Finding the Epileptic Origin of Focal Epilepsy in Patient with FCDIIB.

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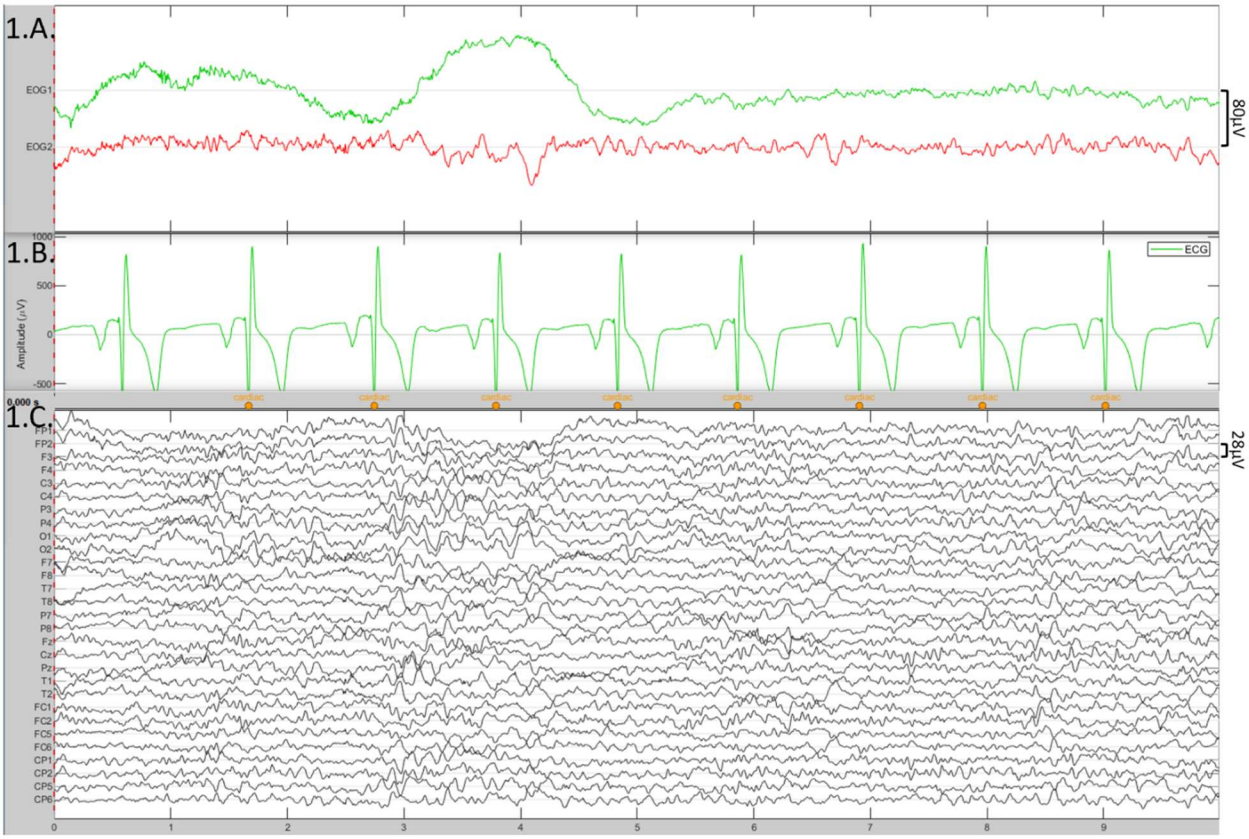

Figure 1 – Original EOG, ECG, EEG waveforms of a 40-year-old focal epilepsy male patient observed over 10 seconds on Brainstorm[1]*. Patient suffers from nocturnal simple and complex partial seizures since 8 years-old. Non-invasive EEG arrangement in double-banana, bipolar montage, with 29 cranial electrodes, 2 EOG electrodes, 1 ECG electrode, 1 EMG electrode; 33 electrodes total. Recording of seizure activity during sleep. Time resolution = 170px/s. Frequency filters: high-pass=0.50Hz, low-pass=80.00Hz, notch=60.00Hz. A) EOG recording; EOG1(green) for left eye movement, EOG2 (red) for right eye movement. Amplitude is in µV (y axis). B) ECG recording of the patient's cardiac activity in µV. C) EEG recording of brain activity over 29 electrodes in µV (y axis), where the 29-channel average has been removed, and the power of each channel is relative to neighbouring channels.*

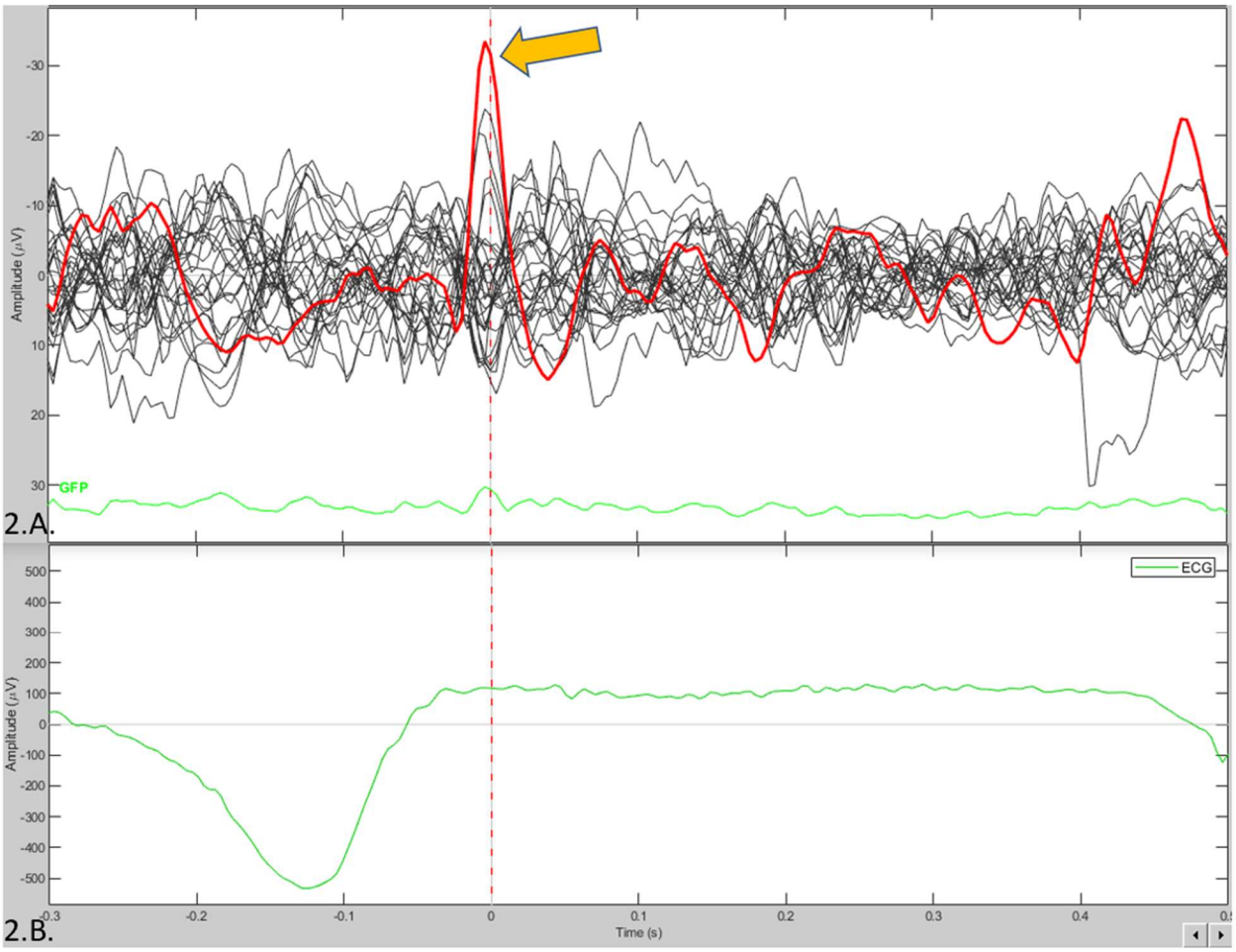

Figure 2 – Flat ECG waveform along the same timeline as epileptic spike in EEG confirms no cardiac contribution. *EEG data was re-referenced to the average of all channel data to subtract the average potential from each channel, hence increasing signal:noise on Brainstorm[1]. A) The yellow arrow indicates the EEG epileptic spike seen at time 0 on channel FC1 highlighted in red at around -35µV. Global Field Power (GFP) line shows where there is most signal variation. Biggest GFP spike also seen at time 0. B) ECG waveform along the same timeline as EEG waveform. The waveform is flat at the same timepoint as the EEG spike (time 0), so the spike observed in the EEG waveform in figure 2.A is not due to cardiac artefact.*

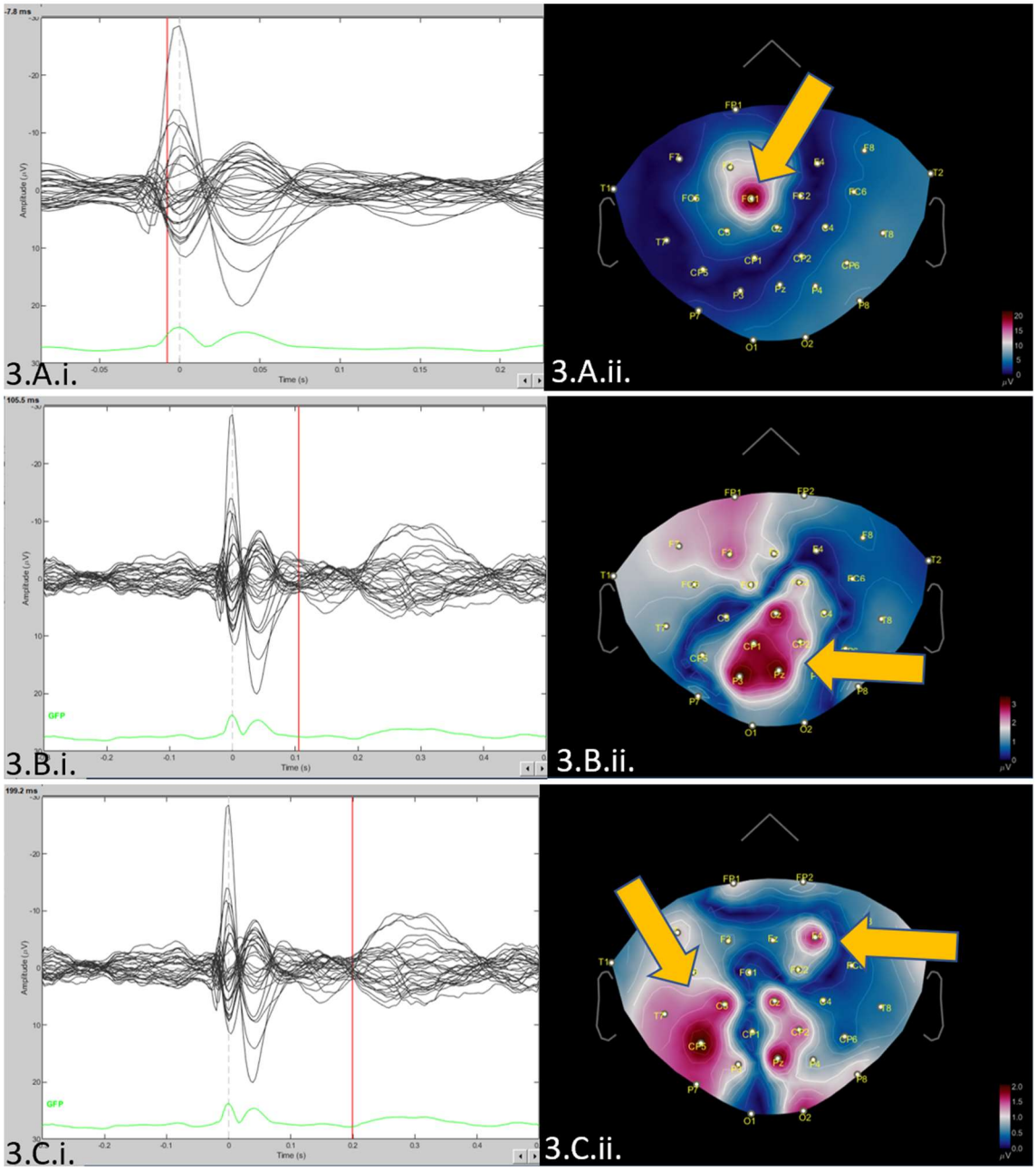

Figure 3 – Timeline of epileptic origin at FC1 and early spread to Pz & P3, then to CP5 on 2D topoplot and EEG waveform. *EEG spikes were all averaged again to remove spikes relative to background activities to substantially increase signal:noise on Brainstorm[1]. A.i) shows the timepoint (-7.8ms) on the EEG waveform where the seizure starts. A.ii) 2D sensor cap of brain activity at the same timepoint as A.i). Yellow arrow points to where there is most activity at that timepoint (dark pink) hence where the seizure originates from (at channel FC1). B.i) Timepoint at 105.5ms after the seizure has started along EEG waveform. B.ii) Yellow arrow points to dark pink area where the seizure activity has spread to at timepoint 105.5ms (channels Cz, CP1, Pz, P3). C.i) Timepoint at 200.0ms after beginning of seizure along EEG waveform. C.ii) Yellow arrows point to 2 areas where the seizure has spread at 200ms (channels C5, CP5, F4).*

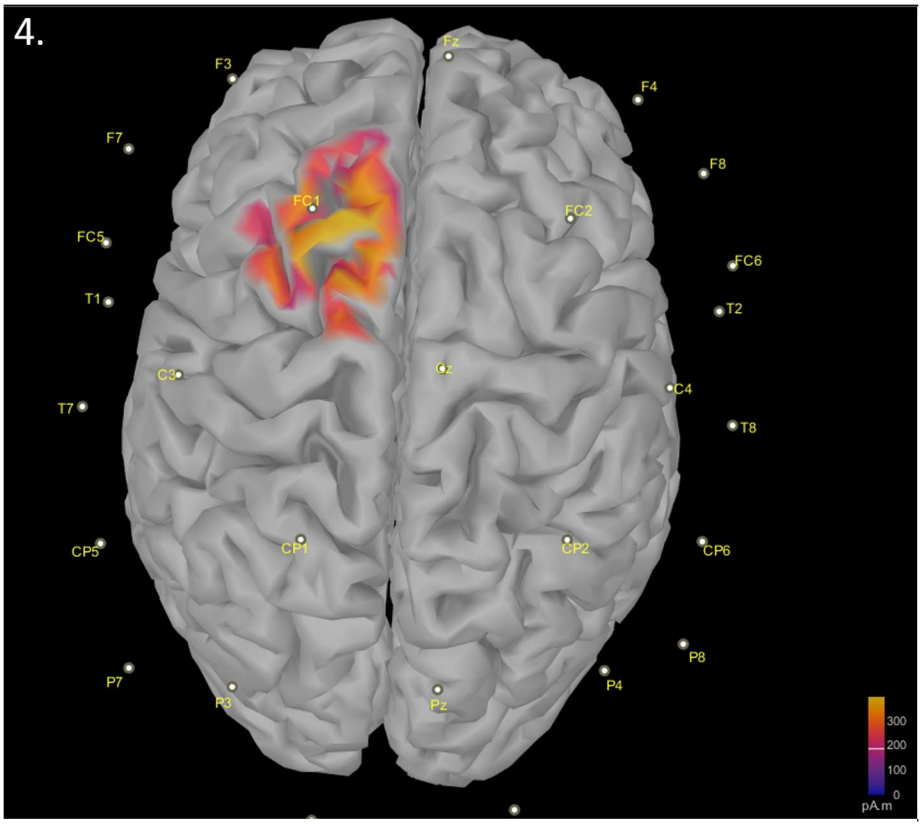

Figure 4 – Epileptic activity origin on 3D computer-generated image based on patient's T1 MRI with Brainstorm[1]*. Red & yellow region indicates neuronal activity above 200pAm, showing epileptic activity origin at FC1 channel as seen in figure 3.A.ii. The axial view of the brain shows the region on the cortex of the left hemisphere (neurological orientation), slightly rostral to the midline. Combining EEG with MRI information is important to line up the electrodes to the precise brain area they measure as EEG signals do not provide enough information about precise origin. MNE can be used for this. This is useful when surgery is required to know which part of the brain to remove.*

Discussion

The patient's focal epilepsy stems from the electrode FC1 as demonstrated in figure 3.A. This electrode measures left superior frontal gyrus activity[2] which is associated with working memory (WM) and spatially-oriented processing[3]. In figure 3.B, we see that the seizure activity has spread to electrodes Pz, P3, and CP1 which measure left precuneus, inferior parietal lobe, and post-central gyrus and superior parietal lobe activity respectively[2]. The precuneus and both inferior and superior parietal cortical thickness and volume (which are reduced in seizure affecting the area) are related to functions in visual memory, visuospatial skills and overall visuoperceptual cognitive performance[4]. The last timepoint observed in figure 3.C showed a

spread of epileptic activity to the CP5 electrode which measures the left superior temporal gyrus[2] which is associated to sublexical speech perception[5]. Based on figure 4's location of the epileptic origin on the patient's computer-generated MRI image, the region of interest (ROI) also lines up with the superior frontal gyrus[6].

The patient followed through a successful frontal lobe resection and was diagnosed with focal cortical dysplasia (FCD) type IIB. FCDIIB is a sporadic developmental malformation of the cortex and is often characterized by the formation of balloon cells, dysmorphic neurons, and lack of cortical layer discrimination[7]. It is a leading cause of pediatric epilepsy which was the case for this patient as he started suffering from epilepsy at 8 years-old. FCDIIB is particularly found in the frontal lobe[8] which was where our ROI was located. FCDIIB is thought to originate from disruptions in the mTOR pathway[9]. The patient's intractability to medication is likely due to his diagnosed FCDIIB[10].

References

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