

5. Hunt SA, Abraham WT, Chin MH, et al. ACC/AHA 2005 guideline update for the diagnosis and management of chronic heart failure in the adult — summary article: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Writing Committee to Update the 2001 Guidelines for the Evaluation and Management of Heart Failure): developed in collaboration with the American College of Chest Physicians and the International Society for Heart and Lung Transplantation: endorsed by the Heart Rhythm Society. *Circulation* 2005;112:1825-52.

DR. LIU AND COLLEAGUES REPLY: We agree with Fiack and Farber with respect to the potential contribution of pulmonary hypertension to death in patients with heart failure. We did not independently assess pulmonary hypertension in the entire cohort; however, we are reviewing the echocardiograms from a subgroup of these patients to look for the presence of right ventricular hypertension. Many patients who have heart failure with preserved systolic function may have elevated ventricular filling pressures, leading to pulmonary hypertension as an important complication affecting their condition. Interestingly, this may also represent an important therapeutic opportunity.

Arias et al. mention that obstructive sleep apnea is an important factor in the development and outcomes of both systolic and diastolic heart failure. Our group has conducted studies showing the relation between sleep apnea and ventricular dysfunction and outcomes.^{1,2} Sleep apnea also contributes to the development of atrial fibrillation, stroke, and hypertension and the worsening of heart failure. Heart failure with preserved systolic function is a heterogeneous disease, and sleep apnea could easily be a contributor to its adverse outcome. Tempering this argument, however, is the sex distribution of the population. Sleep apnea is usually more prevalent in men, whereas in our patient population, preserved systolic function was more common in elderly women.

We agree with Kessler that identification of the causes of death in patients who have heart failure with preserved systolic function will provide important insight into its pathophysiology. We do not have these data available, but we will be examining this issue in other, related data sets. Because patients with heart failure frequently have complex coexisting conditions, they undoubtedly die from causes other than heart failure alone. However, a large proportion of patients with preserved systolic dysfunction are repeatedly hospitalized for heart failure, as are patients with severe systolic heart failure, and they probably ultimately die of heart failure.

Finally, Ahmed et al. remind us that digoxin has been approved for the treatment of heart failure, irrespective of the ejection fraction. Although specific treatment strategies for heart failure with preserved systolic function have yet to evolve, the general principles are meticulous fluid management and treatment of the underlying conditions. We concur that digoxin certainly does have a role when patients continue to have symptoms or their condition continues to be unstable despite the use of existing therapies.

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2. Sin DD, Logan AG, Fitzgerald FS, Liu PP, Bradley TD. Effects of continuous positive airway pressure on cardiovascular outcomes in heart failure patients with and without Cheyne-Stokes respiration. *Circulation* 2000;102:61-6.

2005 Measles Outbreak in Indiana

TO THE EDITOR: Parker et al. (Aug. 3 issue)¹ describe the largest documented outbreak of measles in the United States since 1996, which is of considerable epidemiologic interest. It is surprising that few of the cases were confirmed by laboratory analysis, particularly one of the two involv-

ing “vaccine failure.” Could the authors explain why, with the availability of noninvasive testing methods, more patients were not tested? It is also unclear how local investigators identified the cases and whether any consideration was given to the possibility of asymptomatic infection, since there

is evidence that measles may circulate in vaccinated populations and cause subclinical infection.²

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1. Parker AA, Staggs W, Dayan GH, et al. Implications of a 2005 measles outbreak in Indiana for sustained elimination of measles in the United States. *N Engl J Med* 2006;355:447-55. [Erratum, *N Engl J Med* 2006;355:1184.]

2. Vardas E, Kreis S. Isolation of measles virus from a naturally-immune, asymptotically re-infected individual. *J Clin Virol* 1999;13:173-9.

THE AUTHORS REPLY: We obtained laboratory confirmation for at least one patient in 9 of 11 families infected with measles. Among these 9 families, 14 of 20 patients had disease that was confirmed by laboratory analysis. The remaining two families (with 10 and 4 patients, respectively) declined to have specimens collected from all family members. The parents of the patient who had measles despite receiving two doses of vaccine declined to have specimens collected from any of their children except one whose disease was confirmed by laboratory testing during hospitalization. All patients had classic clinical symptoms of measles that appeared after the appropriate incubation period after exposure to a patient with laboratory-confirmed disease. The percentage of cases that

were confirmed by laboratory testing in the Indiana outbreak (41%) was similar to that in other outbreaks among groups of persons who had declined to receive vaccination.^{1,2}

Case finding involved contacting persons with a known exposure to measles, physician alerts, and media releases. Although the asymptomatic spread of measles could potentially occur, all but one patient had an identified source. This patient worked in a hospital where patients with measles had been treated within 14 days before the onset of her symptoms. Thus, we believe that asymptomatic transmission was unlikely to have played a major role in the Indiana outbreak.

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1. Hanratty B, Holt T, Duffell E, et al. UK measles outbreak in non-immune anthroposophic communities: the implications for the elimination of measles from Europe. *Epidemiol Infect* 2000; 125:377-83.

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Pay-for-Performance Programs in the United Kingdom

TO THE EDITOR: As a general practitioner in England, I and the practice in which I work were directly affected by the changes made in 2004 by the introduction by the National Health Service of a pay-for-performance contract for family practitioners, as reported by Doran and colleagues (July 27 issue).¹ The contract was evidence based, ensuring that the majority of general practitioners approved of its aims.

Doran and colleagues omitted a number of important lessons that can be drawn from that experience. First, the necessity to “tick boxes” to ensure that tasks triggering payment were completed had a major effect on many consultations each day. Second, much bigger than the payments to general practitioners were the increased consequential costs triggered by the quadrupling of prescriptions for statins. Third, there was the effect on the local hospitals of a sudden increase

in referrals for investigative procedures such as echocardiography for heart failure and cardiac referrals for angina — conditions that previously had often been dealt with without referrals. Fourth, there is the increased medication load for patients — typically, a patient with diabetes has to take 10 different therapies.

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1. Doran T, Fullwood C, Gravelle H, et al. Pay-for-performance programs in family practices in the United Kingdom. *N Engl J Med* 2006;355:375-84.

TO THE EDITOR: In his editorial accompanying the article by Doran and colleagues, Epstein¹ encourages the United States to adopt a system similar to that introduced in the United Kingdom.