You're the Flight Surgeon

This article was prepared by Lance Nussbaum, D.O., M.P.H.

You're starting your afternoon clinic in the comforts of a climatecontrolled exam room after completing a lunch-hour workout under the blazing summer sun. Refreshed and ready to go, you greet a 32-yr-old male high-performance aircraft pilot who has concerns about an "episode" he experienced earlier that morning. He indicates that he was participating in his unit physical fitness training and had just run successive 400-m sprints when he suddenly felt lightheaded and fatigued near the end of his last sprint. He recalls sitting on a bench near the track and then passing out. A fellow airman was nearby and witnessed a 5- to 10-s period of loss of consciousness with no shaking or stiffening of the body. The pilot remembers awakening and having a vague memory of people asking him to recall his name and if he knew where he was. He went home after the incident, ate breakfast, drank some water, and then presented to the flight medicine clinic. At the time of exam, he is asymptomatic and feeling well. In reviewing his past medical history, the pilot noted a similar episode occurring approximately 1 yr ago after participating in a fun run, but he did not lose consciousness; he chalked the experience up to overexertion on a hot day. He denies any palpitations or chest pain and denies any personal or family history of seizures, diabetes, sudden cardiac death, or other neurological or cardiovascular diseases. Physical exam is normal and his electrocardiogram reveals sinus bradycardia.

1. Given the above information, what is the most likely reason for this pilot's syncopal "episodes"?

- A. Hypoglycemia secondary to not eating prior to exercising.
- B. Neurocardiogenic syncope leading to decreased cerebral perfusion.
- C. Seizure disorder (without convulsions) triggered by overexertion.
- D. Dehydration secondary to exercising in hot weather conditions.

ANSWER/DISCUSSION

1. B. Generally speaking, syncope is often brief, with spontaneous, complete recovery.⁵ The loss of postural tone, with subsequent horizontal positioning, helps quickly restore cranial blood flow. Men and

women of all ages are susceptible, but prevalence increases with age.⁵ Approximately 3% of emergency department visits and 1% of hospital admissions are related to syncope.¹

In stratifying the etiology of syncope, neurally mediated syncope accounts for approximately 45% of the cases, cardiac syncope accounts for approximately 20% of the cases, and postural hypotension accounts for about 15% of the cases.¹⁰ Approximately 15% of syncopal episodes have undetermined causes.⁵ Subtypes of neurally mediated syncope include vasovagal, situational, and carotid sinus hypersensitivity, and are all commonly referred to as "neurocardiogenic syncope."⁵ The primary mechanism for these subtypes involves the autonomic system driving a paradoxical parasympathetic response leading to bradycardia and/or hypotension.¹¹ Neurogenic syncope is typically marked by premonitory symptoms and lingering malaise after recovery.⁵ This is in contrast to true cardiac-related syncope, where loss of postural tone is usually abrupt with no associated lingering malaise.⁵

Mechanisms for cardiac syncope can be grouped into two broad categories: arrhythmias and structural heart disease with cardiac obstruction.⁵ Arrhythmias can stem from conduction disturbances (long QT syndrome, Wolff-Parkinson-White syndrome, Brugada syndrome, etc.) or from underlying structural heart disease (coronary arterial disease, hypertrophic cardiomyopathy, valvular disease, etc.). Obstruction-related heart disease, such as aortic stenosis, hypertrophic obstructive cardiomyopathy, or severe pulmonary arterial hypertension, leads to decreased cardiac output (due to ventricular outflow being fixed) during periods of vasodilation.⁵

Regardless of the category, cardiogenic syncope has an increased risk of sudden cardiac death, especially if the syncopal episode occurred during exertion. In fact, 1-yr mortality rates for cardiac-based syncope were 18-33%, compared to rates of 0-12% for noncardiac causes and 6% for unknown causes.⁸

Given that his squadron desperately needs instructor pilots, the pilot would like to resume his upgrade training to prevent any delays. He states he feels great and he will ease back on his fitness training to avoid any recurrences.

DOI: https://doi.org/10.3357/AMHP.5058.2018

2. What should be the next step in managing this pilot's condition?

- A. Keep him on flying status and have him return to his squadron to avoid any delays in his training program.
- B. Restrict him from flying duties for a 1-wk observation period and then consider return to flying duties if he remains asymptomatic.
- C. Restrict him from flying duties and place a cardiology referral for additional work-up.
- D. Restrict him from flying duties and place a neurology referral for additional work-up.

ANSWER/DISCUSSION

2. C. Although this pilot has only had two episodes in the past 2 yr, the potential for catastrophic injury related to passing out while flying is simply too high to keep the pilot on flying status. Unmonitored observation is unlikely to yield a true measure of recurrence risk given that the episodes occurred over a long period of time and did not occur with every exercise session. Ultimately, the pilot's episodes are suggestive of a cardiac etiology: a short period of loss of consciousness, symptoms occurring during exertion, and no prolonged disorientation after regaining consciousness. Therefore, referring the pilot to a cardiologist would be the appropriate next step.

To assess for arrhythmias, the pilot underwent event monitoring for 1 wk, which revealed one episode of supraventricular tachycardia (SVT) while exercising. Event monitors are good for capturing cardiac arrhythmias that occur on a less frequent basis (i.e., weekly to monthly). However, they have limited utility in the evaluation of syncope unless the device is continuously worn and has the capability of activating a recording automatically when symptoms arise.⁵

He also underwent exercise stress testing, which was electrically and clinically negative for ischemia or exertional arrhythmia. Exercise testing has a low diagnostic yield in patients with syncope unless the symptoms occur during maximal exertion. In these cases, the following observations could prove meaningful: 1) syncope reproduced during or immediately after exercise in the presence of electrocardiographic abnormalities or severe hypotension; 2) Mobitz type II second degree atrioventricular (AV) block or third degree AV block develops during exercise even without syncope, which is suggestive of a block distal to the AV node and predictive of progression to permanent AV block; or 3) failure to shorten the QT interval with exercise could indicate congenital long QT syndrome.⁶ Despite his symptoms occurring during maximal exertion, exercise stress testing was electrically and clinically negative.

To assess for structural abnormalities, an echocardiogram was accomplished, revealing a mildly enlarged right atrium and a right ventricular systolic pressure consistent with mild pulmonary hypertension (36 mmHg). Generally speaking, echocardiographic findings do not provide a specific causal diagnosis in syncope patients; however, certain findings, including, but not limited to, dilated or hypertrophic cardiomyopathy, severe valvular regurgitation or stenosis, and marked pulmonary arterial hypertension, can provide clues to the diagnostic cause.³ In addition to the cardiac work-up, there was no evidence of underlying pulmonary (thromboembolic) disease and all labs were within normal limits.

3. Given these findings, what should you do next?

- A. Electrophysiology (EP) study.
- B. Tilt-table testing.
- C. Carotid sinus massage.
- D. Magnetic resonance imaging and electroencephalography testing.

ANSWER/DISCUSSION

3. A. Although rarely indicated, EP studies are useful in selected patients with unexplained transient loss of consciousness/syncope in the presence of structural heart disease. EP study is also indicated with documented arrhythmia (SVT in this case) or when syncope occurs during exercise as 50% of conduction abnormalities can be occult.⁷

Head-up tilt-testing is largely used to confirm neurocardiogenic syncope in patients with intermediate pretest probability without structural heart disease. Sensitivity can be as high as 94%, but specificity is often low, especially if the patient is taking vasodilators or beta-blockers.^{5,11}

Carotid sinus syndrome is a rare cause of syncope that is most commonly associated with prior head/neck surgery, irradiation, or age greater than 60 yr old. To unmask carotid sinus hypersensitivity, carefully massage the neck at the angle of the mandible and look for a sinus pause greater than 3–5 s and/or a blood pressure fall greater than 50 mmHg with reproduction of symptoms. It is recommended to avoid this maneuver in patients with a history of transient ischemic attack or stroke within the past 3 mo and in patients with carotid bruits.¹³

Although neurologic disorders such as seizures can cause temporary loss of consciousness, neurologic testing (magnetic resonance imaging, electroencephalography, etc.) is rarely warranted in patients with true syncope. The 2009 European Society of Cardiologists' guidelines recommended neurologic referral in patients in whom transient loss of consciousness is suspected to be epilepsy rather than syncope.¹⁴

During his EP study, a concealed left-sided accessory pathway was found capable of retrograde conduction, supporting the notion that conduction abnormalities can be occult. His pathway was successfully ablated with follow-on electrocardiographs at 2, 3, and 4 mo being normal. He returned to full exercise with no recurrence of his palpitations or syncopal episodes.

4. What is the final aeromedical action/recommendation for this pilot?

- A. Submit a flying waiver in support of disqualification from flying status.
- B. Submit a flying waiver in support of returning to flying status after initiating an antiarrhythmic to help prevent the arrhythmia from recurring.
- C. Submit a flying waiver in support of returning to flying status with no additional therapies initiated.
- D. Return to flying status with no waiver, as arrhythmia no longer exists (it was ablated).

ANSWER/DISCUSSION

4. C. Aeromedical risks with concealed accessory pathways include risk of SVT, syncope, and even sudden cardiac death. However, ablation is considered curative with success rates greater than 95%.² The risk of recurrence of SVT and/or sudden incapacitation is estimated to be less than 1%.¹⁰

The U.S. Air Force medical standards indicate a flying waiver is required for two situations involving SVT: 1) SVTs unless successfully ablated, and not associated with structural heart disease; or 2) history of symptomatic or asymptomatic major dysrhythmias with or without ablation (of which SVT is considered a major dysrhythmia).^{*}

The U.S. Navy medical standards indicate a flying waiver is required for current or history of SVT, including those with coexisting mitral valve prolapse, left or right bundle branch block, mitral regurgitation and/or sarcoidosis, or any arrhythmia originating from the atrium or sinoatrial node, such as atrial flutter and atrial fibrillation, unless there has been no recurrence during the preceding 2 yr while off all medications. No evidence of coronary artery disease can be present.^{16,17}

The U.S. Army medical standards indicate a flying waiver is required for history of SVT unless there has been no recurrence during the preceding 2 yr while off all medications.¹⁵

Lastly, the Federal Aviation Administration medical standards would require a special issuance for this condition only if it required an implanted permanent pacemaker or there was a demonstrated clinically significant abnormality on electrocardiographic examination.⁴

In review of the various military standards, the pilot's underlying SVT would be disqualifying for Air Force, Navy, and Army flying duties. When compared to ablation therapies, antiarrhythmic medications for SVTs typically cost more and have a lower safety profile.¹² Furthermore, none of the antiarrhythmic medications are approved for flying duties due to their potential for blurred vision, fainting, dizziness, shortness of breath, chest pain, and worsening arrhythmias.9 Fortunately, the pilot does not require the use of any antiarrhythmic medication. Additionally, he does not have structural heart disease, has undergone successful ablation of his arrhythmia, and has had no recurrence of his exercise-related symptoms. Therefore, he does not pose a risk to his own safety or the safety of others while performing flying-related duties. All three branches of the military would consider a waiver for his history of SVT; in fact, a waiver was submitted on his behalf and, after approval, he returned to flying duties and has proven to be a great asset to his flying squadron.

Nussbaum L. You're the flight surgeon: loss of consciousness while exercising. Aerosp Med Hum Perform. 2018; 89(8):760–762.

ACKNOWLEDGMENTS

The author wishes to thank Lt. Col. Eddie Davenport, M.D., U.S. Air Force School of Aerospace Medicine, Chief Cardiologist, Internal Medicine Branch, Aeromedical Consultation Service, for his professional review of this article. The views expressed in this article are those of the author and do not necessarily reflect the official policy or position of the Air Force, the Department of Defense, or the U.S. Government.

REFERENCES

- Abi-Samra F, Maloney JD, Fouad-Tarazi FM, Castle LW. The usefulness of head-up tilt testing and hemodynamic investigations in the workup of syncope of unknown origin. Pacing Clin Electrophysiol. 1988; 11(8): 1202–1214.
- Calkins H, Yong P, Miller JM, Olshansky B, Carlson M, et al. Catheter ablation of accessory pathways, atrioventricular nodal reentrant tachycardia, and the atrioventricular junction: final results of a prospective, multicenter clinical trial. The Atakr Multicenter Investigators Group. Circulation. 1999; 99(2):262–270.
- Di Fusco SA, Colivicchi F, Nardi F. Cardiac imaging in patients with cardiac syncope. Minerva Cardioangiol. 2017; 65(6):579–588.
- Federal Aviation Administration. ITEM 36. Heart. In: Guide for aviation medical examiners. Washington (DC): Federal Aviation Administration; 2017:70–76.
- Hanna EB. Syncope: etiology and diagnostic approach. Cleve Clin J Med. 2014; 81(12):755–766.
- Kapoor WN. Evaluation and outcome of patients with syncope. Medicine (Baltimore). 1990; 69(3):160–175.
- Kapoor WN, Hammill SC, Gersh BJ. Diagnosis and natural history of syncope and the role of invasive electrophysiologic testing. Am J Cardiol. 1989; 63(11):730–734.
- 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(4):197–204.
- McEvoy GK, Snow EK. Antiarrhythmic agents, 24:04.04. In: AHFS drug information 2017. Bethesda (MD): American Society of Health-System Pharmacists; 2017:1775–1834.
- Pappone C, Vicedomini G, Manguso F, Saviano M, Baldi M, et al. Wolff-Parkinson-White syndrome in the era of catheter ablation: insights from a registry study of 2169 patients. Circulation. 2014; 130(10):811–819.
- Runser LA, Gauer RL, Houser A. Syncope: evaluation and differential diagnosis. Am Fam Physician. 2017; 95(5):303–312.
- 12. Schilling RJ. Which patient should be referred to an electrophysiologist: supraventricular tachycardia. Heart. 2002; 87(3):299–304.
- Shen WK, Sheldon RS, Benditt DG, Cohen MI, Forman DE, 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. J Am Coll Cardiol. 2017; 70(5): 620–663 [published erratum appears here:http://dx.doi.org/10.1016/j. jacc.2017.08.023.].
- 14. Task Force for the Diagnosis and Management of Syncope, European Society of Cardiology (ESC), European Heart Rhythm Association (EHRA), Heart Failure Association (HFA), Heart Rhythm Society (HRS), et al. Guidelines for the diagnosis and management of syncope (version 2009). Eur Heart J. 2009; 30(21):2631–2671.
- U.S. Army. 2-18. Heart. In: Standards of medical fitness. Washington (DC): Department of the Army; 2016:11. Army Regulation 40-501. [Accessed 1 Nov. 2017]. Available from https://armypubs.army.mil/epubs/DR_pubs/ DR_a/pdf/web/ARN3801_AR40-501_Web_FINAL.pdf.
- U.S. Navy, Bureau of Medicine and Surgery. Article 15-43. Heart. In: Manual of the Medical Department. Washington (DC): Department of the Navy; 2017. NAVMED P-117. [Accessed 1 Nov. 2017]. Available from http://www.med.navy.mil/directives/Documents/NAVMED%20 P-117%20(MANMED)/Chapter%2015%20Medical%20Examinations%20 (incorporates%20Changes%20126%20128%20135-140%20144%20 145%20147%20150-152%20154-156,159%20and%20160%20below).pdf.
- U.S. Navy, Naval Aerospace Medical Institute. 3.22. Supraventricular tachycardia. In: U.S. Navy aeromedical reference and waiver guide. Pensacola (FL): Naval Aerospace Medical Institute; 2016. [Accessed 1 Nov. 2017]. Available from http://www.med.navy.mil/sites/nmotc/nami/ arwg/Documents/WaiverGuide/Complete_Waiver_Guide.pdf.

^{*} U.S. Air Force. Section H: heart and vascular USAF medical standards, H8 & H9. In: Medical standards directory. 2017:21. [Accessed 1 Nov. 2017]. Available from https://kx2. afms.mil/kj/kx4/FlightMedicine/Documents/Medical%20Standards%20Directory%20 (MSD)/MSD%2020170529.pdf to those with access.