non-respondents) reported high levels of satisfaction with continuing medical education (CME) opportunities offered in their departments. Fewer (995 of 1760 [57%]; 14 non-respondents) were satisfied with time and resources provided for CME, but those who were satisfied with both of these aspects of CME reported higher degrees of overall career satisfaction (odds ratio, 1.30). Previous studies have established the relationship between opportunities for professional development and career satisfaction among physicians.[2,3] For physicians, continuing education programs can be intellectually engaging, provide opportunities to connect with colleagues, and support their pursuit of excellent care for patients. However, without sufficient resources, CME requirements can be a burden. Together, our findings and those of Shanafelt et al introduce 3 specific actions related to continuing education that physician leaders can take today to improve physician well-being: (1) encourage (or even require) physicians to pursue educational opportunities and new skills,[1] (2) create time in physician schedules for such activities, and (3) provide resources (tuition and travel reimbursement or locally developed programs).

Recent attention to physician burnout is likely a reflection of rapidly changing times—payment reform, electronic medical records, and board certification requirements, for example. While leaders focus on guiding physicians through these tumultuous and distracting times, the surveys that we and Shanafelt et al conducted suggest that by paying closer attention to one of the oldest traditions of the medical profession—continuing education—leaders can help physicians weather the storm.

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Editor's Note: When publishing a letter that comments on an article published previously in *Mayo Clinic Proceedings*, it is the journal's policy to invite the author(s) of the referenced article to publish a response. Dr Tait Shanafelt was invited to respond, and although he was supportive of this letter, he felt the content of the letter did not require a reply.

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Nonictal Near Sudden Unexpected Death in Epilepsy

To the Editor: The presence of epileptiform discharges and seizures is routinely monitored by electroencephalography (EEG) during Wada testing. Spontaneous cardiac arrhythmias are unexpected.[1] Malignant cardiac arrhythmias, including asystole, are rare complications that occur in patients with focal seizures and have been implicated in sudden unexpected death in epilepsy (SUDEP).[2] We report a case of nonictal near-SUDEP.

Report of a Case. A 30-year-old man with drug-resistant, localization-related epilepsy underwent an evaluation for surgical treatment. Recurrent focal seizures with dyscognitive features and focal seizures evolving to convulsions had continued despite a trial of 4 antiseizure drugs. Video-EEG monitoring captured 3 left temporal seizures characterizing the localization during a presurgical evaluation. Electrocardiography (ECG) revealed normal sinus rhythm with sinus tachycardia during the seizures. An initial Wada test was invalid because of oversedation from amobarbital. During preparation for a repeated study and prior to catheterization, bradycardia and subsequent asystole occurred with generalized tonic stiffening that was first suspected to represent a seizure. Subsequent review of the ECG confirmed asystole causing convulsive syncope. A precordial thump prompted immediate resolution (after 64 seconds) without the need for antiarrhythmic medication. Outpatient Holter monitoring subsequently recorded a 33-second episode of spontaneous asystole. This episode resolved without clinical signs, and a permanent cardiac pacemaker was implanted without recurrence.

Discussion. This patient with drug-resistant focal seizures exhibited spontaneous cardiac asystole undetected during seizure monitoring. Had the Wada test not revealed in-hospital cardiac asystole, sudden cardiac death may have occurred without intervention. Cardiorespiratory disturbances are normally controlled by the autonomic nervous system and in many cases are associated with seizures.[3] A disturbed cerebral-cardiac relationship from brainstem dysregulation of cardiac sympathetic-parasympathetic activity may be the foundation for near death from a malignant arrhythmia in some patients with epilepsy. However, without EEG and ECG monitoring, it would have been difficult during Wada testing to conclude that the witnessed event was nonepileptic. An event misdiagnosed as an epileptic seizure would have been disastrous because of a missed treatment of a malignant cardiac arrhythmia.

Cardiac arrhythmias may account for a substantial number of cases of

SUDEP. The underlying pathophysiology is incompletely understood, although neurogenic cardiac asystole is believed to contribute to SUDEP.[4] SUDEP is reported to occur in about 1.8 per 1000 patients with epilepsy and is increased in patients who have persistent seizures despite antiseizure drugs.[4] Before we are able to prevent SUDEP,[5] we must learn the mechanism or mechanisms that underlie its occurrence. Rather than a single mechanism, our case supports observational findings of nonictal near-SUDEP as a mechanism that may occur independent of seizures. We support recommendations to maintain a heightened suspicion for spontaneous malignant arrhythmias in patients with uncontrolled seizures. Despite the absence of ECG abnormalities during ictal recordings, we recommend combined ECG and EEG monitoring during Wada testing and continued cardiac vigilance in patients with drug-resistant focal epilepsy.

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CORRECTION

In the Original Article entitled "**Risk of Acute Kidney Injury, Dialysis, and Mortality in Patients With Chronic Kidney Disease After Intravenous Contrast Material Exposure**" published in the August 2015 issue of *Mayo Clinic Proceedings* (*Mayo Clin Proc*. 2015;90(8):1046-1053), the middle initial for one of the authors was published incorrectly; his name should read David F. Kallmes.

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