CASE PRESENTATION

A 52-year-old woman was transferred from an outlying hospital where she had been hospitalized for a syncopal spell. She apparently had gone to the living room and became quite dizzy. She went to the kitchen and slumped over a cabinet, falling to the floor and striking her head sustaining an occipital laceration. She then vomited blood and was admitted to the hospital. A nasogastric tube was anchored and she was treated with blood replacement and found to have a peptic ulcer. When the nasogastric tube was removed, however, she developed an episode of vomiting with ventricular asystole, and, once again, nearly fainted. At that time, she was noted to have ventricular asystole with nonconducted P waves for a period up to 10 s. She was then transferred to the present hospital for further evaluation.

Her past history indicates that, on three previous occasions, she had experienced syncopal spells in the postoperative period following unrelated surgical procedures. She also relates a history of passing out associated with vomiting. For many years, she was aware of a heart murmur which had been considered to be produced by prolapse of the mitral valve which was hemodynamically insignificant. A Holter monitor had been done recently, and no apparent arrhythmia has been noted in a 24-h period.

Examination revealed normal vital signs with a blood pressure of 110/60 mm Hg, with no difference in the arms, and no significant drop on standing. No carotid bruits were noted. Carotid sinus stimulation revealed no significant slowing, and no symptom could be reproduced. The cardiac examination disclosed normal precordial pulsations. There was a high-pitched mid-to-late systolic murmur (grade 2/6) at the apex initiated by a mid-systolic nonejection click.

Laboratory evaluation revealed a hemoglobin value of 9.5 g/dl with a white blood cell count of 3,100/mm³. Resting 12-lead electrocardiogram showed normal findings. An echocardiogram disclosed thickening and prolapse of the mitral valve leaflets. Doppler examination disclosed a moderate degree of mitral regurgitation. The chest x-ray film findings were normal.

Questions for the consultants:

1. How are the syncopal episodes best explained? Could there be more than one mechanism involved? If so, do the mechanisms interrelate in any way?
2. What is the best approach to the diagnosis of this problem? At what point would studies such as treadmill testing, EEG, electrophysiologic testing, and Holter monitoring be used?
3. What place would tilt-table testing have? Under what circumstances would additional stimulation with isoproterenol be advisable? Discuss the technique and reasoning behind it, and the sensitivity and specificity of such testing.
4. If a tilt-table test result were “positive,” what is the mechanism for the result and how would this influence therapy?

Comments by George J. Klein, M.D.*

This 52-year-old woman presented with a syncopal spell and was found to be hypovolemic related to a bleeding ulcer. She again had a syncopal spell during her hospitalization related to vomiting during which a 10 s period of asystole due to AV block was noted. It is tempting to attribute both of these spells to “neurocardiogenic” or vasodepressor syncope. First, she had a history of previous episodes associated with vomiting and occurring after surgical procedures. Second, both hypovolemia associated with bleeding and vomiting would be acceptable precipitating causes of vasodepressor syncope. Third, she does not provide a history of organic heart disease, specifically previous myocardial infarction, that would increase the probability of other causes of syncope such as ventricular tachycardia. Alternatively, hypovolemia itself may cause syncope related to postural hypotension. In addition, vagal stimulation from vomiting could be itself cause AV block. Sinus bradycardia or sinus arrest is far more usual with intense vagal stimulation, and some degree of sinus slowing in addition to AV block should be apparent in order to invoke this etiology. Although the issue is not entirely clear, there appears to be some degree of overlap in the various “reflex” syncope, including that induced by vomiting, carotid sinus hypersensitivity, classical vasodepressor syncope, and so on, with the dominant feature being intense vagal discharge causing bradycardia and/or hypotension.

The diagnosis of unknown syncope is best guided by clinical features. Treadmill testing and EEG are not likely to be diagnostic unless the clinical history supports their use. Holter monitoring is rarely diagnostic unless the episodes of syncope are very frequent. Electrophysiologic testing is not likely to result in useful positive results in a patient such as this with a long-standing history of syncope and no

*Director, Arrhythmia Service, University Hospital; Professor of Medicine, University of Western Ontario, London, Canada.