An 84-Year-Old Woman with Syncope and Orthostatic Dizziness

15

Samuel L. Johnston, Jennifer J. Torgerson, Aleksandra Tosic, and Brian Olshansky

15.1 Case Summary

An 84-year-old woman was hospitalized following an apparent loss of consciousness and/or seizure. While seated, she experienced sudden dizziness and a sense of impending doom. The patient awoke feeling anxious and diaphoretic but, otherwise, without complaints. A family member noted convulsions that lasted for about one minute. The patient described a few similar episodes that had occurred in the past year. An extensive in-hospital work-up, including neurological assessment, was negative. The cause for her episodes was undetermined. Upon discharge, she was referred to the electrophysiology service for evaluation.

During her clinic visit, she recounted episodes of orthostatic dizziness. Indeed, rising from a seated position precipitated near-syncope. On evaluation, she was found to have orthostatic hypotension with no significant change in heart rate (supine BP 106/84 mmHg, HR 63 bpm; standing BP 84/66 mmHg, HR 67 bpm) consistent with neurogenic orthostatic hypotension. The patient was instructed to maintain better hydration, liberalize salt intake, and avoid rapid changes in posture. Her ECG is shown (Fig. 15.1). An echocardiogram was essentially normal. An ischemic evaluation (regadenoson myocardial perfusion study) showed nonreversible septal hypokinesis and a left ventricular ejection fraction of 50%. An implantable loop recorder (ILR) was placed due to the uncertainty of the cause for her spells.

S. L. Johnston (✉) · B. Olshansky
Department of Medicine / Division of Cardiology,
University of Iowa Hospitals and Clinics, Iowa City, IA, USA
e-mail: samuel-johnston@uiowa.edu; Brian-olshansky@uiowa.edu

J. J. Torgerson
Department of Cardiology, Covenant Medical Center,
Waterloo, IA, USA
e-mail: Jennifer.torgerson@mercyhealth.com

A. Tosic
Department of Cardiology, MercyOne Waterloo Heart Care Device Clinic IA, Waterloo, IA, USA
e-mail: Aleksandra.tosic@mercyhealth.com

What clues to the patient's diagnosis can be made from the history and physical exam? What can be inferred from the patient's ECG? Was it inappropriate to place an ILR at this point in the patient's work-up?

15.2 Case Discussion

Over 40 years ago, Kapoor [1] and others reported that, even with a careful and complete evaluation, the cause for syncope remained unknown in 40% of patients. Despite advances in knowledge and technology, the inability to quickly establish the cause for syncope remains frustratingly similar in the modern era [2]. Oftentimes, the evaluation is now even more elaborate and expensive, but is as misdirected and fruitless as it was then.

A carefully performed history and physical examination and an ECG remain the pillars of the evaluation to determine the cause for, and risk of adverse events related to syncope [3, 4]. Clinical assessment requires a precise understanding of the circumstances in which the episodes occur [5], as well as associated patient characteristics, complaints and underlying comorbid conditions [6]. This assessment is far more important than ordering a battery of tests. Only with this knowledge can the broad differential diagnosis be intelligently narrowed [1–4].

The clinical assessment helps direct appropriate testing and helps to determine where that evaluation should take place. As stated in the guidelines [3, 4], the evaluation is for risk assessment *and* diagnostic purposes. Certain risk factors portend a worse prognosis [4]. Age >60, palpitations, loss of consciousness without prodrome, syncope during exertion, syncope in the supine position, known heart disease and family history of premature sudden cardiac death suggest a cardiac cause for syncope and should be addressed explicitly in the cardiac electrophysiologist's documentation (creating a checklist within the electronic health record may facilitate this attention to detail) [7]. Cardiac syncope (Table 15.1) is associated with heightened risk of mortality or severe morbidity and sometimes necessitates in-hospital evaluation and treatment [3, 4].

A. Natale et al. (eds.), *Cardiac Electrophysiology*, https://doi.org/10.1007/978-3-030-28533-3_15

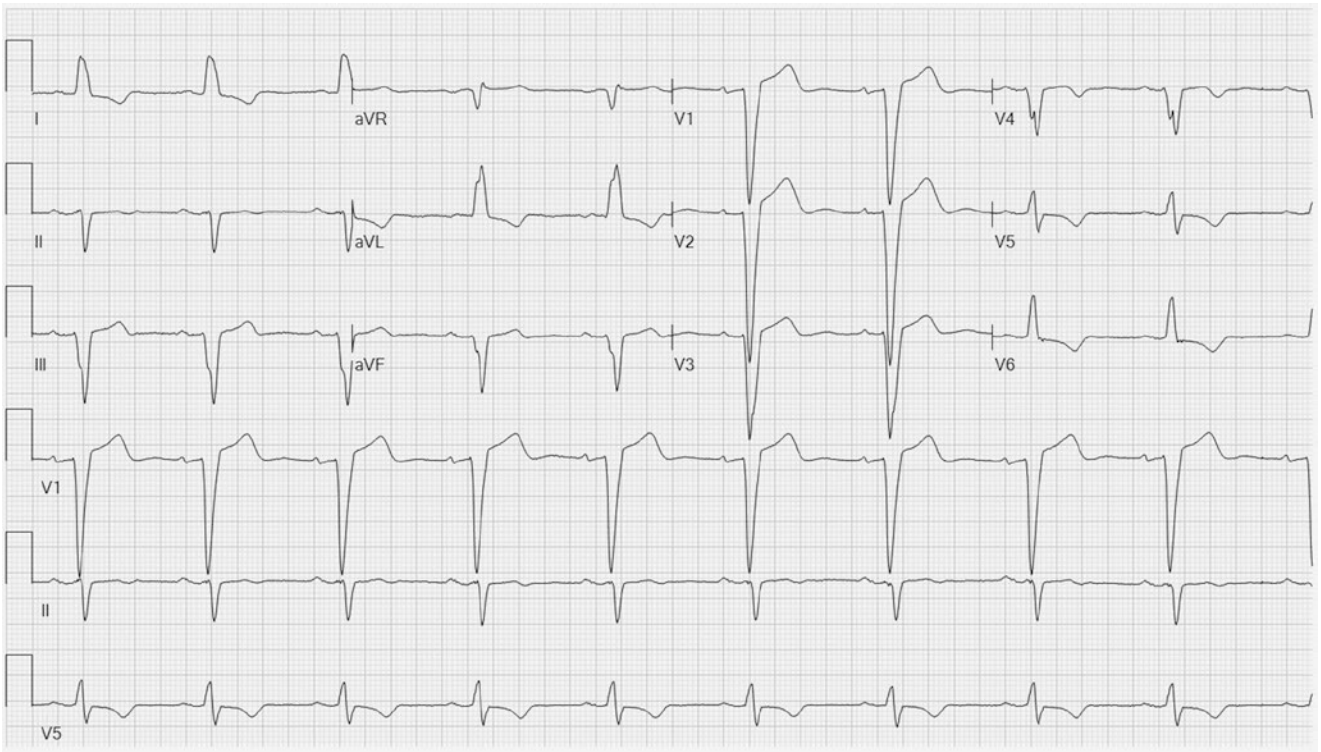

Fig. 15.1 Presenting ECG

Table 15.1 Cardiac causes for syncope

Cardiac causes for syncope	Mechanism
Arrhythmia	
– Bradyarrhythmia (often paroxysmal)	• Sinus node dysfunction (transient asystole) • Atrioventricular block (transient asystole)
– Tachyarrhythmia (often at onset)	• Supraventricular (hypotension) • Ventricular (hypotension)
Acute myocardial infarction or ischemia	• Tachy- or bradyarrhythmia • Bezold Jarisch reflex
Structural heart disease	
– Ischemic cardiomyopathy – Dilated cardiomyopathies – Congenital heart diseases – Infiltrative cardiomyopathies	• Transient positional hypotension—especially in the context of heart failure medications • Paroxysmal arrhythmias • Tachyarrhythmia (VT/VF) • Bradyarrhythmias (especially heart block) and tachyarrhythmias (AT/AF or VT/VF)
Obstructions to cardiac output	
– Aortic stenosis – Hypertrophic obstructive cardiomyopathy (HOCM) – Cardiac masses – Massive pulmonary embolism	• Obstruction per se • Reflex drop in heart rate and blood pressure
Acute pericardial tamponade	• Transient hypotension • Reflex circulatory collapse
Acute aortic dissection	• Tamponade • Coronary infarct or ischemia
Pulmonary hypertension	• Reflex circulatory collapse

Additional testing may include echocardiography, stress testing, and long-term heart rhythm monitoring. Echocardiography is usually the most appropriate initial screening test to assess the presence of structural heart disease. Stress testing is useful, not just to evaluate cardiac ischemia, but also to unmask potential arrhythmias, cardiac outflow tract obstruction, autonomic failure, pulmonary hypertension, and other diagnoses. Long-term heart rhythm monitoring (e.g., >1 year) using an ILR has revolutionized our ability to eventually diagnose the cause for syncope in patients who have infrequent events.

The electrophysiology study is less often helpful but may be of diagnostic value in older patients, patients with infarct-related scar but preserved left ventricular ejection fraction, and patients with other forms of structural heart disease. A carefully obtained history and physical examination are usually sufficient to secure the diagnosis of vasovagal syncope (VVS) or orthostatic intolerance, but head-up tilt table (HUTT) testing may be of use in equivocal cases [8]. Despite widespread use among some healthcare practitioners, carotid ultrasound, brain imaging (computed tomography, magnetic resonance imaging) and un-directed laboratory testing (BNP, troponin, stool Guaiac) are almost never helpful in the work-up of syncope [3, 4].

Shortly after placement of the implantable loop recorder, our 84-year-old woman had another episode of syncope, which was recorded on the ILR (Fig. 15.2). A dual-chamber pacemaker was implanted and, in long-term follow-up, syncope had resolved.

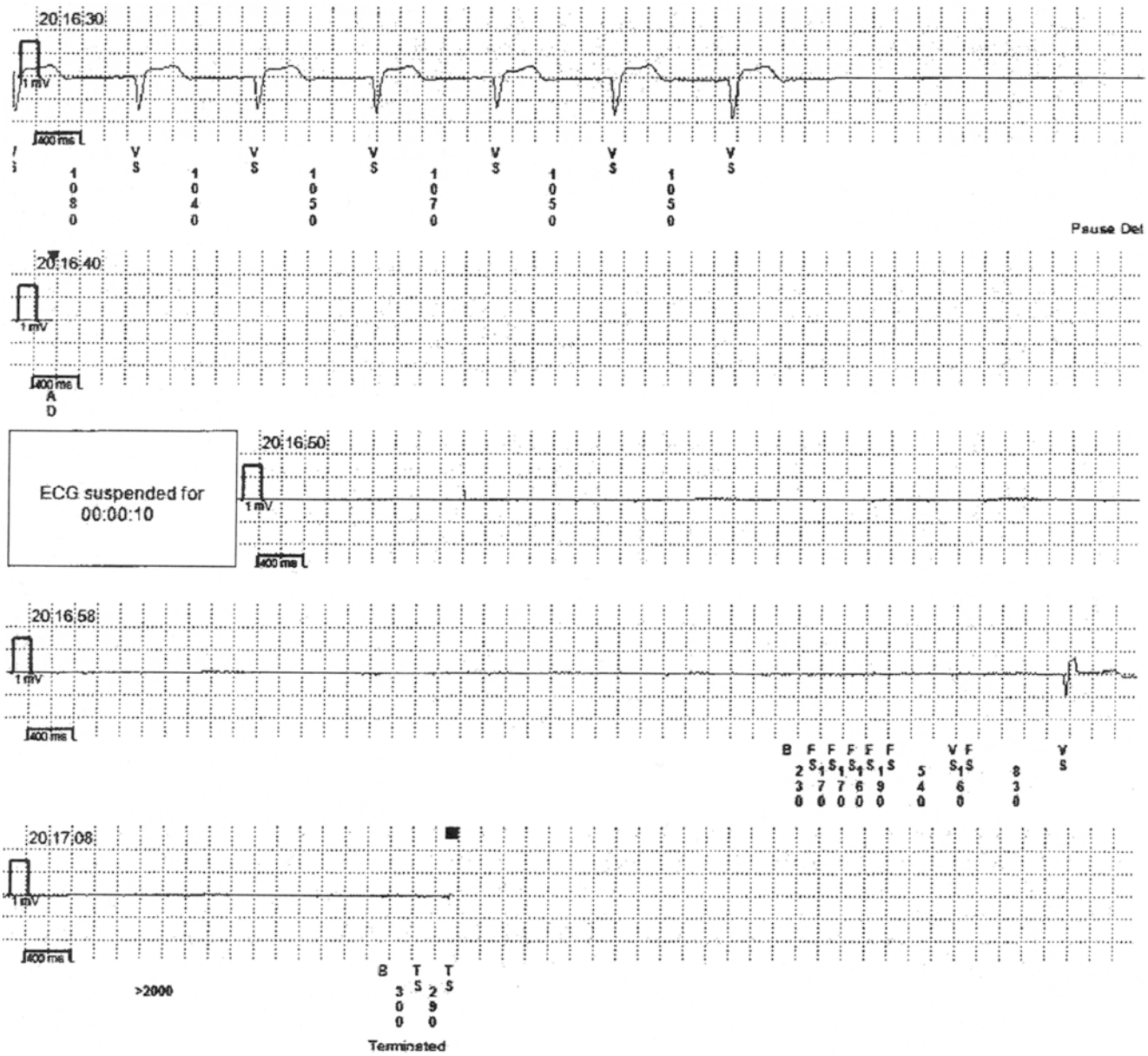

Fig. 15.2 Implanting loop recorder tracing corresponding to syncope

This patient exemplifies a common conundrum—particularly in elderly patients—with loss of consciousness; there are multiple potential explanations for why the patient may have passed out. Although this patient has orthostatic hypotension (a frequent cause for syncope in older patients), the clinically relevant cause was ultimately diagnosed as transient complete heart block. Had this patient undergone a HUTT test and had the results been positive for hypotension, then the premise that she was suffering from symptomatic orthostatic hypotension would have been incorrectly affirmed. In fact, the in-office physical examination had already shown symptomatic orthostatic hypotension. More importantly, the patient's history pointed to an alternative diagnosis, as her spells occurred while she was sitting or supine and without warning in a paroxysmal fashion.

The ECG can establish, but more often suggests (as in this case), the diagnosis. Sinus node dysfunction is suggested by sinus bradycardia, atrial enlargement criteria, non-sinus p wave morphology and frequent atrial ectopy. However, these clues are rarely directly actionable per se. Evidence of infra-Hisian conduction disease, such as left bundle branch block, is another matter.

The presence of left bundle branch block in this patient indicated infra-Hisian disease and suggested the possibility of intermittent complete heart block. Had an ambulatory 24-h (Holter) monitor or 30-day event monitor (EVM) been placed, it would have been unlikely to capture an event that occurred only a few times per year. She may have logged symptoms in her diary that occurred with postural changes that were not associated with a clinically significant change in heart rate or rhythm. As a result, short-term monitoring

would have been falsely reassuring. In contrast, the ILR recorded the patient's diagnosis. This patient illustrates the important principle that presence of one mechanism of syncope does not exclude the potential for other causes.

One may argue that it would have been appropriate to place a permanent pacemaker as the initial management strategy in this patient. Recent data from the SPRITELY Trial [9] (presented as a late-breaking trial at Heart Rhythm 2018) support this concept and support clinical decision making that has existed for many years [10]. In the SPRITELY Trial, patients with syncope and bi-fascicular block were randomized to receive either a permanent pacemaker or an ILR. Only 19/57 patients in the pacemaker arm had syncope during follow-up whereas 44/58 patients in ILR arm passed out ($P < 0.0001$).

These data were consistent with the PRESS Trial, demonstrating that, when compared to prolonged cardiac monitoring, an empiric pacemaker as first strategy is reasonable in patients with bi-fascicular block [11]. Ultimately, one's decision to monitor versus treat empirically with a pacemaker rests on clinical suspicion but, for a patient who has bi-fascicular block and no other plausible cause for recurrent syncope, transient asystole is the most likely explanation [12].

The apparent neurological sequelae (convulsions) in this patient were due to transient cerebral ischemia, rather than a seizure disorder. In many instances, patients, such as this, would get an extensive, expensive, and inappropriate neurological assessment.

References

1. Kapoor WN, Karpf M, Wieand S, Peterson JR, Levey GS. A prospective evaluation and follow-up of patients with syncope. N Engl J Med. 1983;309:197–204.
2. Joy PS, Kumar G, Olshansky B. Syncope: outcomes and conditions associated with hospitalization. Am J Med. 2017;130:699–706 e6.
3. Brignole M, Moya A, de Lange FJ, Deharo JC, Elliott PM, Fanciulli A, et al. 2018 ESC Guidelines for the diagnosis and management of syncope. Eur Heart J. 2018;39:1883–948.
4. Shen WK, Sheldon RS, Benditt DG, Cohen MI, Forman DE, Goldberger ZD, et al. 2017 ACC/AHA/HRS Guideline for the evaluation and management of patients with syncope: a report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines and the Heart Rhythm Society. Circulation. 2017;136:e60–e122.
5. Olshansky B. A Pepsi challenge. N Engl J Med. 1999;340:2006.
6. Goldschlager N, Epstein AE, Grubb BP, Olshansky B, Prystowsky E, Roberts WC, et al. Etiologic considerations in the patient with syncope and an apparently normal heart. Arch Intern Med. 2003;163:151–62.
7. Gawande A. The checklist manifesto: how to get things right. London: Profile Books; 2011.
8. Garcia-Civera R, Ruiz-Granell R, Morell-Cabedo S, Sanjuan-Manez R, Perez-Alcala F, Plancha E, Navarro A, et al. Selective use of diagnostic tests inpatients with syncope of unknown cause. J Am Coll Cardiol. 2003;41:787–90.
9. Krahn AD, Morillo CA, Kus T, Manns B, Rose S, Brignole M, et al. Empiric pacemaker compared with a monitoring strategy in patients with syncope and bifascicular conduction block—rationale and design of the Syncope: Pacing or Recording in ThE Later Years (SPRITELY) study. Europace. 2012;14:1044–8.
10. Kwoh CK, Beck JR, Pauker SG. Repeated syncope with negative diagnostic evaluation. To pace or not to pace? Med Decis Making. 1984;4:351–77.
11. Santini M, Castro A, Giada F, Ricci R, Inama G, Gaggioli G, et al. Prevention of syncope through permanent cardiac pacing in patients with bifascicular block and syncope of unexplained origin: the PRESS study. Circ Arrhythm Electrophysiol. 2013;6:101–7.
12. Kusumoto FM, Schoenfeld MH, Barrett C, Edgerton JR, Ellenbogen KA, Gold MR, et al. 2018 ACC/AHA/HRS Guideline on the evaluation and management of patients with bradycardia and cardiac conduction delay. Circulation. 2018. https://doi.org/10.1016/j.jacc.2018.10.043.