that these healthcare professionals may not be fully aware of the potential risks associated with oral anticoagulation or the particularly low risk of stroke in this population.

Jonathan C. Hsu, MD, MAS
Paul S. Chan, MD, MSc
Fengming Tang, MS
Thomas M. Maddox, MD, MSc
Gregory M. Marcus, MD, MAS

Author Affiliations: Cardiac Electrophysiology Section, Division of Cardiology, Department of Medicine, University of California, San Diego (Hsu); Mid America Heart Institute, Kansas City, Missouri (Chan, Tang); Department of Internal Medicine, University of Missouri, Kansas City (Chan); Veterans Affairs Eastern Colorado Health Care System, Denver (Maddox); Division of Cardiology, University of Colorado School of Medicine, Denver (Maddox); Cardiac Electrophysiology Section, Division of Cardiology, Department of Medicine, University of California, San Francisco (Marcus).

Corresponding Author: Gregory M. Marcus, MD, MAS, Cardiac Electrophysiology Section, Division of Cardiology, Department of Medicine, University of California, San Francisco, 505 Parnassus Ave, Room M188OB, San Francisco, CA 94143 (marcusg@medicine.ucsf.edu).

Published Online: April 13, 2015.

Author Contributions: Drs Hsu and Marcus had full access to all the data in the study and take responsibility for the integrity of the data and the accuracy of the data analysis.

Study concept and design: Hsu, Marcus.

Acquisition, analysis, or interpretation of data: Hsu, Chan, Tang, Maddox.

Drafting of the manuscript: Hsu, Marcus.

Critical revision of the manuscript for important intellectual content: Hsu, Chan, Tang, Maddox.

Statistical analysis: Tang.

Study supervision: Hsu, Marcus.

Conflict of Interest Disclosures: Dr Hsu reported receiving honoraria from Medtronic. Dr Chan reported receiving a Career Development Grant Award K23HL102224 from the National Heart, Lung, and Blood Institute. Dr Maddox reported receiving a Health Services Research and Development Career Development Award from the US Department of Veterans Affairs. Dr Marcus reported receiving research support from Medtronic and SentreHeart Inc. No other disclosures were reported.

Funding/Support: This research was supported by the American College of Cardiology Foundation's National Cardiovascular Data Registry (NCDR).

Role of the Funder/Sponsor: The PINNACLE (Practice Innovation and Clinical Excellence) Registry is an initiative of the American College of Cardiology Foundation. Bristol-Myers Squibb and Pfizer Inc are founding sponsors of the PINNACLE Registry. The PINNACLE Registry and the NCDR had no role in the design and conduct of the study and management, analysis, or interpretation of the data. The manuscript was approved with minor editorial suggestions by the PINNACLE Registry Research and Publications Committee prior to submission.

Disclaimer: The views expressed in this article represent those of the authors and do not necessarily represent the official views of the NCDR or its associated professional societies identified at http://www.ncdr.com.


LESS IS MORE

Unnecessary Hospitalization and Related Harm for Patients With Low-Risk Syncope

Testing in patients admitted for syncope rarely identifies an underlying cause.1-2 The San Francisco Syncope Rule (SFSR) was developed to identify patients with syncope at low risk for short-term serious outcomes who were unlikely to benefit from hospital admission.3 During hospitalization, many patients experience adverse events.4 Admission and testing can also lead to incidental findings of unclear clinical significance (“incidentalomas”) that can trigger clinical cascades of further testing.5 We performed a retrospective cohort study examining outcomes of patients who presented with low-risk syncope and were unnecessarily admitted to the hospital.

Methods | From January 1, 2010, to December 31, 2012, all patients with an International Classification of Disease, Ninth Revision (ICD-9) diagnosis of syncope (codes 780.2, 780.0, 458.0, or 780.4) were reviewed by internal medicine physicians (J.V.C., E.A., H.H., and C.K.). Hospitalizations were excluded if there was an obvious alternative reason for admission on presentation; the admission involved drug intoxication, alcohol intoxication, or trauma; patients were directly admitted or transferred; or determinants of SFSR were not available.

We focused on patients with SFSR ratings of 0 (systolic blood pressure >90 mm Hg on triage, hematocrit >30% [to convert to proportion of 1.0, multiply by 0.01], no history of congestive heart failure, no shortness of breath, and no nonsinus rhythm or new electrocardiographic changes) because they are at low risk for bad outcomes and likely did not require admission.3,6 Data abstraction included length of stay, laboratory testing, imaging testing, procedures, and consultations (Table 1). Adverse events were determined using the Institute for Healthcare Improvement tool.4 Reviewers also noted unexpected incidental findings during patient evaluation.

Descriptive statistics were computed using Microsoft Excel (Microsoft Corp). This study was approved by the institutional review board of the University of Maryland, Baltimore. The need for informed consent was waived by the institutional review board. Data were not deidentified because it would not have been possible to access medical records without the information.

Results | A total of 507 admissions were identified by ICD-9 codes, of which 213 met inclusion criteria. Of 213 admissions
with syncope, 72 of the admissions (34%) were for low-risk syncope. These patients have a mean 1.73-day length of stay and 10.8 tests. Clinical data are reported in Table 1. Eleven adverse events were identified in 9 admissions for low-risk syncope (13% [95% CI, 0.06-0.23]). Four of these adverse events were classified as serious and included delirium, transfusion error, hypoglycemia, and fall. Other adverse events included missed medication errors and complications from intravenous and urinary catheter placement.

Twenty-three patients (32% [95% CI, 0.22-0.44]) had incidental findings of unclear significance (Table 2). Of these, 3 patients (13%) had additional evaluation conducted during that inpatient visit, and an additional 11 patients (48%) were advised to have further evaluation performed as an outpatient.

Five patients (7% [95% CI, 0.01-0.13]) had potentially beneficial incidental findings resulting in treatment changes: dysautonomia, paroxysmal atrial fibrillation, inadequately treated hypothyroidism, obstructive hypertrophic cardiomyopathy, and urinary tract infection.

Discussion | Approximately one-third of patients admitted with syncope to a tertiary care center likely represented unnecessary admissions. Many patients underwent testing advised against by Choosing Wisely, including 88% with computed tomography of the head, 20% with magnetic resonance imaging of the head, and 25% with carotid Doppler ultrasonography. Of patients with low-risk syncope, 13% experienced an adverse event related to being in the hospital and 32% had an incidental finding of unclear significance. One-half of the patients with incidental findings required follow-up testing or evaluation, which is a burden for patients and potentially triggers other events. In contrast, 5 patients (7%) had clinically relevant unexpected findings identified that were treated.

This study is limited by its use of retrospective data from a single center and relying on the SFSR, some patients with an SFSR rating of 0 may require admission for nonmedical reasons. Strengths of this study include using the current criterion standard for adverse event identification.

Although previous studies of syncope have identified the lack of benefit of testing for low-risk syncope, to our knowledge, this is the first study demonstrating patient harm associated with hospitalization for low-risk syncope. Risk for adverse events and incidental findings should be considered before admitting patients with low-risk syncope.

Jenna VanLiere Canzoniero, MD, MS
Elham Afshar, MD
Helene Hedian, MD
Christina Koch, MD
Daniel J. Morgan, MD, MS

Author Affiliations: Department of Internal Medicine, School of Medicine, The Johns Hopkins University, Baltimore, Maryland (Canzoniero); Department of Internal Medicine, University of Maryland Medical Center, Baltimore (Afshar, Hedian, Koch); Department of Epidemiology, Veterans Affairs Maryland Healthcare System, Baltimore (Morgan); Department of Epidemiology and Public Health, School of Medicine, University of Maryland, Baltimore (Morgan).
Corresponding Author: Jenna VanLiere Canzoniero, MD, MS, Department of Internal Medicine, School of Medicine, Nelson 207, The Johns Hopkins University, 600 N Wolfe St, Baltimore, MD 21287 (lcanzonieri@jhmi.edu).

Conflict of Interest Disclosures: None reported.


Author Contributions: Dr Canzoniero had full access to all of the data in the study and takes responsibility for the integrity of the data and the accuracy of the data analysis.

Study concept and design: Canzoniero, Morgan.

Acquisition, analysis, or interpretation of data: All authors.

Drafting of the manuscript: Canzoniero, Morgan.

Critical revision of the manuscript for important intellectual content: All authors.

Statistical analysis: Canzoniero, Morgan.

Administrative, technical, or material support: Morgan.

Study supervision: Morgan.


COMMENT & RESPONSE

Corrections Regarding Prostate-Specific Antigen Editorial

To the Editor: There are a number of factual errors in the Editorial by Merenstein,1 to which I would like to offer the following corrections.

Merenstein1(p16) refers to prostate cancer as “the sixth leading cause of cancer death in men.” This is incorrect. Prostate cancer is the second leading cause of cancer death in men, second only to lung cancer. Here are the data, as presented on the Centers for Disease Control and Prevention website:1

Table: Leading Causes of Cancer Death Among Men

- Lung cancer (57.9%): First among men of all races....
- Prostate cancer (20.8%): Second among white, black, American Indian/Alaska Native, and Hispanic* men. Fourth among Asian/Pacific Islander men.
- Colorectal cancer (18.1%): Third among men of all races....

The numbers in parentheses are the death rates per 100,000 population. Because smoking is associated with over 90% of lung cancers, in men who have never smoked, prostate cancer is the number one cause of cancer death.

Merenstein1(p17) also misquotes critics of the US Preventive Services Task Force (USPSTF) as attacking that organization for having “no oncologists on committees.” However, oncologists treat prostate cancer after it has spread outside the prostate; they are not experienced in preventive or screening measures. The specialty of physicians with the deepest involvement and the most intense experience in performing early, curative, diagnosis and treatment of prostate cancer are urologists. The fact that no urologists were invited to serve on the USPSTF cost that committee a great deal of credibility among physicians. They did include a gynecologist and a pediatrician, neither of whom performs prostate examinations on adult male patients as an attending physician and so could not provide much experience or perspective in interpreting the randomized trials of prostate-specific antigen (PSA) screening.

The great biochemist who helped develop the PSA test, Richard J. Ablin, PhD, has stated that PSA should not be used for prostate cancer screening, but he appears to favor the use of PSA velocity, as in the following quotation: “More important than whether or not to screen is how one acts upon the data from a single test; with the exception of extremely high double- or triple-digit levels of PSA, it is prudent only to use a single PSA determination as a baseline, with biopsy and cancer treatment reserved for those with significant PSA changes over time [emphasis added], or for those with clinical manifestations mandating immediate therapy… absolute levels of PSA are rarely meaningful; it is the relative change in PSA levels over time that provides insight, but not definitive proof of a cancerous condition necessitating therapy.”

Major lack of adherence to the study protocols in the randomized trials of PSA screening falsely diminished the reported benefits of intervention. For example, in one European Randomised Study of Screening for Prostate Cancer (ERSPC) study center, 24% of the men invited to be screened never showed up to have even a single PSA test drawn, yet they were included in the intention-to-treat analysis. For a patient who is already sitting in your office with his arm bared for the phlebotomist, the benefits of PSA screening must therefore be higher than those reported by ERSPC. Similarly, the 52% of control patients in PLCO who received off-protocol PSA testing but were counted as not being screened also falsely attenuated the benefits of screening.

We are left with one important epidemiological observation: the introduction of widespread PSA screening in the United States was followed over the years by a 40% drop in prostate cancer deaths. This pattern has been observed in every country that has adopted widespread PSA screening, and not in countries (like England) that have restricted the use of PSA. Better randomized studies are needed, but until good science is available, I suggest that we practice the art of medicine by carefully monitoring and evaluating the changes in PSA levels of healthy men older than 50 years.

David L. Keller, MD

Corresponding Author: David L. Keller, MD, PO Box 14295, Torrance, CA 90503 (DavidLKellerMD@gmail.com).

Conflict of Interest Disclosures: None reported.
