Syncope and Presyncope: Same Mechanism, Causes, and Concern

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Syncope is a symptom and not a diagnosis. Properly defined, it is the transient loss of consciousness with spontaneous recovery, resulting in a return to a preexisting neurologic condition. It is not surprising that when physicians use different definitions and outcomes, various studies become hard to interpret. Analysis with meta-analysis is further confusing.[1-4] It is great that international syncope researchers have gotten together to clearly define syncope, its outcomes, and priorities for management.[5,6] Clearly identifying the right patients has led and will continue to lead to research advances and improvements in the emergency department (ED) management of these patients.

The basic pathophysiologic mechanism for syncope is the same regardless of the cause, that is, the global hypoperfusion of the cerebral cortex or reticular activating center leading to a loss of consciousness. Most of the causes are benign such as orthostasis and reflex-mediated vagal syncope. Occasionally, serious causes such as a malignant cardiac arrhythmia can be a forerunner to sudden death. In fact, for patients with cardiac arrhythmia causing syncope it is like being dead, but they wake up. As physicians, we worry that the next time they may not be so lucky, and thus we struggle with syncope and its elusive serious causes in the ED. It is impossible for emergency physicians to diagnose with certainty the true causes of syncope, and thus we depend on risk stratification in our management paradigms.

Similar and even more confusing are the identification, definition, and outcomes of presyncope. This makes the importance of presyncope unclear in the literature.[7,8] It is likely accurate that true presyncope or near syncope has the same pathophysiologic mechanism as syncope, with the global hypoperfusion not being significant enough to cause complete loss of consciousness. If one believes the mechanism is the same, then it is not a stretch that the causes are the same. This means that individuals with malignant cardiac arrhythmia causing presyncope were almost dead but never realized it. Before a study in this month's *Annals* was published,[9] true presyncope suffered from being poorly defined, often being lumped in with general dizziness and chronically ill-defined "lightheadedness."[7] Some investigators include it in syncope research, whereas others have just excluded presyncope, given its elusive definition. In general, presyncope studies were small and their outcomes confusing. Because of this, one respected society has previously concluded that the literature for syncope cannot be applied to presyncope.[10] In addition, presyncope lacks the dramatic effect of the loss of consciousness, often raising less concern among patients, bystanders, and even physicians. This study shows we should be just as concerned with presyncope.

This study was the largest report on presyncope to date. The investigators clearly defined presyncope and then measured physician agreement to ensure patients enrolled had presyncope. Patients were followed up with the same standardized guidelines for reporting outcomes for syncope research, allowing one to make comparisons.[1,11,12] Compared with patients in similar studies on syncope, those with presyncope were slightly younger (aged 56 versus 59 years) and tended to have fewer comorbidities known to be associated with serious outcomes and syncope. In particular, patients with syncope had more existing heart disease, especially a history of congestive heart failure (3% versus 6%). Thus, it is not surprising that the absolute rates of serious outcomes are higher in patients with syncope. In fact, it may also be true that patients with certain preexisting conditions (such as congestive heart failure and a corresponding low ejection fraction) have syncope because they have little reserve to compensate and have a greater decrease in cerebral perfusion. At first glance, one may look at the overall serious event rate and see that patients with presyncope have about half as many serious outcomes (5% versus 10%) and be reassured that they are lower risk. However, the risk is not so low that presyncope can be dismissed, and a closer look shows that the most important outcomes (death and arrhythmia) are not infrequent among presyncope patients (2.6% versus 5.0%). Furthermore, physicians in this study had poor appreciation of the risk of presyncope compared with those in studies on syncope in which physician judgment was excellent when risk was predicted.[13]

The implications of this research should change our appreciation of presyncope patients in terms of its mechanism, its causes, and our level of concern. It does not tell us what the optimal management of these patients should be. The management of patients with syncope continues to be scrutinized as inefficient. To suggest that we just admit more of these patients is wrong. Reported admission rates for ED patients with syncope range from 30% to 85%[1,2,14] in the United States are approximately 50% in Europe,[15] 40% in Australia,[16] and 15% in Canada.[11] Our Canadian colleagues clearly admit the fewest patients with syncope and still discharge those with few serious outcomes.[17] It is unclear whether admission would have any effect on those outcomes. In reality, risk aversion, inpatient and

outpatient resources, and the availability of ED observation units drive overall admission rates. There is more use of ED observation units[18] and a trend to use more novel ambulatory monitoring devices,[19] making the use of admission as a proxy for appropriate management both problematic and irrelevant.

Finally, risk stratification plays a role in the eventual management and evaluation of patients with syncope and should be used for patients with presyncope as well. Most investigators included presyncope in the development of their risk stratification tools, and, given that presyncope likely has the same mechanism and causes, it is doubtful that a separate group of risk factors exists.[1] The validation of these risk factors in a large presyncope cohort and the possible identification of better risk factors is an area of potential research.

Presyncope presents with less drama and can be confused with ill-defined symptoms and diagnosis. However, with a careful history we can clearly define and identify these patients. Just like syncope research, this study shows the importance of identifying the right patients with consistent definitions and erases any doubt about the importance of near syncope or presyncope as a potential symptom of arrhythmia and sudden death.

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