Editors’ Note: In reference to “Postictal generalized EEG suppression: An inconsistent finding in people with multiple seizures,” Dr. Sethi draws attention to the likely multifactorial relationship between postictal generalized EEG suppression and sudden unexpected death in epilepsy. Authors Lamberts et al. respond. Drs. Lemmer et al. and authors Shellhaas and Barks discuss how the particular model of regional cerebral saturation sensor can affect the data obtained.

—Megan Alcauskas, MD, and Robert C. Griggs, MD

POSTICTAL GENERALIZED EEG SUPPRESSION: AN INCONSISTENT FINDING IN PEOPLE WITH MULTIPLE SEIZURES

Nitin K. Sethi, New York: Lamberts et al.1 reported on postictal generalized EEG suppression (PGES) inconsistency in patients with multiple convulsions. PGES following convulsive seizures occurs in some but not all patients.1,2 Patients with PGES are at higher risk for sudden unexpected death in epilepsy (SUDEP), especially if tonic-clonic seizures are uncontrolled; they are on antiepileptic drug (AED) polypharmacy; AED levels are subtherapeutic, indicative of noncompliance; they live alone; or they have coexisting cognitive deficits.2,3 While the link between PGES and SUDEP is intriguing, there are more variables to be considered than simply the duration or consistency of suppression on EEG after a convulsion. Accompanying cardiac autonomic variability and instability, presence or absence of postictal central apneas, and preexisting cardiac and pulmonary status determine which episode of PGES may lead to SUDEP.

Author Response: Robert J. Lamberts, Athanasios Gaitatzis, Josemir W. Sander, Christian E. Elger, Rainer Surges, Bonn, Germany; Roland D. Thijs, Heemstede, the Netherlands: The authors thank Dr. Sethi for his comments and agree that SUDEP is likely caused by the fatal coexistence of several predisposing and triggering factors.4 In most ictal recordings of SUDEP, PGES appears to be an EEG hallmark preceding cardiorespiratory arrest.5 Its mechanism is unclear, yet we believe that the value of PGES as a SUDEP risk marker is more complex than suggested by Dr. Sethi. PGES greater than 20 seconds after a convulsive seizure was associated with higher SUDEP risk, which increased proportionally with PGES duration.6 However, this association could not be confirmed in a larger study.7 Our finding of a high intraindividual variability of PGES may explain these conflicting results. PGES is not a reliable predictor of SUDEP, as the occurrence of PGES is critically dependent on the number of seizures recorded. Sleep and AED reduction appeared to facilitate the occurrence of PGES greater than 20 seconds. These findings together with previously reported facilitating cofactors, including peri-ictal hypoxemia,8 may help to unravel this complex but intriguing EEG hallmark of SUDEP pathophysiology.

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