Editors' Note: Rosenfield comments that Lu et al. may have been misled by the brain imaging findings in their study on people who stutter, especially when considering that stuttering is a response to an underlying abnormality and not the abnormality itself. Meyer agrees with the results of Poh et al. in their study of autonomic changes in seizures and discusses additional data that further explain the potential pathophysiology of sudden unexpected death in epilepsy. Ciceri expands on the hypothetical role of venous engorgement in the pathophysiology of Hirayama disease.

Chafic Karam, MD, and Robert C. Griggs, MD

NEURAL ANOMALY AND REORGANIZATION IN SPEAKERS WHO STUTTER: A SHORT-TERM INTERVENTION STUDY

David B. Rosenfield, Houston: The article by Lu et al.¹ and accompanying editorial by Dr. Kell² documented successful therapy for people who stutter by examining normalized brain imaging connectivity between the speech network and the supplementary motor area. They also examined decreased resting-state functional connectivity with the midline cerebellum.

The authors and the editorialist also studied brain activity at rest (not speaking) and provide insight into what might transpire in brain activity during a task-dependent state (speaking, fluently or otherwise). The obfuscating factor is that if stuttering is a response to an underlying abnormality and not the abnormality itself, then the meaning of the data should be questioned.

For example, it is possible that stuttering is the speaker's response to an instability in an altered speech-motor-auditory feedback loop.^{3,4} Improvement of stuttering behavior and output may be a valid finding, but only if improvement is a response to the underlying deficit causing stuttering and may not directly reflect any improvement in the deficit. The "core" of the stuttered response remains and may have nothing to do with the changes in imaging at rest, during speech, or following therapy.

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AUTONOMIC CHANGES WITH SEIZURES CORRELATE WITH POSTICTAL EEG SUPPRESSION

Sascha Meyer, Homburg; Matthias Strittmatter, Merzig, Germany: In their study on potential contributing factors for sudden unexpected death in epilepsy (SUDEP), Poh et al.¹ demonstrated that the magnitude of both sympathetic activation and parasympathetic suppression increased with duration of EEG suppression after tonic-clonic seizures.

Their findings suggest autonomic dysfunction as a pathophysiologic correlate of postictal EEG suppression, which may be vital to the concept of SUDEP. We agree with this concept but would like to add further data. It is likely that one of the most important mechanisms of SUDEP is cardiac arrhythmia precipitated by seizure discharge acting via the autonomic nervous system.

Generators of autonomic dysbalance include the insula, anterior cingulate gyrus, and ventromedial prefrontal cortex.² Previous studies have demonstrated hemispheric lateralization in the control of the central autonomic nervous system, which is in large part mediated by the right insular cortex.² In addition, cardiac autonomic control is under particular control of the right cerebral hemisphere.^{3,4} Moreover, clinical studies have also shown that profound desaturations below 90% were significantly correlated with hemispheric lateralization in localization-related epilepsy (odds ratio right vs left = 2.098; 95% confidence interval 1.078–4.085).⁵

The knowledge about lateralization of the anatomy and pathophysiology of the central autonomic nervous system may contribute to a better understanding of SUDEP and has the potential to detect patients who may be particularly susceptible to this devastating clinical entity.

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David B. Rosenfield

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