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Quantitative electroencephalography characteristics of tilt-induced neurally-mediated syncope among youth

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HIGHLIGHTS

- Tilt-induced syncope is associated with changes in several quantitative EEG (QEEG) parameters.
- QEEG changes include a shift in power from higher to lower frequency ranges.
- Laterality of QEEG changes is not different from a probability of 0.5.

ABSTRACT

Objective: To characterize the quantitative electroencephalographic (QEEG) patterns associated with tilt-induced syncope in youth.
Methods: Several QEEG parameters were analyzed. Data were calculated for peak or nadir changes with syncope for amplitude-EEG, fast Fourier transform (FFT) power in several frequency ranges, 8–13 Hz/1–4 Hz frequency ratio, and FFT edge.
Results: Changes in QEEG parameters were present among all patients with tilt-induced syncope (n = 76). These changes included increases in the low frequency FFT power (1–4 Hz range), decreases in the power ratio (8–13 Hz/1–4 Hz) and decreases in the FFT edge (95%, 1–18 Hz). All patients had suppression of EEG amplitudes that closely followed loss of consciousness. Asymmetry indices demonstrated cerebral hemisphere lateralization at multiple periods during the evolution of syncope, but the side of lateralization did not differ from 0.5 probability.
Conclusions: QEEG parameters can be used to characterize EEG changes associated with tilt-induced, neurally-mediated syncope.
Significance: QEEG may serve as a useful tool for the study of syncope neurophysiology, and the modeling of changes with syncope may improve our understanding of other neurologic disorders caused by defects in cerebral perfusion.

1. Introduction

Syncope is defined as a transient loss of consciousness resulting from global cerebral hypoperfusion and characterized by rapid onset, short duration, and spontaneous and complete recovery (Freeman et al., 2011;Moya et al., 2009;Shen et al., 2017;Brignole et al., 2018). Neurally-mediated syncope is associated with activation of the autonomic nervous system, impaired vasoconstriction and increased vagal tone, causing hypotension, variable degrees of bradycardia, and, ultimately, diminished cerebral perfusion (van Dijk et al., 2009; Freeman et al., 2011). Neurally-mediated syncope is common among young individuals, with estimated rates as high as 30–40% (Ganzeboom et al., 2003;Serletis et al., 2006), yet the specific neural pathways that cause syncope are not fully understood.

Electroencephalography (EEG) provides a real-time assessment of the function of cortical neurons during syncope. Two EEG

Abbreviations: EEG, electroencephalography; QEEG, quantitative EEG; FFT, fast Fourier transform; aEEG, amplitude-EEG; EASI, EEG asymmetry index; REASI, relative EEG asymmetry index.

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patterns have been described for syncope induced during tilt-table testing: a slow-flat-slow pattern and a slow-only pattern (van Dijk et al., 2014;Heyer et al., 2016;Ammirati et al., 1998;KARP et al., 1961;Grubb et al., 1991;Ladwig et al., 1997;Sheldon et al., 1998; Mercader et al., 2002). The difference between patterns appears to reflect the degree of impaired cerebral perfusion during the syncope event (Wieling et al., 2009). With the slow-flat-slow pattern, the EEG flattens when cerebral blood flow drops below a critical value (Astrup et al., 1981). Syncope episodes that result in greater deficits in cerebral blood flow cause EEG flattening at the nadir of perfusion (Wieling et al., 2009). High-amplitude slowing of EEG frequencies occurs as hypotension exceeds the autoregulatory capacity of the cerebral vasculature, followed by flattening and then a second period of slowing during recovery, producing the slow-flat-slow pattern (Wieling et al., 2009). Most patients with a slow-flat-slow EEG pattern also have corresponding asystole, although asystole is not necessary (Heyer et al., 2016;van Dijk et al., 2014;Grubb et al., 1991;Sheldon et al., 1998). The slow-only EEG pattern reflects a hypotensive response with syncope that causes impaired cerebral perfusion and loss of consciousness, but the threshold for EEG flattening is not met (Wieling et al., 2009).

Regardless of whether the EEG pattern is slow-only or slow-flat-slow, the build-up of high-amplitude delta slowing typically precedes signs of loss of consciousness (Heyer et al., 2016). Myoclonus occurs during periods of EEG slowing when some degree of cortical activity is present (van Dijk et al., 2014). When the slow-flat-slow pattern is present, tonic posturing may evolve during the flat phase (Shmuely et al., 2018). Presumably, a brainstem pathway generates the tonic posturing response. A previous study demonstrated left cerebral hemisphere lateralization during the initial phase of EEG slowing in 5 out of 6 patients with tilt-induced syncope or near-syncope (Mercader et al., 2002), but the finding could not be reproduced from a study with a larger patient cohort (Heyer et al., 2016). Few reports are available documenting quantitative EEG (QEEG) changes with syncope (Mercader et al., 2002;Moeller et al., 2011). Most studies of the EEG correlates of syncope use visual analysis of the raw EEG only. An analysis of multiple QEEG parameters characterizing the associated changes with tilt-induced, neurally-mediated syncope from a large cohort has not yet been published.

Accordingly, in the present study we explored which QEEG parameters demonstrated informative changes in EEG frequencies, amplitudes, and laterality during tilt-induced syncope from young patients without neurologic impairments. QEEG amplitude and frequency changes were calculated during the tilted baseline and during tilt-induced syncope. Certain parameters were characterized in relation to raw EEG changes, signs signifying the onset of loss of consciousness, and timing of tilt-table lowering. We hypothesized (A) that changes in several QEEG parameters would be discernable during the evolution of syncope and (B) that EEG lateralization with syncope would not differ between cerebral hemispheres. Understanding how the EEG changes in relation to hypotension and syncope may help to elucidate the neural pathways that produce the syncope response. QEEG may be particularly helpful as a complementary tool in studies of syncope neurophysiology. Moreover, the QEEG changes with syncope may be useful in modeling the neurophysiologic changes that occur with other neurologic disorders caused by defects in cerebral perfusion.

2. Patients and Methods

2.1. Subjects

From January 2015 to June 2017, we conducted a cross-sectional study of sequential patients with neurally-mediated syncope during head-upright tilt-table testing with concurrent video-EEG. All patients had normal cardiac evaluations (minimally including 12-lead electrocardiograms and normal cardiac exams) prior to testing. Patients were included in the study if tilt-induced syncope occurred without the use of medicine provocation. Loss of consciousness with syncope was confirmed by (A) the presence of at least two clinical signs (eye opening when initially closed, eye deviation, loss of postural tone, myoclonus, or tonic posturing) and by (B) the patient report of perceived loss of consciousness upon recovery. Patients were excluded from study if loss of consciousness could not be confirmed, if the timing of loss of consciousness could not be established by video review with 1-s resolution, or if artifact or equipment malfunction prevented accurate interpretation of any EEG parameters. Additional exclusion criteria were prior seizures, baseline EEG abnormalities, developmental delays requiring academic accommodations, neurologic impairment suggestive of brain lesion, known brain injury demonstrated by prior neuroimaging, known history of traumatic brain injury, or prenatal or perinatal brain injury. None of the patients included in this study met diagnostic criteria for neurogenic orthostatic hypotension or postural tachycardia syndrome (POTS). The clinical indications for tilt-table testing included refractory syncope, transient loss of consciousness of unknown etiology, and frequent symptoms of orthostatic intolerance. Age was not a recruitment factor; all patients referred to our pediatric clinic who met study criteria were eligible.

The study was approved by the Institutional Review Board at Nationwide Children's Hospital. Written informed consent (parents and patients $\geq$18 years of age) and assent (patients 9–17 years) were obtained prior to all testing.

2.2. Definitions

Tilt-induced syncope was defined as a transient loss of consciousness associated with hypotension, with or without bradycardia, and with typical corresponding raw EEG changes (van Dijk et al., 2014; Heyer et al., 2016). As described above, all patients had clinical signs and reported the perception of loss of consciousness with their syncope events. Asystole was defined as an interval $\geq$3 s between QRS complexes. The onset of asystole was determined by the timing of the last QRS complex preceding the $\geq$3-s interval.

2.3. Protocol

All medicines that can affect orthostatic tolerance were discontinued $\geq$5 half-lives prior to testing. EEG electrodes were placed according to the International 10–20 system; 21 EEG channels plus two EMG and one ECG channels were recorded. Video EEG (Comet AS-40, GRASS systems, Warwick, Rhode Island, USA) was synchronized with continuous heart rate and blood pressure (Portapres, Finapres Medical Systems, Amsterdam, Netherlands) monitoring at baseline and during tilt testing. The EEG video camera was mounted on the ceiling and focused on the patient during tilt. Since the static video system can miss subtle signs of loss of consciousness during table lowering, a separate high-resolution video camera was attached by a welded bar to the tilt table, overlooking the patient, and moving up and down as the table was raised and lowered. Following at least 30 min of recumbency patients were tilted upright, 70 degrees. They were lowered with syncope, upon request with intolerable symptoms, or after 45 min. The raw video-EEG was reviewed offline to determine onset of delta slowing, estimated timing of loss of consciousness (1-s resolution), and timing of initial table lowering. The table-mounted video was used to confirm signs of loss of consciousness when $\geq$2 signs were not present on the ceiling-mounted video. The table-mounted

video was particularly helpful in confirming eye opening and eye deviation. No patient in this study had closed eyes during loss of consciousness. Table lowering takes approximately 12 s. Loss of consciousness occurred prior to table lowering or during the first four seconds of table lowering in all patients.

2.4. Quantitative EEG parameters, settings, and potential EEG artifacts

The raw EEGs were converted to QEEG files using Persyst 13 software [Persyst, Solana Beach, California, USA]. Several QEEG parameters were explored. Each study parameter was chosen based on its consistency across QEEG samples and its relevance in characterizing syncope. All parameters were measured independently from the left and right cerebral hemispheres. The QEEG parameters included amplitude-EEG (aEEG) in microvolts, the EEG asymmetry index (EASI), the relative EEG asymmetry index (REASI), the fast Fourier transform (FFT) power in the 1–4 Hz, 4–8 Hz, and 8–13 Hz frequency bands, the FFT power ratio (8–13 Hz/1–4 Hz), and the FFT edge (95%, 1–18 Hz). The following FFT parameter settings were used for all samples: sampling rate = 128 Hz; FFT points per window = 128; window duration = 1 s; epoch duration = 1 second; windows per epoch = 1; smoothing = 1; and the step command set at 1 second. Since movements occur (head drops, blinking, eye deviation, tonic posturing, etc.) in association with tilt-induced syncope, potential artifacts are unavoidable. The raw EEG tracing and several QEEG parameters were inspected to determine the presence of artifact. When artifact was present, but did not interfere with analyses, the sample was retained.

2.5. Quantitative EEG parameters that characterize EEG slowing and amplitude changes

The FFT analyses provide the relative contribution of a given EEG frequency range across the power spectrum. As hypotension progresses, the composition of EEG frequencies slows, producing increases in the power of the 1–4 Hz and 4–8 Hz EEG frequencies and a decrease in higher frequency ranges, resulting in a lower FFT (8–13 Hz/1–4 Hz) ratio. The FFT edge, with settings of 95% and 1–18 Hz, determines the EEG frequency below which 95% of the specified frequency range is present. During the evolution of syncope, the FFT edge decreases, reflecting lower total frequencies within 95% of the 1–18 Hz frequency range. The aEEG spectrogram plots the amplitude characteristics (0–100 microvolts) of the filtered and rectified representation of the EEG over of time.

2.6. Quantitative EEG parameters that characterize cerebral laterality

The EASI and REASI compare absolute and relative differences in the FFT power spectrum between homologous left- and right-EEG electrodes for a given time period. The frequency range of 1–18 Hz was used for all asymmetry analyses. The EASI increases with increasing differences in amplitudes between homologous electrodes, providing a quantitative measure of the degree of asymmetry between cerebral hemispheres. The REASI provides the laterality with a deflection upward for right hemisphere-weighted asymmetry and a deflection downward for left-weighted asymmetry. Baseline 4-s EASI epochs, chosen at 90–120 s following tilt upright, prior to the onset of hypotension in all cases, were compared to 4-s epochs beginning at the onset of QEEG slowing with syncope. All epochs were chosen to exclude eye-blinking artifacts and other movement artifacts. The REASI measures were chosen for three time points during syncope: the initial decrease in the FFT ratio (8–13 Hz/1–4 Hz), the peak of FFT power in the 1–4 Hz frequency range, and the resolution of the FFT ratio (8–13 Hz/1–4 Hz) changes. Each REASI value was designated as lateralizing to the right cerebral hemisphere or to the left cerebral hemisphere, and laterality was compared as a binomial variable for each time point (see statistical analysis below).

2.7. Statistical analysis

Descriptive statistics were calculated for the total duration of QEEG changes. The onset of QEEG slowing with syncope was determined by initial changes in FFT power for the 1–4 Hz frequency range, the 8–13 Hz/1–4 Hz ratio, and the edge parameters. The Student's t-test was used to compare periods from onset of QEEG slowing to the first sign of loss of consciousness between patients with and without asystole. Four-second epochs were chosen at a period of initial hemodynamic stability, 90–120 s following tilt upright, to represent the mean baseline values for each FFT parameter. The paired-samples t-test was used to compare mean FFT values at baseline to point values (selected as peak or nadir values) at syncope. All epochs were chosen to exclude eye-blinking and other movement artifacts. As described above, there was a decrement in EEG amplitudes (measured from aEEG) that followed clinical signs of loss of consciousness in all patients. Timing of the onset of this decrement was calculated relative to the timing of loss of consciousness and table lowering.

The paired-samples t-test was used to compare quantitative differences in EASI epochs at baseline and syncope onset. The binomial test was used to compare right- and left-hemisphere lateralization on REASI against the probability of 0.5 for each designated syncope epoch. Since the initial publication suggesting left cerebral hemisphere laterality demonstrated changes in 5 out of 6 patients (Mercader et al., 2002), our much larger sample size should be adequate to counter those previous findings if no differences in laterality were present.

Data are presented as mean ± standard deviation. Statistical analyses were performed using SPSS Version 25 (SPSS Inc., Chicago, IL, USA). All relevant analyses were performed as 2-tailed. The significance threshold was set at 5%.

3. Results

During the study period 177 patients developed tilt-induced syncope or presyncope: 61 were excluded because loss of consciousness could not be confirmed by video; 22 were excluded because they had disconnection of at least one EEG lead during syncope or other equipment malfunction that prevented analysis; and 18 were excluded because of movement artifacts that prevented analyses of at least one of the studied EEG parameters. EEGs were analyzed from the remaining 76 patients with tilt-induced syncope and minimal movement artifact. Age in the study cohort ranged from 5-23 years (mean 15.1 ± 2.7 years); 58 patients (76.3%) were female. The average time to tilt-induced syncope was 1166 ± 624 s. Twenty-four patients (31.6%) had asystole during syncope.

3.1. Onset and duration of QEEG changes with syncope

Three QEEG parameters changed consistently with syncope onset and remained changed through syncope resolution: the FFT power in the 1–4 Hz frequency range, the FFT power ratio (8–13 Hz/1–4 Hz), and the FFT edge (95%, 1–18 Hz). Table 1 lists values of QEEG parameter changes with syncope for the peak aEEG, the FFT powers with common EEG frequencies (1–4 Hz, 4–8 Hz, 8–13 Hz), the nadir FFT power ratio (8–13 Hz/1–4 Hz), and the nadir FFT edge (95%, 1–18 Hz). Fig. 1 illustrates each QEEG parameter change with a slow-flat-slow syncope pattern. Among the 24 patients with asystole, 14 had QEEG slowing prior to the onset of

Table 1
Comparison of quantitative EEG parameters between tilted baseline and syncope.

Quantitative EEG parameter	Baseline values (mean ± SD)	Values with syncope (mean ± SD)	Range of values with syncope, n = 76	P values
aEEG peak, left hemisphere (microvolts)*	Not assessed	37 ± 13	20–89	Not assessed
aEEG peak, right hemisphere (microvolts)*	Not assessed	38 ± 14	18–94	Not assessed
Peak FFT power (1–4 Hz), left hemisphere	3.5 ± 1.3	34.2 ± 11.3	17.2–72.7	<0.001
Peak FFT power (1–4 Hz), right hemisphere	3.5 ± 1.4	35.8 ± 13.3	16.6–84	<0.001
Peak FFT power (4–8 Hz), left hemisphere	1.4 ± 0.5	10.1 ± 3.3	4.2–19.5	<0.001
Peak FFT power (4–8 Hz), right hemisphere	1.4 ± 0.5	10.3 ± 3.3	4.8–20.3	<0.001
Peak FFT power (8–13 Hz), left hemisphere	1.2 ± 0.4	3 ± 1.2	1.5–6.6	<0.001
Peak FFT power (8–13 Hz), right hemisphere	1.3 ± 0.4	2.9 ± 1.2	1.4–6.8	<0.001
Nadir FFT power ratio (8–13 Hz/1–4 Hz), left hemisphere	1.1 ± 0.3	0.07 ± 0.02	0.03–0.14	<0.001
Nadir FFT power ratio (8–13 Hz/1–4 Hz), right hemisphere	1.1 ± 0.3	0.07 ± 0.02	0.03–0.14	<0.001
Nadir FFT edge, (95%, 1–18 Hz) left hemisphere	13 ± 2	3 ± 0.5	2–4	<0.001
Nadir FFT edge, (95%, 1–18 Hz) right hemisphere	13 ± 2	3 ± 0.5	2–4	<0.001

SD = standard deviation; aEEG = amplitude-EEG; FFT = fast Fourier transform; Hz = Hertz.
* Due to rapid fluctuations in aEEG, the baseline values were not assessed.

asystole (mean 3.2 ± 1.7 s; range 1–6.2 s), and 10 had asystole prior to QEEG slowing (mean 2.7 ± 1.4 s; range 0.6–4.9 s). Patients with asystole had briefer periods of QEEG changes prior to signs of loss of consciousness compared to patients without asystole (7 ± 5 versus 11 ± 4 s, $p < 0.001$).

All patients had high-amplitude EEG slowing prior to signs of loss of consciousness. The EEG amplitude changes with syncope are characterized by aEEG changes across slow-flat-slow and slow-only raw EEG patterns. All patients had an aEEG decrement that interrupted the periods of high-amplitude slowing at syncope onset and recovery. With the slow-flat-slow EEG pattern, the aEEG decrement fell to zero microvolts (see Fig. 1). Fig. 2 depicts the aEEG decrement from a patient with a slow-only raw EEG syncope pattern. Signs of loss of consciousness always preceded the aEEG decrement by $\leq$3 seconds, with several patients developing signs of loss of consciousness during the gradual transition from high-amplitude slowing to amplitude suppression. Fig. 3 illustrates the raw-EEG correlate of the aEEG decrement from a slow-only EEG syncope pattern. The aEEG decrement began prior to tilt-table lowering in 30 (39%) of the 76 patients, suggesting that it was not a consequence of blood pressure changes related to changing gravitational forces during table lowering.

3.2. Asymmetry of QEEG changes during syncope

The EASI epoch during the tilted baseline (21.3 ± 4.5) did not differ from the EASI epoch during syncope onset (20.3 ± 3.9), $p = 0.16$. While QEEG signs of cerebral hemisphere lateralization were present on REASI at each studied epoch during syncope, lateralization did not differ from the preset binomial probability of 0.5 (Table 2).

4. Discussion

The present study characterized QEEG parameter changes associated with tilt-induced neurally-mediated syncope from a cohort of young patients without baseline EEG abnormalities or neurologic impairments. The analyses led to several important findings: (1) regardless of the raw EEG pattern (slow-flat-slow or slow-only), an aEEG decrement, bounded by high-amplitude slowing, always followed signs of loss of consciousness; (2) cerebral hemispheric lateralization of the EEG changes during syncope did not appear to differ from the binomial probability of 0.5; and (3) significant changes in FFT power occur with tilt-induced syncope across several QEEG parameters.

During the evolution of neurally-mediated syncope, the blood pressure drops to values that impair cerebral perfusion, eventually leading to loss of consciousness. Changes in the QEEG low-frequency power (1–4 Hz), the FFT edge (95%, 1–18 Hz), and the 8–13 Hz/1–4 Hz power ratio provided consistent measures of EEG changes as hypotension affects cortical neuron function. Over half of the patients with asystole had QEEG slowing that preceded the heartbeat pause. The presence of early EEG slowing relative to asystole suggests that the hypotension related to impaired systemic vasoconstriction and orthostatic blood pooling played a greater role in the initiation of tilt-induced syncope than the pause in heartbeat for these patients.

The aEEG parameter quantifies changes in EEG amplitudes. The present study demonstrated that a decrement in aEEG interrupted periods of high-amplitude EEG slowing during syncope onset and recovery, which was present in all syncope studies regardless of whether a slow-flat-slow or slow-only frequency pattern was present on raw EEG. We hypothesize that the spectrum of amplitude suppression during the aEEG decrement represents variations in the degrees of cerebral perfusion when it reaches its nadir, with the greatest degree of suppression present when impaired cerebral perfusion reaches a critical value that causes the EEG to flatten (Astrup et al., 1981). Since signs of loss of consciousness always occur after the initial onset of EEG slowing, but before the aEEG decrement, the degree of impaired perfusion with tilt-induced syncope appears to exceed what is necessary to impair consciousness, which might explain why syncope with asystole is more likely to cause a slow-flat-slow EEG pattern. Patients with asystole had significantly briefer periods of EEG slowing prior to loss of consciousness, suggesting more rapid changes in cerebral perfusion compared to patients without asystole. As all patients with syncope developed some degree of amplitude suppression, perhaps the patterns of EEG slowing with syncope should be viewed across a spectrum from mild amplitude suppression to EEG flattening, rather than slow-only and slow-flat-slow.

The laterality of EEG changes with syncope appears to be no different than the flip of a coin. The previous finding of EEG activity lateralizing to the left cerebral hemisphere with syncope (Mercader et al., 2002) could not be confirmed in the present study. In this larger cohort, EEG lateralization at the onset of QEEG changes, the peak of FFT power in the 1–4 Hz frequency, and at QEEG resolution did not differ from a probability of 0.5.

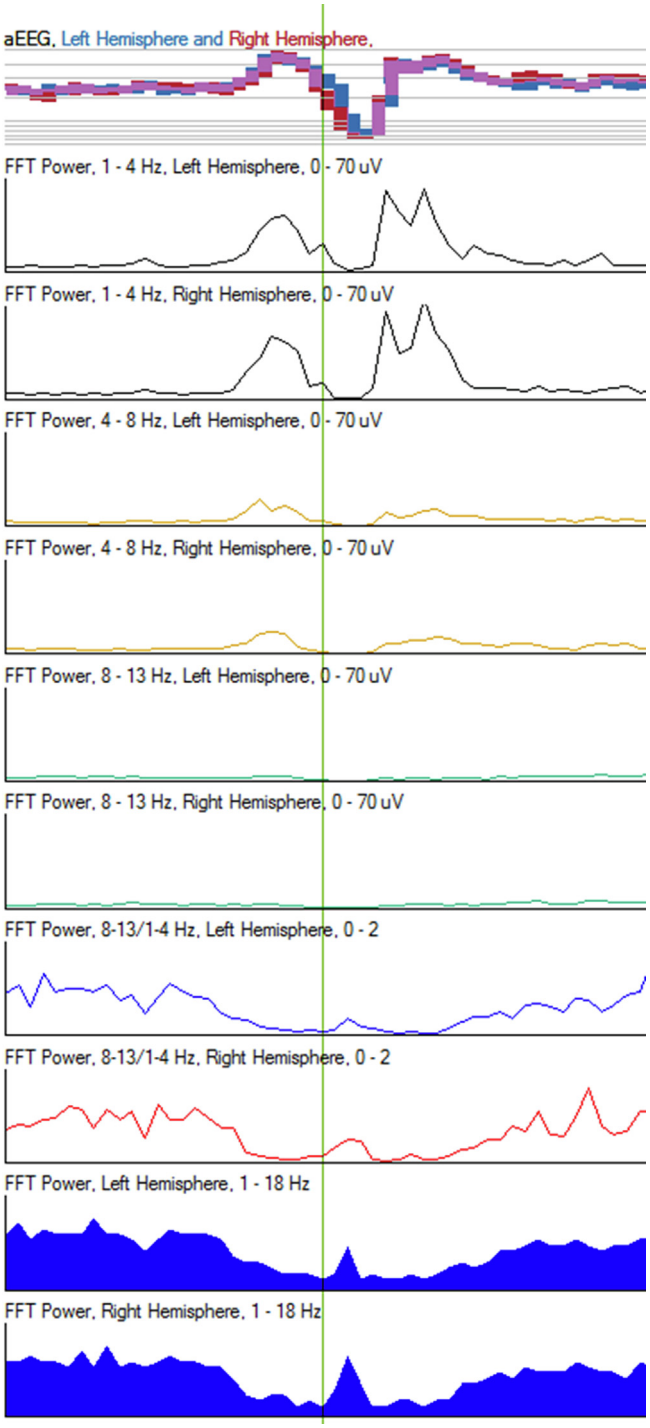

Fig. 1. QEEG parameter changes associated with a slow-flat-slow EEG pattern with syncope. The aEEG (top row) demonstrates a substantial decrement in EEG amplitude that coincides with flattening of the raw EEG. Syncope occurred during the transition from the first slow phase to the flat phase, the beginning of the aEEG decrement. The peaks in the FFT power (1–4 Hz range) follow the amplitude nadir (rows 2 and 3). FFT power ratio (rows 4 and 5) and FFT edge (rows 6 and 7) have nadirs that follow the aEEG decrement. Vertical grid marks represent 30 s.

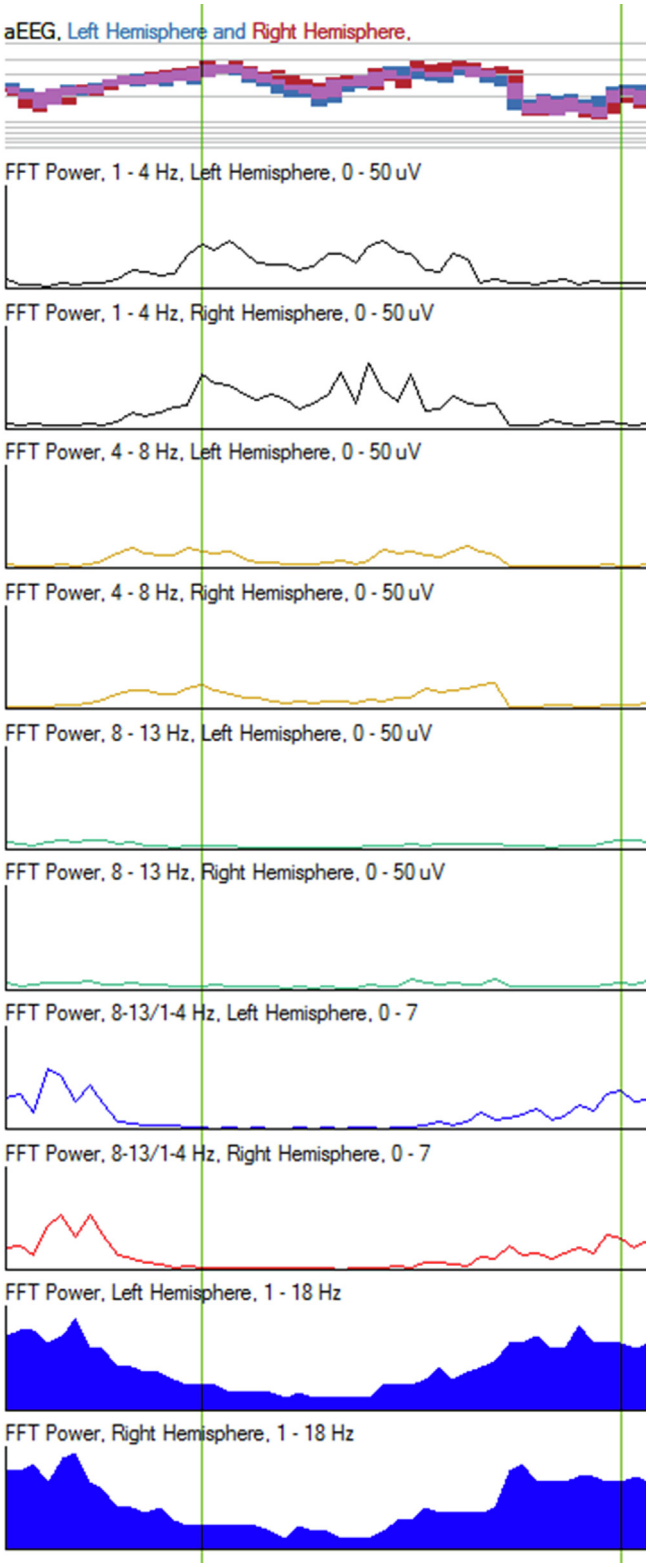

Fig. 2. QEEG parameter changes associated with slow-only raw EEG pattern with syncope. The aEEG decrement is present, but not to the extent seen with the slow-flat-slow EEG pattern from Fig. 1. Syncope occurred during the initial downward deflection in amplitude. The FFT power (1–4 Hz), FFT power ratio, and FFT edge demonstrated changes similar to Fig. 1. Vertical grid marks represent 30 s.

4.1. Limitations

We acknowledge several study limitations. First, artifacts related to movements, blinking, eye deviation, and the convulsion with syncope were present in every sample. Some studies were excluded because artifact on EEG prevented an accurate assessment of one or more QEEG parameters. Since it is not possible to remove artifact, the present study provides a realistic assessment of QEEG changes with tilt-induced syncope averaged over a large

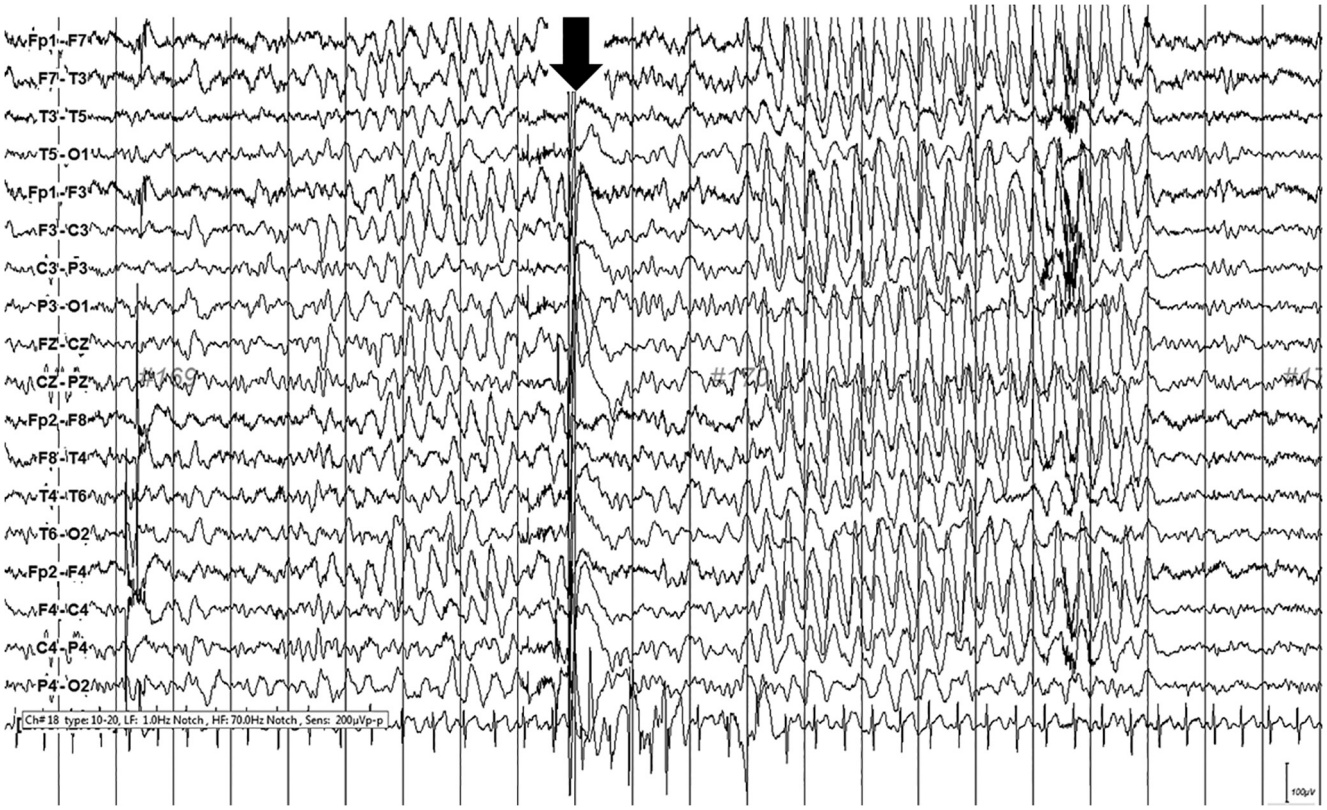

Fig. 3. The slow-only pattern on raw EEG demonstrates relative amplitude suppression with high-amplitude slowing during syncope onset and recovery. Loss of consciousness (black arrow) tends to occur near the transition from high-amplitude slowing to suppression. The degree of amplitude suppression varies across samples and likely represents a continuum from mild suppression to EEG flattening (seen with the slow-flat-slow pattern), representing the degree of impaired cerebral perfusion with syncope.

Table 2
Cerebral hemisphere lateralization during syncope does not differ from the binomial probability of 0.5.

REASI epochs	Cerebral hemisphere	n (observed probability)	p-values
Syncope onset	Right hemisphere	35 (0.46)	0.57
	Left hemisphere	41 (0.54)	
FFT power (1–4 Hz) peak	Right hemisphere	44 (0.58)	0.73
	Left hemisphere	32 (0.42)	
Syncope resolution	Right hemisphere	36 (0.47)	0.73
	Left hemisphere	40 (0.53)	

number of cases. Second, some patients closed their eyes prior to syncope which altered the distribution of QEEG frequencies; however, this effect was minor compared to the large changes in FFT power in the lower EEG frequency ranges. Third, we used onset of loss of consciousness as a study measure, yet determining the actual timing of loss of consciousness from video-EEG is not straightforward. Relying on two or more typical signs (eye opening when initially closed, eye deviation, loss of postural tone, myoclonus, or tonic posturing) for the determination, it is likely that potential errors in timing would reflect later onsets (i.e., the determined onset would be later than true loss of consciousness). The 1-s resolution in timing corrects for most, if not all, of these potential errors. Fourth, hyperventilation normally coincides with (or precedes) the onset of tilt-induced syncope, and the potential effects of hyperventilation on EEG rhythms were not addressed in the present study. However, since hyperventilation can increase the proportion of lower EEG frequencies, it may have played a substantial role in the power changes in the present study. As above, data from the present study provide a realistic assessment of EEG changes with tilt-induced syncope. Fifth, in this exploratory study, results were not stratified by patient age because group sizes in the younger and older age ranges were relatively small. Further study is required to determine if age affects QEEG changes with syncope. Lastly, we presented data that supports EEG amplitude suppression as a relative measure of impaired cerebral perfusion, but we did not present nadir blood pressure data because the blood pressure signal was often lost during the tonic phase of syncope.

5. Conclusion

Certain QEEG parameters help to characterize the EEG frequency and amplitude changes associated with tilt-induced, neurally-mediated syncope. The onset of EEG slowing is readily identified from QEEG measures of delta-range frequencies, the FFT edge, and the alpha/delta-range frequency ratios. An EEG amplitude decrement follows signs of loss of consciousness, regardless of the raw EEG pattern of slow-only or slow-flat-slow. The aEEG decrement probably reflects the nadir in cerebral perfusion with syncope. Lastly, at the onset of syncope and during its evolution, cerebral hemisphere lateralization appears to be no different from a coin flip. QEEG may serve as a useful tool for the study of syncope neurophysiology and may be helpful in modeling other neurologic disorders caused by defects in cerebral perfusion.

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