroborates a similar observation that I have recently published (2). Using Doppler echocardiography, I also demonstrated an increase in estimates of pulmonary capillary wedge pressure beyond baseline while bending forward, in normal patients as well as in those with known chronic heart failure (CHF). The present study adds to our knowledge of this phenomenon by examining a larger number of patients and measuring hemodynamics invasively.

Thibodeau et al. (1) did not emphasize what I have found over the years to be the clinical utility of recognizing this symptom. Shortness of breath bending, although nonspecific, is clinically helpful when it resolves with diuresis. Teaching and recognizing this symptom aids in the early clinical identification of patients with decompensated CHF, and can help prevent unnecessary hospitalizations.

Another unrecognized clinical symptom that I believe sometimes signals volume overload is a watery post-nasal drip, especially in the elderly. I propose that many patients labeled with “senile rhinitis” (3) have occult, chronic elevation in left ventricular filling pressures. The nasal discharge in these patients appears to be a symptom of right heart failure, similar to the appearance of isolated peripheral edema. I have seen many patients whose “post-nasal drip” resolves completely with diuresis and effective CHF treatment, including one recently whose severe, acute pulmonary edema was preceded only by 1 week of nasal drip, which never occurred previously and resolved completely after treatment. This is another nonspecific, but clinically useful, symptom for clinicians to be aware of and for researchers to study and characterize further.

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REPLY: Bendopnea and Pseudobendopnea

We appreciate the interest of Drs. RuDusky and Brandon in our study (1). Patients had been referred for diagnostic right heart catheterizations for clinical indications. A minority (19%) had a concomitant left heart catheterization. The current study was not able to address the diagnostic utility of bendopnea.