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Multisensory Temporal Function and EEG Complexity in Patients with Epilepsy and Psychogenic Nonepileptic Events

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Abstract

Cognitive and perceptual comorbidities frequently accompany epilepsy and psychogenic nonepileptic events (PNEE). However, and despite the fact that perceptual function is built upon a multisensory foundation, little knowledge exists concerning multisensory function in these populations. Here, we characterized facets of multisensory processing abilities in epilepsy and PNEE patients, and probed the relationship between individual resting-state EEG complexity and these psychophysical measures in each patient. We prospectively studied a cohort of epilepsy (N = 18) and PNEE (N = 20) patients who were admitted to Vanderbilt's Epilepsy Monitoring Unit (EMU) and weaned off of anticonvulsant drugs. Unaffected age-matched controls staying with the patients in the EMU (N = 15) were also recruited as controls. All participants performed two tests of multisensory function: an audio-visual simultaneity judgment and an audio-visual redundant

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None of the authors has any conflict of interest to disclose.

7. Ethical Publication Statement

We confirm that we have read the Journal's position on issues involved in ethical publication and affirm that this report is consistent with those guidelines.

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target task. Further, in the cohort of epilepsy and PNEE patients we quantified resting state EEG gamma power and complexity. Compared with both epilepsy patients and control subjects, patients with PNEE exhibited significantly poorer acuity in audiovisual temporal function as evidenced in significantly larger temporal binding windows (i.e., they perceived larger stimulus asynchronies as being presented simultaneously). These differences appeared to be specific for temporal function, as there was no difference among the three groups in a non-temporally based measure of multisensory function - the redundant target task. Further, patients with PNEE exhibited more complex resting state EEG patterns as compared to their non-psychogenic counterparts, and EEG complexity correlated with multisensory temporal performance on a subject-by-subject manner. Taken together, findings seem to indicate that PNEE patients bind information from audition and vision over larger temporal intervals when compared with control subjects as well as patients with epilepsy. This difference in multisensory function appears to be specific to the temporal domain, and may be a contributing factor to the behavioral and perceptual alterations seen in this population.

Keywords

Multisensory; Temporal; Epilepsy; Psychogenic; EEG; Neural Complexity

1. Introduction

Patients with epilepsy and those with psychogenic non-epileptic events (PNEE) often experience cognitive (e.g., episodic memory) and perceptual (e.g., auditory hallucinations) impairments [1, 2]. Although the difficulty these patients have when interacting with their environment may stem from disturbances in higher-order brain networks, they may also be a result of changes in lower-level sensory function (or some combination of these). Indeed, there has been a recent focus on examining changes in sensory processing in epilepsy patients [3–6], and although a recent account of PNEE reports no systematic study of sensory function in this population [7], several case studies do suggest sensory abnormalities in this understudied population [8, 9]. However, this work, in both epilepsy and PNEE patients, has largely been restricted to examining single sensory systems. Studies of multisensory function (i.e., the ability to synthesize information across the different senses) in the context of epileptic disorders are rare, a surprising gap given the importance of multisensory function in the construction of veridical perceptual and cognitive representations [10, 11].

Cases of atypical sensory processing have been linked to an imbalance between neuronal excitation and inhibition, which is a key mechanism in the generation of epileptic seizures [12–14]. At a cellular level, recent work has illustrated the importance of synaptic inhibition in gating multisensory integration [15]. This recent observation is well in line with prior work suggesting that inhibition narrows the tuning functions of sensory neurons to their preferred responses and alters the timing and reliability of sensory driven spike output [16]. Collectively, this work reinforces presumptive links between the changes in inhibitory processes known to accompany epilepsy and fundamental mechanisms of multisensory integration. Lastly, gamma-aminobutyric acid (GABA), the principle inhibitory

neurotransmitter in the cerebral cortex, in addition to playing a key role in sensory filtering and being deficient in epilepsy [17], has been shown to contribute to the generation of gamma band oscillations [18]. An oscillatory power which spontaneous activity in epileptic patients has been suggested to index the onset of an epileptic event [19, 20], and a frequency band taken to dictate the degree to which individuals bind information from distinct sensory modalities [21, 22]. Indeed, recent work has suggested a tripartite relationship between GABA concentration, gamma power, and multisensory binding [23].

It is under this framework, that the study of multisensory temporal binding in an epileptic population is interesting beyond its clinical applicability. A key question within the study of neural information processing is the manner by which information is integrated. Influential theoretical views have posited a privileged status regarding information integration for neural oscillations within the gamma range (specifically 40Hz) in particular as it relates to temporal and/or feature binding [24, 25]. Neural complexity, which is aberrant during seizure [26], is also reflective of neural integration and has been put forward as an important indicator of perceptual awareness [27], a state that is characterized by the unity of our perceptual experiences [28]. Thus, we may expect that the unity or integration of the perceptual world to be fundamentally different in epileptic patients than in the general population. Hence, a study of this clinical population may provide important neurobiological insights into the general question of information binding.

In the current study, we specifically tested multisensory (i.e., audiovisual) function in epilepsy and PNEE groups, taking advantage of psychophysical tasks of both general (redundant target) and temporal (simultaneity judgment) abilities. The focus on a temporal task was grounded in the importance of inhibition (and by extension E/I balance) in mediating temporal processes. In addition, we related multisensory abilities to neural function, particularly resting state gamma power and EEG complexity (measured by Lempel-Ziv complexity, see *Methods*).

2. Methods

2.1 Participants

As detailed in Table 1, we prospectively enrolled 53 participants (25 females, mean age = 40.37 years, range = 19–62 years; duration of disease = 13.8 ± 16.2 years). The diagnosis of epilepsy or as exhibiting PNEE was determined by attending epileptologists via video-EEG monitoring and was not known to the investigators at the time of recruitment or psychophysical testing. After completion of the study it was determined that there were 20 patients with PNEE (11 females, mean age = 40.60 years), and 18 epileptic patients (7 females, mean age = 38.55 years). In addition, 15 age-matched controls (7 females, mean age = 43.26 years) were recruited. Consistent with a higher incidence of PNEE in women [29], the patient groups did differ in sex (55% females in the group of patients exhibiting PNEE vs. 39% females in the epileptic group, $p = 0.009$), as well as disease duration (PNEE 3.5 ± 2.6 years, 24 ± 18 years epileptic, $p < 0.001$). Control participants were family members or friends of the patients who stayed with the patients in the EMU, and thus had the same EMU environmental exposure as the patients. All anticonvulsant medications were stopped during the course of the stay at the EMU as well as during psychophysical and EEG

testing. Patients were gradually weaned off of medication over the course of several days, and psychophysical testing occurred 2–4 days following medication stoppage. All participants had normal or corrected-to-normal visual acuity and self-reported normal auditory acuity. Control participants self-reported to have no psychiatric or neurological history. Vanderbilt University Medical Center's Institutional Review Board approved all experimental protocols, and written informed consent was obtained from all participants.

2. 2. Materials and Apparatus

2. 2. 1. Audio-Visual Simultaneity judgment—Visual and auditory stimuli were controlled via a purpose-made microcontroller (Arduino, refresh rate 10 KHz) and driven by in-house experimental software (ExpyVR, direct serial port communication with microcontroller, [30]). Visual stimuli were presented by means of a red LED (7000 mcd, 640 nm wavelength, 348 radiance angle), and auditory stimuli were generated by the activation of a piezo speaker (75 dB at 0.3 meters, 3.0 kHz). An audiovisual device was built by assembling the auditory and visual stimuli into a 5 cm X 3 cm X 1 cm opaque rectangular box (see Figure 1A). Both visual and auditory stimuli had a duration of 10 ms and were presented within a range of stimulus onset asynchronies (SOAs) that included 0 ms, ± 20 ms, ± 50 ms, ± 100 ms, ± 150 ms, ± 200 ms, ± 300 ms, and ± 500 ms. By convention, positive SOAs indicate conditions in which visual stimuli preceded auditory stimuli. Participant's responses were made via button press. Accurate timing of all components involved in the procedure above-mentioned was verified via oscilloscope.

2. 2. 2. Audio-Visual Reaction time: Multisensory Redundant Target Task—In order to probe auditory, visual, and audio-visual reaction times, we presented participants with sensory stimuli in 9 different conditions in a 3 X 3 factorial design (3 intensities of visual stimuli X 3 intensities of auditory stimuli). Visual and auditory stimuli were presented on a computer monitor and controlled via E-Prime software (Psychology Software Tools). Visual stimuli were either absent (V0) or a white circle presented for 100 ms on a gray background at an intensity of either 0.0036 (V1) or 0.0108 (V2) Michelson Contrast. Auditory stimuli were absent (A0), or a pure tone at 2000 Hz, presented for 100 ms at an intensity of either 15 dB (A1) or 35 dB (A2) SPL. There was no stimulus onset asynchrony between the auditory and visual stimuli in the case of audio-visual presentations.

2. 2. 3. EEG resting state—Patients, but not control subjects, underwent continuous video-EEG monitoring in order to ascertain the focus of their seizures. As part of their clinical assessment, a resting-state eyes-closed epoch for at least 5 minutes was collected. By —resting-state here, we refer to the fact that participants were not actively completing an experimental task and were simply instructed to relax and keep their eyes closed. Spontaneous cortical electrical activity was recorded with a 19-channel EEG system (EEG-1000/EEG-1200, Nihon Kohden, Inc., Tokyo, Japan), filtered through a 0.53–120 Hz band-pass filter, and sampled at 200 Hz. EEG was recorded with the electrodes positioned according to the international 10–20 system (i.e., Fp1, Fp2, F3, F4, C3, C4, P3, P4, O1, O2, F7, F8, T3, T4, T5, T6, Fz, Cz, Pz) using a linked ears reference. For some patients, additional electrodes were added if clinically necessary. Electrode impedances were kept below 5 k Ω . For each patient, a 300-s artifact-free, resting-awake segment was manually

selected by visual inspection using Neuroworkbench software (Nihon Kohden, Inc., Tokyo, Japan).

2. 3. Procedure

Patients (both PNEE and epileptic) and control participants performed both a Simultaneity Judgment task (SJT) and a Multisensory Redundant Target (MRT) task. All participants, including controls, were comfortably seated within their clinical rooms in the EMU. In the case of the SJT, participants were asked to judge whether an audiovisual event happened synchronously or asynchronously and to indicate their response via button press. Accuracy was emphasized over speed. They completed two separate experimental blocks, each consisting of 120 trials (8 repetitions X 15 SOAs), for a total of 240 trials (16 repetitions per condition). Trial order within each block was fully randomized, with an inter-trial interval between 1 and 2 s (uniform distribution).

In the MRT, participants were asked to respond via button-press as fast as possible when they first detected any stimuli presentation (unisensory audio or visual, or multisensory audiovisual). A total of 240 experimental trials were presented equally divided between the 8 experimental conditions (i.e., A0V1, A0V2, A1V0, A1V1, A1V2, A2V0, A2V1, A2V2), which were each repeated 30 times. In addition, 60 catch trials (i.e., A0V0) were also presented. The inter-trial interval was 2000 ms $\pm$ 700ms. Trial order was fully randomized, and task order (SJT vs. MRT) was counter-balanced between participants. Total experimental duration was approximately 45 minutes.

2. 4. Analysis

2. 4. 1. Behavioral—For the SJT, distributions of perceived simultaneity (i.e., report of synchrony) as a function of SOA were compiled and averaged for each participant. All trials were included in the analysis and there was no response time restriction. Individual participant's average report of synchrony as a function of SOA was fitted with a two-term Gaussian (Eq 1., Figure 2A) whose amplitude (amp, fraction of perceived simultaneity), mean, and standard deviation were free to vary. A two-term Gaussian was utilized in order assure an accurate description of the underlying shape of the distribution detailing reports of synchrony as a function of SOA both in the control and patient populations. The shape of the normal distribution proved to accurately describe the reports of synchrony (mean $R^2 = 0.95$; One-Way ANOVA, $p = 0.88$). The mean of the first term was taken as the point of subjective simultaneity (PSS; the stimuli asynchrony at which participants are most likely to report synchrony), and the distribution's standard deviation (first term) as a measure of the temporal binding window (TBW; the temporal extent over which participants are highly likely to report stimuli as being synchronous, [43]). In this manner, although a two-term Gaussian was utilized in order to restrict parameters and most faithfully describe the underlying function we were fitting, the analysis was performed solely on the first terms, as in all previous accounts of normal distributions describing the shape of reports of synchrony as a function of SOA. For the MRT, reaction time and detection data were compiled for each participant as a function of audiovisual stimuli intensity, and then averaged across subjects.

$$P(response|SOA) = amp \times \exp\left(-\left(\frac{(SOA-PSS)^2}{2SD^2}\right)\right) \times amp_2 + \exp\left(-\left(\frac{(SOA-PSS_2)^2}{2SD_2^2}\right)\right) \quad (\text{Eq. 1})$$

2. 4. 2. EEG – Lempel-Ziv Complexity—The Lempel-Ziv (LZ) complexity algorithm calculates the approximate amount of non-redundant information contained within a string by estimating the minimal size of the vocabulary⁴ necessary to describe the entirety of the information contained within the string in a lossless manner [31]. LZ is used to quantify distinct patterns in symbolic sequences, especially binary signals and has recently been employed to analyze resting state EEG patterns in a number of neuropsychiatric diseases [32, 33]. To apply the LZ algorithm, as we first converted the signals in all electrodes to a binary sequence by thresholding our voltage data based on the instantaneous amplitude of the Hilbert transform for each particular channel. Data points of a particular channel above the mean of that channel were assigned 1⁴, while those under the mean were assigned a value of 0⁴. Next, binary strings were constructed by column-wise concatenating the values at each of the 19 electrodes [32] (Figure 1B). Finally, the LZ complexity algorithm determined the size of the dictionary needed to account for the pattern of binary strings observed. No normalization was undertaken, as all epochs compared were of equal length.

2. 4. 3. EEG – Gamma Band Power—We applied a fast Fourier transform (FFT) to the resting state EEG data described above to obtain the absolute spectral power for each channel in the low-gamma band, defined as frequencies between 30 and 50Hz, at a 4Hz resolution.

3. Results

Because seizures are linked with imbalances in excitatory/inhibitory signaling, we expected that epilepsy patients would exhibit alterations in multisensory function, in particular on the temporal task. In the more general measure of audiovisual integration, the redundant target task, we found no significant performance differences across the three groups (control, epilepsy, and PNEE). A two-way mixed-model ANOVA [Stimulus Intensity (Within-subject variable) X Group (Between-subject variable)] demonstrated that reaction times were generally faster ($F(7, 287) = 27.73, p < 0.001$) for more intense stimuli (e.g., A2V2: $M = 304.34$ ms, $SD = 80.62$ ms) when compared with less intense stimuli (e.g., A1V1; $M = 494.35$ ms, $SD = 180.25$ ms). Importantly, however, there was no main effect of group ($F(2, 41) = 2.56, p = 0.089$), nor a Stimulus Intensity X Group interaction ($F(14, 287) < 1, p = 0.94$). The accuracy results for this task revealed an equivalent pattern, demonstrating a main effect of Stimulus Intensity ($F(7, 287) = 168.45, p < 0.001$), but no main effect of Group ($F(2, 41) = 1.66, p = 0.20$), nor an interaction between these factors ($F(14, 287) = 1.58, p = 0.22$).

In contrast, significant differences were found for the audiovisual temporal task. Surprisingly, the key finding here was that PNEE patients had poorer audiovisual temporal acuity, as evidenced by larger multisensory binding windows, when compared with controls or epilepsy patients (Figure 2). A one-way ANOVA ($F(2, 53) = 10.87, p < 0.001$)

demonstrated that PNEE patients exhibited significantly larger temporal binding windows ($M = 165.96$ ms, $SD = 95.57$ ms) as compared to their epileptic counterparts ($M = 64.72$ ms, $SD = 53.60$ ms; unpaired t-test vs. psychogenic patients, $t(37) = 3.87$, $p < 0.001$), as well as controls ($M = 82.75$ ms, $SD = 47.00$ ms; $t(38) = 3.25$, $p = 0.002$). A second measure of multisensory temporal function (but not acuity), the point of subjective simultaneity (PSS), revealed no differences between the three groups. For this measure, a one-way ANOVA ($F(2, 53) = 1.44$, $p = 0.24$) revealed that PNEE patients ($M = 4.43$ ms, $SD = 59.43$ ms), epileptic patients ($M = 14.34$ ms, $SD = 48.32$ ms), and control participants ($M = 32.18$ ms, $SD = 33.76$ ms) all displayed moderately positive PSS values. A positive PSS is consistent with the sensory statistics of natural audiovisual stimuli in the environment and in which the arrival of visual energy at the sensory apparatus invariably precedes the arrival of auditory energy [34]. Lastly, there was no difference with regard to the maximum amplitude of the distributions which best described the reports of synchrony as a function of group ($F(2, 53) < 1$, $p = 0.99$; PNEE, $M = 0.98$, $SD = 3.06$; epileptic, $M = 0.95$, $SD = 2.59$; control, $M = 1.00$, $SD = 4.05$), which argues against the differences in TBW size being a consequence of a response bias. That is, it is unlikely that the difference in audiovisual temporal performance across groups was simply due to the fact that one group was simply more (or less) likely to report synchrony across all SOAs.

Prior studies suggested a strong relationship between gamma band power and temporal function and abilities, and thus we sought to quantify gamma power in PNEE and epileptic patients. Although on average, as demonstrated via the FFT, PNEE patients exhibited numerically higher power within the gamma range ($M = 26.07 \times 10^{-3} \mu V^2/Hz$, $SD = 36.56 \times 10^{-3} \mu V^2/Hz$) as compared to non-psychogenic epilepsy patients ($M = 14.63 \times 10^{-3} \mu V^2/Hz$, $SD = 11.27 \times 10^{-3} \mu V^2/Hz$), this difference failed to reach statistical significance ($t(21) = 1.032$, $p = 0.33$) within our dataset. Nonetheless, because of the difficulty in recording reliable gamma band activity via scalp EEG and the possibility that participants transition between wakefulness and drowsiness states during the resting-state recording period, these results must be interpreted cautiously.

On the other hand, Lempel-Ziv complexity analysis of the resting state EEG demonstrated that PNEE patients ($M = 1136$, $SD = 246$) had more complex resting states EEGs than epileptic patients ($M = 977$, $SD = 282$). Interestingly, both within the PNEE ($R = 0.64$, $P = 0.02$) and the epileptic ($R = 0.41$, $P = 0.05$) groups, there were significant correlations between measures of complexity and audiovisual temporal acuity. In this analysis the relationship was such that the more complex a particular individual's EEG resting state, the greater the size of their temporal binding windows (i.e., the poorer their audiovisual temporal acuity, see Figure 3). Such a correlation did not hold between the complexity of an individual's resting state EEG and either unisensory or multisensory reaction times (as measured in the redundant target task), thus highlighting the specificity of the association between the neural complexity measure and audiovisual temporal acuity.

4. Discussion

The major finding of the current study is that PNEE patients, but not epileptic patients, exhibit changes in multisensory function when compared with nonepileptic controls,

contrary to our original hypothesis. Furthermore, this difference was unique to the multisensory temporal task, with no significant difference between groups found in a more generalized measure of multisensory function – the redundant target task. To the best of our knowledge, this study represents the first attempt to characterize the multisensory processing abilities of these two patient groups. Because multisensory binding is a core component in the creation of veridical perceptual representations [10, 30], the observed changes in multisensory processing may play an important contributing role in the etiology and cognitive comorbidity seen in PNEE patients [35].

Epileptic patients did not differ from control subjects in the performance of either of these psychophysical tasks. Given the evidence for changes in excitatory/inhibitory balance in epilepsy and the importance of excitatory/inhibitory signaling in the creation and maintenance of sensory and multisensory filters, this negative result was unexpected. One possibility is that disruptions in excitatory/inhibitory balance in the epilepsy patients in this study did not substantially affect the network responsible for either of the multisensory tasks. Prior work has suggested that performance on the multisensory temporal task used in the current study is mediated by a network centered on the posterior superior temporal sulcus and involving reciprocal connectivity with visual and auditory cortices [36]. Although six of the patients enrolled in the current study were diagnosed with focal temporal lobe epilepsy (Table 1), the majority of patients did not have seizures originating from this area, and even for the six with focal temporal lobe epilepsy it is possible that the epileptiform focus was far from the temporal lobe areas implicated in multisensory temporal function. Future studies of patients with lateral temporal lobe seizures may identify differences in audiovisual multisensory processing in these patients.

The pathophysiological causes of PNEE remain unknown [35], but it is clear that it is not associated with gross structural or physiological abnormalities. Therefore, we were surprised that PNEE patients exhibited significant differences in audiovisual temporal function when compared with epileptic patients and normal controls. This finding suggests that the brain networks involved in audiovisual temporal integration, including regions of the superior temporal sulcus, may be affected in PNEEs. Interestingly, a recent diffusion tensor imaging study demonstrated that PNEE patients had significantly higher fractional anisotropy values in the left superior temporal lobe relative to control subjects [37]. An alternative possibility in these patients is that the changes in higher-order (i.e., cognitive) networks responsible for their psychogenic seizures also give rise to multisensory temporal changes. More specifically, it could be that the effort associated within segregating sensory stimuli in time or the willingness to report a closely spaced audiovisual stimulus as simultaneous differs in these patients, thus giving rise to a larger binding window in the PNEE patients. Although such a change in attention or effort in PNEE in comparison to controls and epileptic patients is seemingly unlikely here due to the specificity of the psychophysical anomaly described (no difference in reaction time and only at select temporal disparities under the simultaneity judgment task), future work could begin to tease out the respective contributions of stimulus statistics and cognitive biases to this task, and to the differences observed between control, epileptic, and PNEE patients.

Multisensory processing differences, most notably in the temporal dimension, are being increasingly recognized in diseases such as autism [38, 39] and schizophrenia [40, 41]. Studies in autism have associated the observed reduced multisensory temporal acuity to higher-order domains, such as language and social communication [38, 40]. Such work is suggestive that the changes in audiovisual temporal binding in PNEE patients may contribute to some of the clinical and cognitive deficits seen in these individuals.

Because epilepsy patients, but not PNEE patients, have hypersynchronous discharges, we expected that PNEE patients would exhibit greater complexity within their EEG resting state traces than their non-psychogenic counterparts as measured using the Lempel-Ziv algorithm. Indeed, our results confirmed this hypothesis, and perhaps even more interesting in this analysis was the fact that for individuals within both the PNEE and epileptic groups measures of EEG resting state complexity directly correlated with multisensory temporal acuity. That is, the greater the —dictionary needed to fully explain the spatio-temporal patterns of voltages across the scalp of a particular individual during a resting state EEG, the larger the participant's binding window. In this sense, our results are the counterpart of Gazzaniga's seminal observation that split brain patients have i) less complex brain dynamics – as they effectively possess two-half brains as opposed to an integrated whole, and ii) an uncanny ability to segregate information in a rapid serial visual presentation paradigm [42]. Here, our results suggest that the more complex an individual's resting state EEG, the more this individual integrates sensory information. A limitation of the current study, however, is that we were not able to measure resting state EEG data from control participants, and thus, do not know whether PNEE patients exhibit a different EEG complexity during resting state than controls. Namely, it is entirely possible that PNEE patients do not show abnormally complex resting states EEGs, but rather that epileptic patients show abnormally simple EEG resting states. Further, it will be interesting in future work to extend these analyses to healthy individuals in an effort to determine if there is a positive correlation between resting state EEG complexity and TBW size in the general population.

In conclusion, we demonstrated that PNEE patients, but not epileptic patients, exhibited enlarged temporal windows within which they bind together audiovisual information relative to control subjects. This reduced audiovisual acuity may be associated with the cognitive deficits in these individuals, and may be a result of changes in networks responsible for the computation of audiovisual temporal relations, networks responsible for cognitive biases, or a combination of these networks.

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Highlights

- We measured temporal multisensory function in epileptic and psychogenic patients.
- Psychogenics exhibited poorer multisensory temporal acuity than epileptics.
- We measured via EEG resting state gamma power and neural complexity.
- Psychogenic patients exhibited more complex resting state neural states.
- Neural complexity and temporal acuity negatively correlated.

Figure 1.

Experimental Protocol and Analysis. A) An LED and Piezo Speaker were mounted onto a single audiovisual device, and their onset was manipulated in order to be synchronous (top row), or asynchronous (bottom two rows). Participants were to judge the simultaneity (or lack thereof) of the stimuli presented. B) The Lempel-Ziv algorithm analysis was undertaken in order to measure resting state neural complexity (detail provided in text).

Figure 2.

Reports of Synchrony and Temporal Binding Window. A) Reports of synchrony as a function of stimulus onset asynchrony (SOA; x-axis) and participant group. Patients with PNEE (black) show a greater tendency, as compared to epileptic patients (red) and controls (blue), to perceive audiovisual stimuli presented at large asynchronies (audio-leading on the left and visual-leading on the right). B) Temporal binding windows (standard deviation of the distribution of reports of synchrony) are significantly larger for psychogenic than for epileptic and control patients.

Figure 3.

Correlation between Lempel-Ziv Complexity and Temporal Binding Window. Patients with PNEE (black) did not only exhibit on average a higher degree of neural resting state complexity than Non-Psychogenic epileptic (red) patients did, but also within groups, the degree to which an individual's resting state neural response was complex (y-axis) correlated with the size of the individual's temporal binding window(x-axis).

Table 1

Participant's demographics. Gender, age, years since diagnosis (PNEE 3.5 ± 2.6 years; epileptic 24 ± 18 years), diagnosis, and seizure focus and medication (if applicable) for all participants recruited.

Gender	Age	Disease Duration	Diagnosis	Seizure Focus	Medications
F	49	5	Epilepsy	R Frontal Focus	Keppra, Lamictal
F	30	16	Epilepsy	Idiopathic Generalized Epilepsy	Zonegran, Keppra
M	40	32	Epilepsy	Idiopathic Generalized Epilepsy	
F	25	12	Epilepsy	Primary Generalized Epilepsy	Vimpat, Lamotrigine
F	34	15	Epilepsy	Generalized Epilepsy	Oxcarbazepine
M	20	7	Epilepsy	R Hemisphere Epilepsy	Zonisamide
M	40	39	Epilepsy	Complex Partial LT	Divalproex, Vimpat
M	29	13	Epilepsy	RT Epilepsy	Vimpat, Keppra
M	62	60	Epilepsy	Focal Epilepsy RH	Trileptal, Keppra
M	30	16	Epilepsy	Partial Epilepsy L&RH	Lamotrigine, Topomax
M	37	11	Epilepsy	RT Epilepsy	Keppra, Lamictal
F	40	28	Epilepsy	Idiopathic Generalized Epilepsy	Keppra, topomax
M	48	10	Epilepsy	Idiopathic Generalized Epilepsy	Keppra
M	24	16	Epilepsy	Cryptogenic Partial Epilepsy	Oxcarbazepine
F	21	7	Epilepsy	Idiopathic Generalized Epilepsy	Lamictal, Vimpat
M	51	41	Epilepsy	LT Epilepsy	Zonegran, Clonazepam
F	62	60	Epilepsy	Partial Epilepsy L&RH	Topomax, Keppra, Lamictal
M	52	44	Epilepsy	Idiopathic Generalized Epilepsy	Zonisamide
M	22	3	PNEE		Keppra, Trileptal
F	56	5	PNEE		Gabapentin, Topomax
F	51	1	PNEE		Levothyroxine
M	49	Since childhood	PNEE		
F	22	2	PNEE		Vimpat
M	47	6	PNEE		
F	41	3	PNEE		Levetiracetam, Gabapentin
F	53	2	PNEE		Divalproex, Gabapentin

Gender	Age	Disease Duration	Diagnosis	Seizure Focus	Medications
F	49	7	PNEE		
M	19	2	PNEE		Trileptal
F	25	9	PNEE		Keppra, Lemotrigine
M	36	8	PNEE		Vimpat
M	56	3	PNEE		Lamictal
F	21	3	PNEE		Levetiracetam
M	48	1	PNEE		Clonazepam
F	22	3	PNEE		Keppra, Topomax
F	60	1	PNEE		Keppra
M	40	1	PNEE		Vimpat, Gabapentin
F	58	6	PNEE		Topomax, Gabapentin
F	35		Control		
M	41		Control		
F	40		Control		
M	37		Control		
M	41		Control		
M	34		Control		
M	47		Control		
F	54		Control		
M	37		Control		
F	48		Control		
M	37		Control		
M	43		Control		
F	51		Control		
F	42		Control		
F	47		Control		