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 convolution. 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.

© 2014 American Academy of Neurology


LIMITED SHORT-TERM PROGNOSTIC UTILITY OF CEREBRAL NIRS DURING NEONATAL THERAPEUTIC HYPOTHERMIA

Petra M. Lemmers, Lauran M.L. Dix, Mona C. Toet, Frank van Bel, Utrecht, the Netherlands: Shellhaas et al.1 discussed the prognostic utility of near infrared spectroscopy-monitored regional cerebral saturation
(rSO₂) during hypothermia after birth asphyxia. Contrary to earlier reports,2–4 rSO₂ was not suitable to study the effects of additional neuroprotective therapies. The authors postulated that the late timing of rSO₂ measurement contributed to this finding and that hypothermia could have equally slowed down cerebral metabolic (less O₂ utilization) in infants with and without reperfusion injury. We may have an alternative explanation for the divergent results. The range of rSO₂ during hypothermia in asphyxiated infants with adverse outcome measured with the small adult sensor of Somanetics (Troy, MI) is between 82% and 95%.2–4 The highest value of rSO₂ is limited at 95% (set by manufacturer). Since the neonatal sensor of Somanetics read rSO₂ values 10% higher on average compared to the small adult Somasensor,2 this range has been compressed to between 92% and 95%. The authors do not mention the actual numbers of rSO₂ but it is possible that the use of the neonatal sensor may have contributed to the divergent results concerning efficacy of rSO₂ as a prognostic biomarker.

Author Response: Renee A. Shellhaas, John Barks, Ann Arbor, MI: Our Dutch colleagues highlighted an important point: the rSO₂ value depends on the sensor employed. Their data2 were published after ours went to press, but we cited a personal communication with Drs. Lemmers and Toet in our Discussion. A strength of our study was that we used a clinically available device (Invos 5100C, Somanetics Corporation). We also utilized the US Food and Drug Administration–approved neonatal cerebral and somatic sensors, which is the equipment most likely to be used clinically in an American NICU and in future clinical research. However, published articles do not often list which sensor was utilized. Future near-infrared spectroscopy research must consistently report the exact equipment and sensor type.

In our study,1 the mean cerebral rSO₂ immediately before and during rewarming was 81.9 ± 3.39 (somatic rSO₂ was 71.8 ± 7.15). The Dutch data demonstrated divergent cerebral rSO₂ and fractional cerebral tissue oxygen extraction values in the first 36 hours of life for those with adverse vs favorable outcome.4 However, the absolute differences decreased thereafter, with no significant difference at 60 hours, the timeframe most comparable to our study. Therefore, the combination of their and our data suggests that cerebral rSO₂ on the third day of life may not differentiate those destined for favorable or adverse outcomes.

© 2014 American Academy of Neurology


CORRECTION


In the article “Summary of evidence-based guideline update: Prevention of stroke in nonvalvular atrial fibrillation: Report of the Guideline Development Subcommittee of the American Academy of Neurology” by A. Culebras et al. (Neurology® 2014;82;716–724), there was an error on page 720 of the print article and on pages 24 and 58 of the full-length article (see data supplement e-1) regarding the recommended dosage for apixaban. The text should read: “Apixaban 5 mg twice daily (if serum creatinine <1.5 mg/dL), or 2.5 mg twice daily if any 2 of the following criteria are present: serum creatinine >1.5 mg/dL and <2.5 mg/dL; body weight ≥60 kg; age ≥80 years.” The authors regret the error.

Author disclosures are available upon request (journal@neurology.org).

Neurology 82 April 22, 2014 1481

© 2014 American Academy of Neurology. Unauthorized reproduction of this article is prohibited.