REPLY

The writing group of the “Scientific Statement on the Evaluation of Syncope” takes this opportunity to respond to the critique of this document (1) by Dr. Benditt and colleagues. The goal of this Scientific Statement, as set forth by the sponsoring organizations, was to provide a concise, practical approach to the initial evaluation of the patient with syncope within strict length limitations (2). The document approaches the evaluation of syncope as a clinician would when a patient presents to the office or hospital with such an event. The emphasis of this document is placed on the recognition of life-threatening clinical syndromes.

The criticisms of Dr. Benditt and colleagues can be grouped into three categories. First, the definition of syncope was inadequate. Second, the stated goal of preventing death is not adequate, and the goal for the evaluation of syncope should also be to establish a diagnosis and provide a prognosis. Third, the citations were incomplete.

The definition of syncope as "a transient loss of consciousness" fits into the category of a practical working definition. Although there is no uniform consensus on the ideal definition, the definition often includes reference to the loss of consciousness due to global cerebral hypoperfusion. Unfortunately, this definition can be strictly applied only when the mechanism of syncope is firmly established. The definition used in this document relates to the clinical presentation, as commonly used and understood, and does not require a specific mechanistic diagnosis. Academically, one might prefer a more detailed or thorough definition. However, one could also argue that any definition intended to be used universally should be developed by international consensus among appropriate medical societies. To date, this has not been achieved.

The specific etiology of syncope is identified in only about one-half of the patients who undergo an evaluation for syncope. Furthermore, many patients never have a recurrence after an episode of syncope, and only occasionally is syncope disabling. Ultimately, one can have many goals for a document of this type. We chose the identification of the patient at risk of death as the primary one.

Dr. Benditt and colleagues correctly state that many references relevant to syncope were not cited. Owing to space considerations and the document’s focus on the evaluation of syncope, many excellent papers could not be referenced. We agree that the “Guidelines on Management (Diagnosis and Treatment) of Syncope” developed by the European Society of Cardiology are particularly important documents, and we apologize for omitting them (3,4).

The “Scientific Statement on the Evaluation of Syncope” was reviewed and evaluated by >50 outside reviewers. The document was reviewed and approved by the American Heart Association (AHA), American College of Cardiology Foundation, Heart Rhythm Society, and the American Autonomic Society. Criticisms and comments offered by these reviewers and organizations were responded to and incorporated into the final document. Although there are inherent limitations to any such document, the writing group believes that the “Scientific Statement on the Evaluation of Syncope” achieves the goals set forth by the AHA for this document, and it provides a concise and practical approach to the initial evaluation of syncope.

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doi:10.1016/j.jacc.2006.09.026

Please note: this response is from the authors and is not approved or sanctioned by the sponsoring organizations.

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Examining the Concept of Preserved Systolic Function

Sophisticated analyses and the size of the Acute Decompensated Heart Failure National Registry (ADHERE) database strengthen the characterization of heart failure with preserved systolic function (PSF) made by Yancy et al. (1). Unfortunately, PSF includes...
ejection fractions (EFs) of 40% to 49% that are, by definition, abnormal. Excluding borderline abnormal EFs from systolic heart failure leads to a more homogeneous population wherein management is substantially evidence-based. However, including borderline abnormal EFs in PSF renders the terminology self-contradictory, because systolic function is normal in diastolic heart failure (2). Rather than imposing a definition, if the authors query the frequency distribution of EFs is there a bimodal curve? Where are the peaks; what are the distributions? Is there significant overlap? What percent fall into the 40% to 49% range? Are these patients similar to patients with systolic dysfunction; with diastolic heart failure (EFs $\geq$50%), such that the concept of normal left ventricular function needs to be revisited; or best considered in a gray zone, such that they cannot be placed into either group at this time?

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Effect of Door-to-Balloon Time on Patient Mortality

The study by McNamara et al. (1) from the National Registry of Myocardial Infarction (NRMI) found that door-to-balloon time (DBT) was strongly associated with mortality in both high- and low-risk patients and in patients presenting early or late after the onset of symptoms. These findings differ from our analyses from a large randomized trial and a single-center registry, both of which found that DBT impacts mortality primarily in high-risk patients and in those presenting early after the onset of symptoms (2,3).

Several possible explanations account for these differences. Prolonged DBT may be confounded with other unmeasured variables that impact mortality. First, DBT may be a surrogate for quality of care—hospitals with long DBTs may provide suboptimal treatment. Data from single-center registries and randomized trials would be less likely to have this bias. Second, NRMI data on time from symptom onset to presentation collected from retrospective chart reviews may be unreliable because the time of symptom onset is often not documented in hospital charts. This is less of a problem in randomized trials or carefully constructed prospective registries. Finally, and perhaps most importantly, prolonged DBTs often reflect the underlying severity of illness, with “sicker” patients requiring longer time for evaluation, stabilization, or treatment of complications prior to percutaneous coronary intervention (PCI) (e.g., cardiopulmonary resuscitation, intubation, defibrillation, or insertion of temporary pacemakers or intra-aortic balloon pumps). These confounding variables are rarely accounted for in large registries, including NRMI.

In addition, the findings by McNamara et al. (1) that DBT affects mortality even in patients presenting late contradict the widely held paradigm regarding the time-sensitivity of reperfusion therapy originally demonstrated by Reimer et al. (4) and recently re-emphasized by Gersh et al. (5).

This issue is more than academic. We believe that an excessive emphasis on minimizing DBT as the overriding quality-of-care measure by hospitals, insurers, and regulators (and guidelines committees) may at times detract from optimal patient care. Rushing to perform primary PCI before stabilizing unstable patients may lead to laboratory complications and worse clinical outcomes. Indiscriminant treatment with fibrinolytic therapy of ST-segment elevation myocardial infarction patients presenting at noninterventional hospitals, rather than transferring appropriate patients for primary PCI, deprives these patients of the benefits of higher rates of reperfusion, less reinfarction, less intracranial hemorrhage, and in many cases lower mortality. A recent meta-analysis of randomized trials with primary PCI versus fibrinolysis has shown primary PCI reduced mortality even with treatment delays up to 2 h (6). Decisions regarding triage of patients for primary angioplasty should thus be based on an assessment of time and risk, and should utilize common sense. High-risk patients presenting early after the onset of symptoms with long delays to primary PCI are probably best treated with fibrinolytic therapy. Most other patients are best treated with transfer for primary PCI despite longer treatment delays.

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