THE EUROPEAN SOCIETY OF MEDICINE
Medical Research Archives, Volume 12 Issue 8

CASE SERIES AND REVIEW ARTICLE

What happens to your brainwaves when the heart stops? ASYSTOLE AND THE EEG - a case series and review

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OPEN ACCESS

PUBLISHED
31 August 2024

CITATION
Oster, J., M., 2024. What happens to your brainwaves when the heart stops? ASYSTOLE AND THE EEG - a case series and review. Medical Research Archives, [online] 12(8). https://doi.org/10.18103/mra.v12i8.5756

DOI
https://doi.org/10.18103/mra.v12i8.5756

ISSN
2375-1924

ABSTRACT

Introduction-aim and scope: This article highlights a rare observation in 2 patients admitted to the hospital who were evaluated with simultaneous EEG and EKG monitoring due to having episodic clinical spells suspected as being consistent with seizure activity. What however was noted were episodes of asystole causing syncope and brain wave activity persisted for approximately 24 seconds with other clinical features noted in the case histories including an incipient actual seizure in one case. We believe that these patients were fortunate to have such diagnosis made as risks of sudden cardiac death with asystole are high and may be associated with catastrophic neurologic injury and outcomes, and these patients were able to be referred for subsequent cardiac electrophysiologic evaluation and further management[1-4].

Rationale: This report highlights 2 clinical patients with asystole while being monitored with Video EEG (Electroencephalogram) with EKG (Electrocardiogram) that have a complete dataset available for study. One patient exhibited clinical symptoms of syncope and hypoperfusion and one patient after asystole exhibited subsequent electrographic seizure activity. This article descriptively and retrospectively reviews these rarely noted cases and the clinicopathologic details.

Methods: In a hospital system with currently a level 4 epilepsy center that is a member of the US NAEC-National Association of Epilepsy Centers, two patients with asystole were identified as above. This publication provides a retrospective descriptive analysis of two cases that had a complete clinical data set.

Results: Asystole is associated with clinical cerebral hypo perfusion in one case, and in another case, hypo perfusion with the EEG features of hypo perfusion similar to the first case however such is followed by actual electrographic seizure activity. We postulate that these findings may be rarely-recorded manifestations during asystole and deserve further study. Successful clinical management of these cases along with the EEG manifestations are described in the article. Electrocerebral rhythms in both cases persist for approximately 24 seconds after asystole although the exact delineation of the cerebral flow dynamics are not completely understood in this retrospective observational study.

Conclusions: The cases delineate that the pathophysiology and the concomitant use of EEG and EKG monitoring with video during asystole deserve further study as clinical manifestations may not be identified otherwise without such recordings. Monitoring patients with simultaneous Video EEG and EKG recordings may be necessary for optimal diagnostic assessment and therapeutic outcome where clinical decision making by multidisciplinary subspecialty care teams can ensue in real time as noted in this article.

Keywords: asystole, cardiac arrest, seizures, EEG monitoring, hypo perfusion, SUDEP (Sudden unexplained death in epilepsy patients)

Presentation of cases

We admitted a 72 year old patient experiencing loss of consciousness and falls to our Epilepsy Video-EEG monitoring unit since referring physicians strongly suspected on clinical grounds that she had epilepsy. We recorded an EKG channel, a video of patient activities, and Electroencephalographic activity simultaneously using standard 10/20 EEG electrode placement. Spells in which the patient experienced asystole up to about 90 seconds or so were noted. The neurology service referred her to cardiology for cardiac electrophysiology study and ultimately she had pacemaker insertion. Spells resolved and she was doing well in an approximately 10-year follow up period and then was lost to follow up. This patient's video EEG record with EKG monitoring demonstrates the importance of objectively characterizing definitive episodic spells that are alleged to be seizures for diagnostic evaluation and highlight the clinical manifestations that asystole or hypo perfusion of the brain produces. Panels and corresponding video accompaniment illustrate that electroencephalographic activity and alertness persists for approximately 24 seconds after asystole. Subsequently, attenuation of the electroencephalogram and loss of consciousness and likely loss of muscle tone occurs probably due to hypo perfusion. Ultimately EEG activity returns in this case after sustained cardiac rhythm is re-established. See Panels. In retrospect, detailed analysis of the patient's cardiogram showed multifocal and distinct QRS complexes of varied morphologies and durations that were likely indicative of aberrant cardiac conduction patterns..

In the second case, an 81 year old patient with a history of a chronic subdural hematoma was admitted with multiple clinical generalized tonic clonic seizures and clinical status epilepticus. He was treated with escalating doses of benzodiazepines. He became unresponsive. The patient was intubated for airway protection and he was placed on EEG monitoring to effect a burst suppression pattern pharmacologically to stop the seizures. He was infused with fosphenytoin by our institution's standard nursing protocol. Within a few hours after he had received intravenous phenytoin, the patient experienced asystole. Attenuation of the EEG is noted which is similar to that of the first case. Because he was intubated and sedated, the complete clinical characteristics of what was exhibited in the first case likely does not occur to the same extent. Nursing staff however was alerted due to the event alarm on the EKG monitor and clinical treatment was begun with cardiopulmonary resuscitation (CPR). However, during the CPR, the patient experiences subsequently an electrographic seizure which terminates on its own. See Panels. The neurology team, cardiac team, and ICU team noted these findings in real time. As he had another seizure despite fosphenytoin, IV loading bolus of another medication (leviteracetam) followed by maintenance doses was prescribed to mitigate against further seizure activity. An extensive cardiac evaluation including exclusion of ischemia was done using serologic markers (troponin I, Echocardiogram, and consideration for angiography and pacemakers were performed). Sedation was weaned after several days, and the patient ultimately recovered and was doing well with an approximately 5 year follow up period.

Discussion

In a 22-year period, approximately over 2000 Video EEG monitoring studies were estimated to have been performed within our networked institutions and 5 cases presented with asystole on EKG, 2 of which patients have a complete dataset and are described and reviewed in this case series review. Therefore, the prevalence of this finding approximates 0.25 % of such cases studied in the inpatient hospitalized setting in patients suspected as having a diagnosis of seizure disorder when the actual primary diagnosis is asystole or cardiac syncope when evaluated in this manner as a grossly conservative estimate.

We believe that these patients were fortunate to have such diagnosis made as risks of sudden cardiac death with asystole are high and may be

catastrophic, and all patients were fortunately expediently referred for subsequent cardiac electrophysiologic evaluation and management[1-4].

Seizure like activity with fainting, incontinence, drop attacks, loss of consciousness and often with a vasomotor prodrome have been described in cardiac arrest or asystole and this article speculates that potentially these symptoms and even other signs were construed as evidence of seizures which is why these patients were presumed to have a seizure disorder and placed on EEG and cardiac telemetry primarily to optimize and delineate treatments for an assumed suspected seizure disorder[1-4,10].

Unfortunately, cardiac arrest and asystole may involve impairment of cardiac output to a significant extent and in general have a high morbidity and mortality[1-3]. Hypo perfusion after approximately five minutes is associated with cerebral anoxic damage along with neurologic disability and poor outcomes[4-6]. The survival rate of patients receiving initial care outside of the hospital setting or by ambulance for cardiac arrest is about 2 %, and only about 15 % of such patients have the return of circulation.[7-8] A review concerning in hospital survival of cardiac arrest while in hospital estimates survival to discharge at about only 14 %, with a range of about 0-28%[9].

Symptoms of cardiac arrest or asystole or syncope may be confused with seizures and there may be limitations from taking a history especially after events to distinguish these different entities by clinical description alone[10]. Seizure may cause cardiac arrhythmias leading to cardiac arrest and hypo perfusion or sudden death, and seizures are postulated to cause and or contribute to cardiac arrhythmias and likely account for a significant proportion of SUDEP cases (Sudden Unexplained Death in Epilepsy)[11-15]. Falls have been reported in seizure induced asystole and seizures may also trigger syncope by triggering ictal asystole[16-17]. Seizure induced asystole has been reported previously[16-18]. The second clinical case may exhibit seizures due to the asystole but effects from prior subdural cannot be excluded, but it has been speculated that asystole may cause seizure and is an exceedingly rare clinical scenario[17].

Confounding variables that may be involved in such cases include medications that may block the AV Node particularly in elderly or other susceptible patients that may lead to arrhythmias, such as may have occurred with phenytoin or actually fosphenytoin in the second case[19]. Typically fosphenytoin has a much lower risk of blocking the AV note than phenytoin, and also such risks generally occur largely during the time of the infusion, which is unlike that occurs in this case report as the cardiac arrest occurred many hours post completion of the infusion[19]. Seizure medications and other medications therefore may present a significant cardiovascular risk to certain patient populations since they may impact cardiac conduction in an unpredictable fashion causing or predisposing the patient to cardiac arrhythmias[16].

The clinical effects and the EEG during hypo perfusion is described and identified in the initial case. The second case indicates that electrographic seizure activity may be an additional sign or result subsequently of similar EEG changes reflecting hypo perfusion but the delineation as noted in this article it is likely under reported. It is also possible that the presence of the prior subdural hematoma contributed to the pathophysiology of the seizure by inducing cortical irritability but such factors remain unknown. The emergence of seizure during asystole may indicate that somehow the hypoperfusion caused or exacerbated or was associated with cortical brain irritability and if such seizure activity were untreated or if undertreated this article queries and opines whether such events might contribute to neuronal changes and further cerebral ischemia and damage, likely reducing good functional outcome. We note that generally CPR (Cardio Pulmonary Resuscitation) occurs without EEG monitoring so the identification of the noted electrographic seizure activity that occurs in the second case is likely a rare manifestation subsequently of asystole and deserves further study both in its

inciting pathophysiology and what effects it may cause. It can be postulated that somehow the hypo perfusion in the presence of a subdural hematoma led to its development or that perhaps the asystole was a direct contributor. It has been noted previously that approximately 1 in 700 partial seizures may be associated with an asystole[20].

Tachycardia has been reported in up to approximately 96 % of seizures and its prevalence is highest in mesial temporal epilepsy and is lowest in non-lesional cortical temporal epilepsy and may be due to more left sided insular region involvement and bradycardia may be seen with right sided cerebral localization of seizures[21-23]. Ictal bradycardia may be seen in frontotemporal epilepsy predominantly on the left and may be fatal[24]. Various ectopic rhythms and ventricular tachycardia may occur and might be due to the limbic cortex and the cingulate gyrus and insular regions may be involved in other cardiovascular effects of seizures[25-26]. There is additionally an emerging literature regarding devices such as vagal nerve stimulation that also monitors heart rhythms and specifically the presence of tachycardia that may be related to seizure activity to improve both detection and treatment of seizure activity in ambulatory outpatients that may relate to the above or other unknown neuroanatomical substrates[21-28].

Additionally there is a literature emerging indicating that chronic epilepsy may be associated with cardiac conduction and vascular structural changes and epilepsy seems to be associated with an approximately increased to 300% risk for SUDEP – Sudden Unexplained Death in Epilepsy risk compared with controls in patients with such effects, and that T wave irregularities might be a harbinger of arrythmias or seizure activity, predict generalized seizures, and potentially distinguish with generalized seizures from those with psychogenic nonepileptic spells additionally[29-31]. How these studies and what specific substrates and mechanisms underly the pathophysiological manifestations in this series remain unknown at this time.

Conclusions:

The cases in this series delineate that the pathophysiology regarding asystole and video EEG findings with EKG monitoring deserve further study as definitive diagnosis and integrating clinical symptoms and signs and overall manifestations may not be identified otherwise without such study and recordings. In our series, these cases represented approximately 0.25% of all our monitored cases among video EEG monitoring candidates, and in these two cases electrocerebral rhythms persisted for approximately 24 seconds after asystole although the exact parameters and overall characterization or delineation of the cerebral blood flow dynamics remain unknowns. Monitoring patients with simultaneous Video EEG and EKG recordings may be necessary for optimal therapeutic outcome where clinical decision making by multidisciplinary subspecialty care teams can ensue rapidly and accurately in real time as noted in this article although there is an emerging practice and literature on how devices that detect heart rhythm may impact the detection and treatment of seizures in ambulatory patients and how epilepsy itself may alter cardiac conduction and contribute to arrythmias and other cardiac pathologies.

Conflict of Interest:

None

Funding Statement:

None.

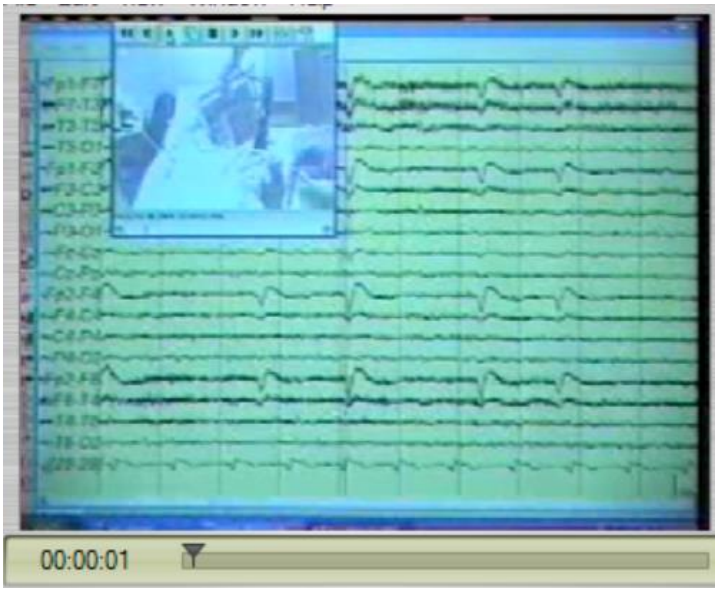
Acknowledgements:

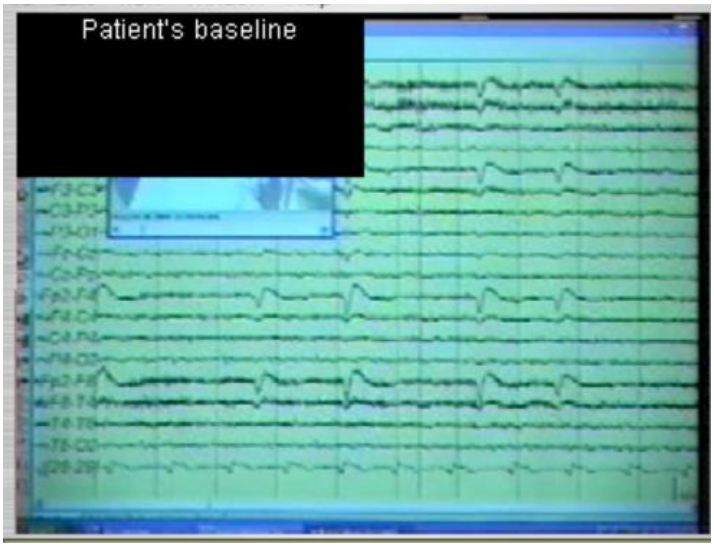
None.

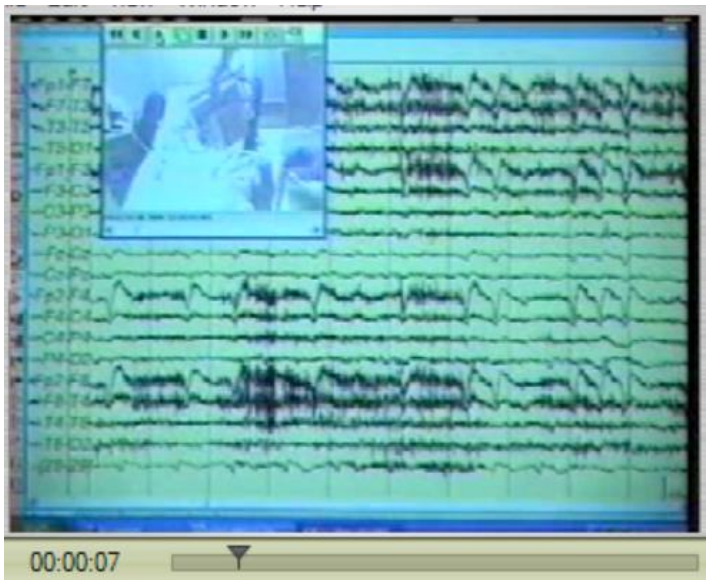
Representative panels

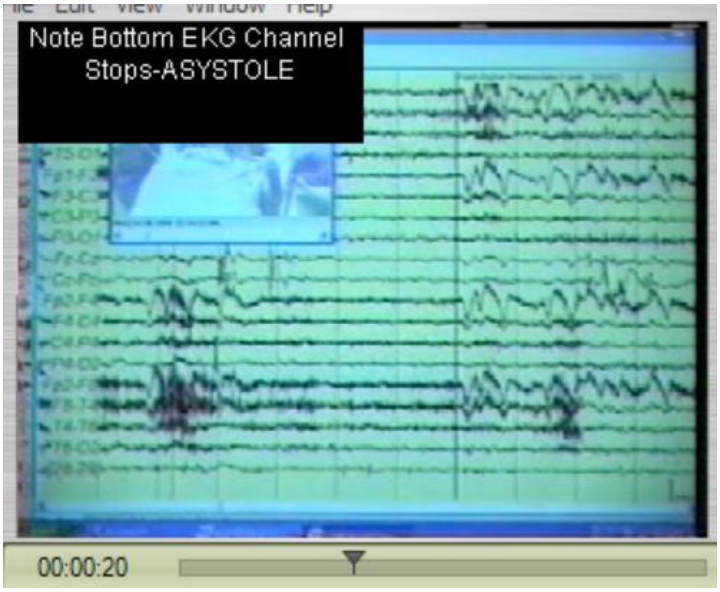
CASE 1 :

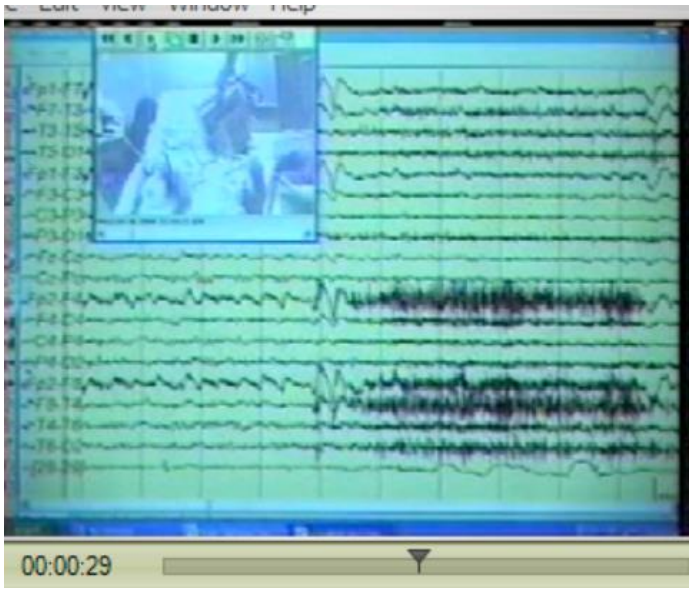
EEG sensitivity for all panels is: 7 uV/mm, LFF = 1.0 Hz, HFF = 70 Hz, 60 Hz Notch filter

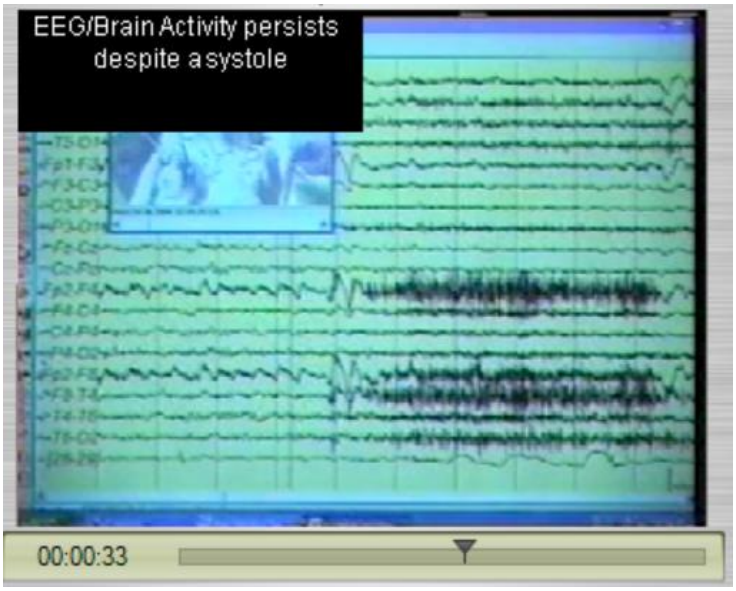


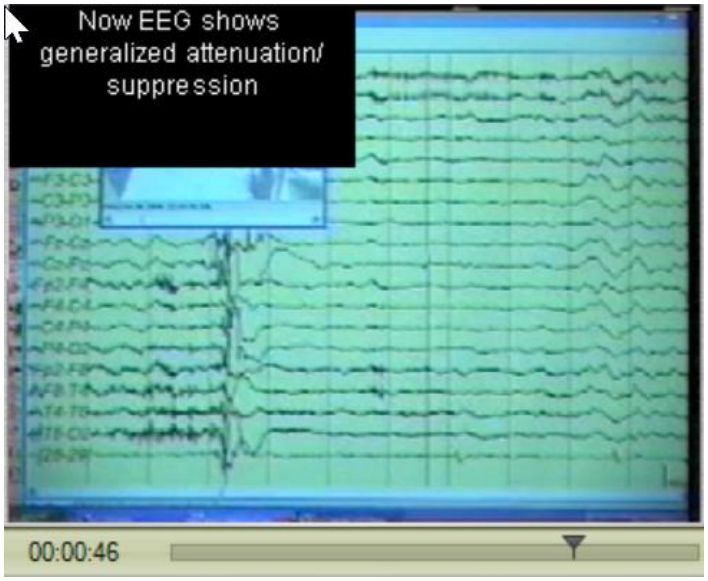


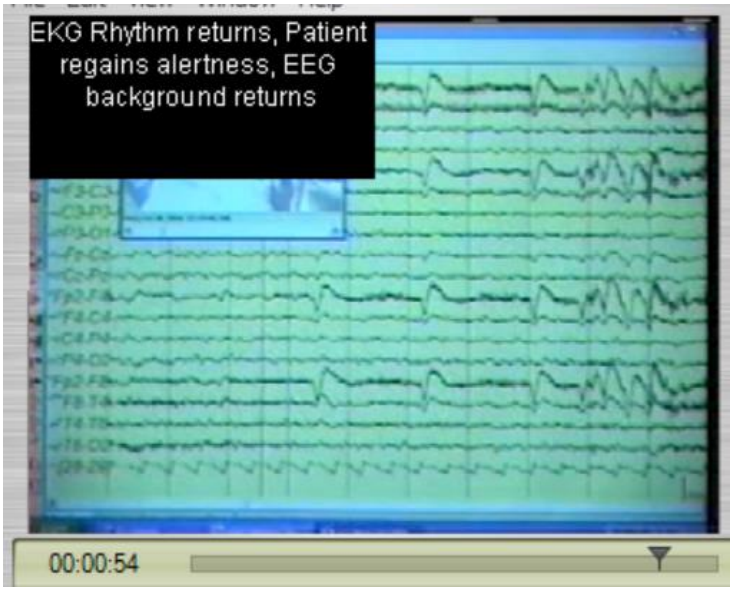






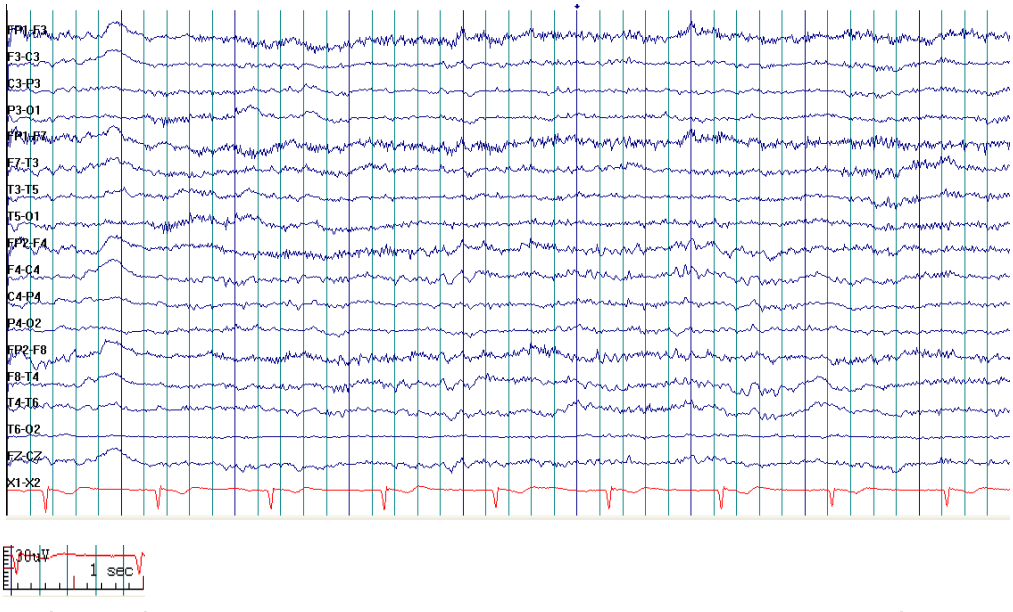




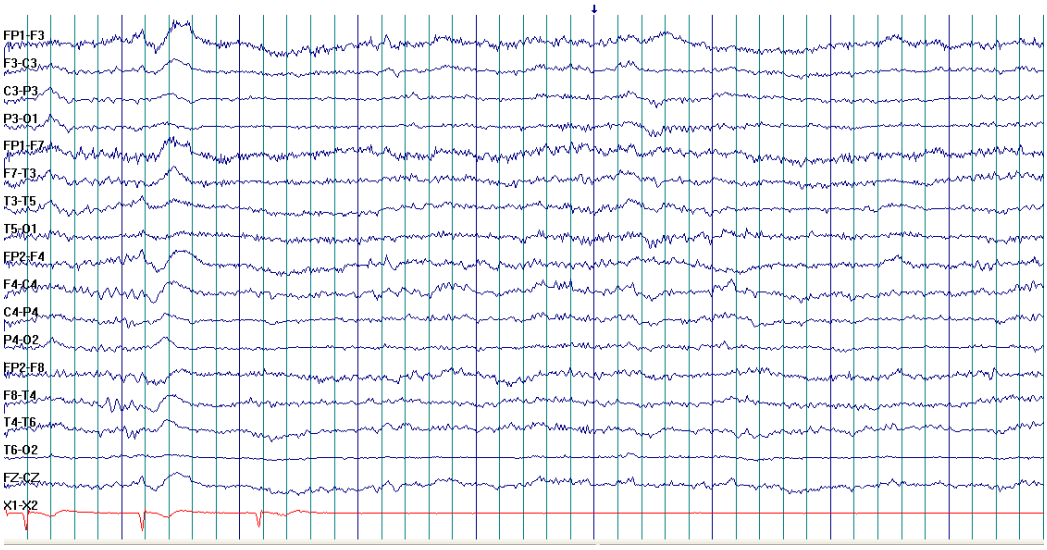


CASE 2:

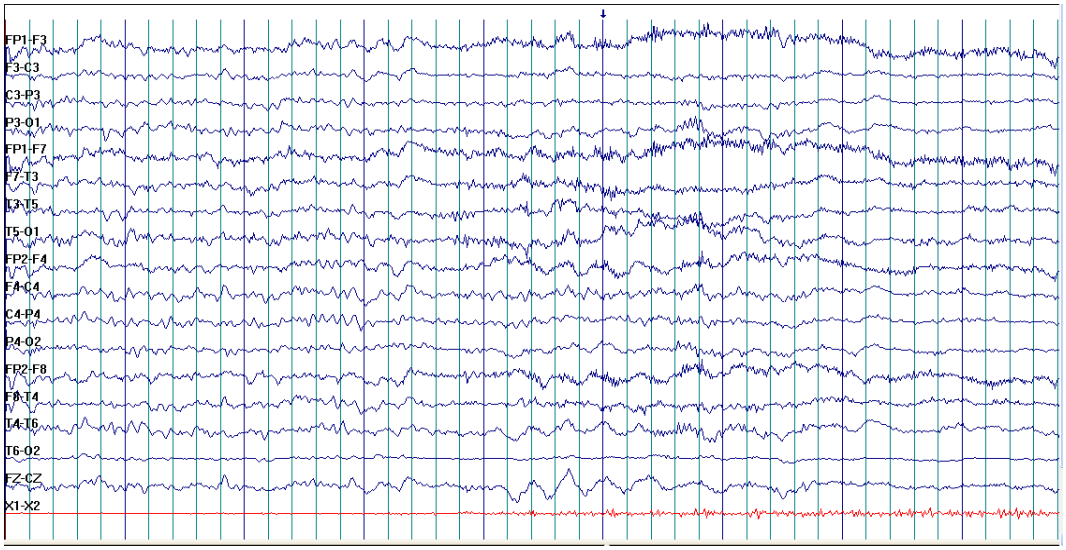
EEG sensitivity for all panels is : 3 uV/mm, LFF = 1.0 Hz, HFF = 70 Hz, 60 Hz Notch filter



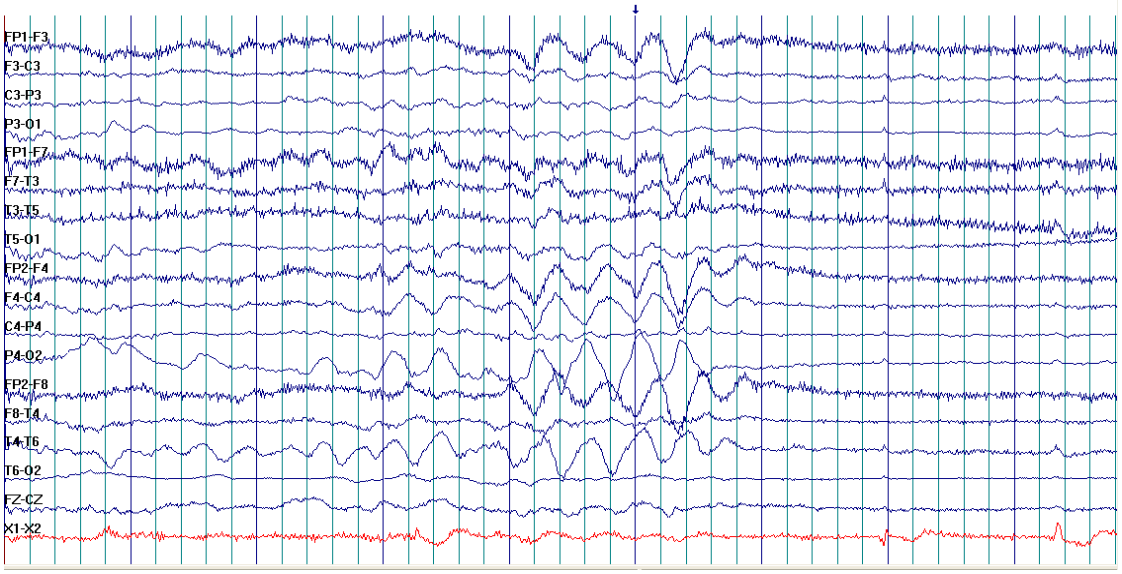
Note the presence of mixed frequencies on the EEG and a predominantly regular EKG derived from 2 electrodes placed at shoulders, approximating the Einthoven II electrode.



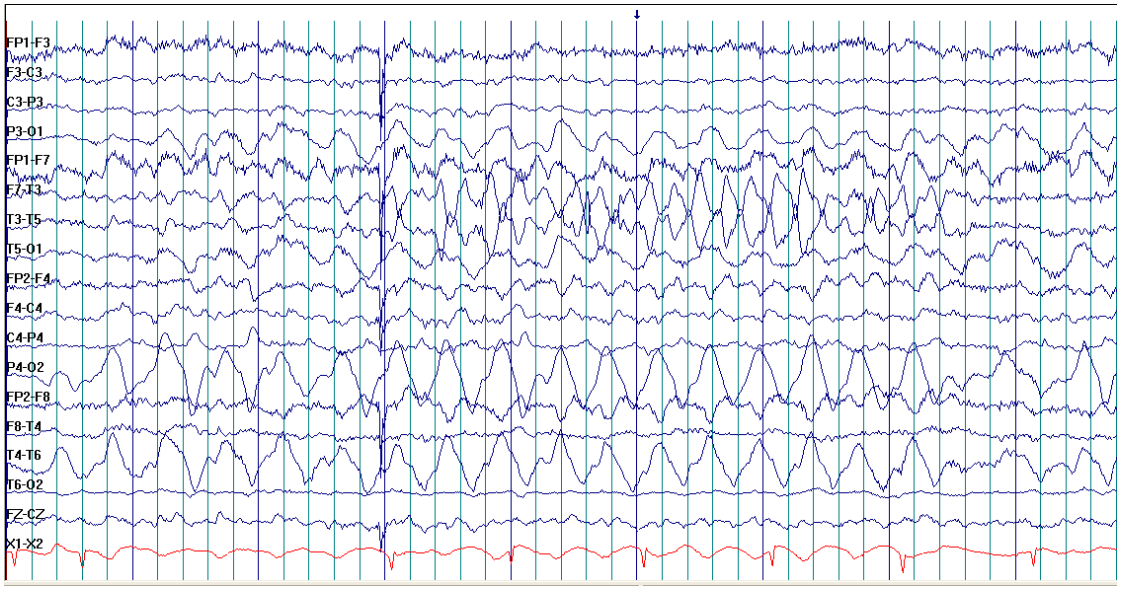
Note the cardiac arrest. There is persistence of electro cerebral rhythms in the EEG derivations.



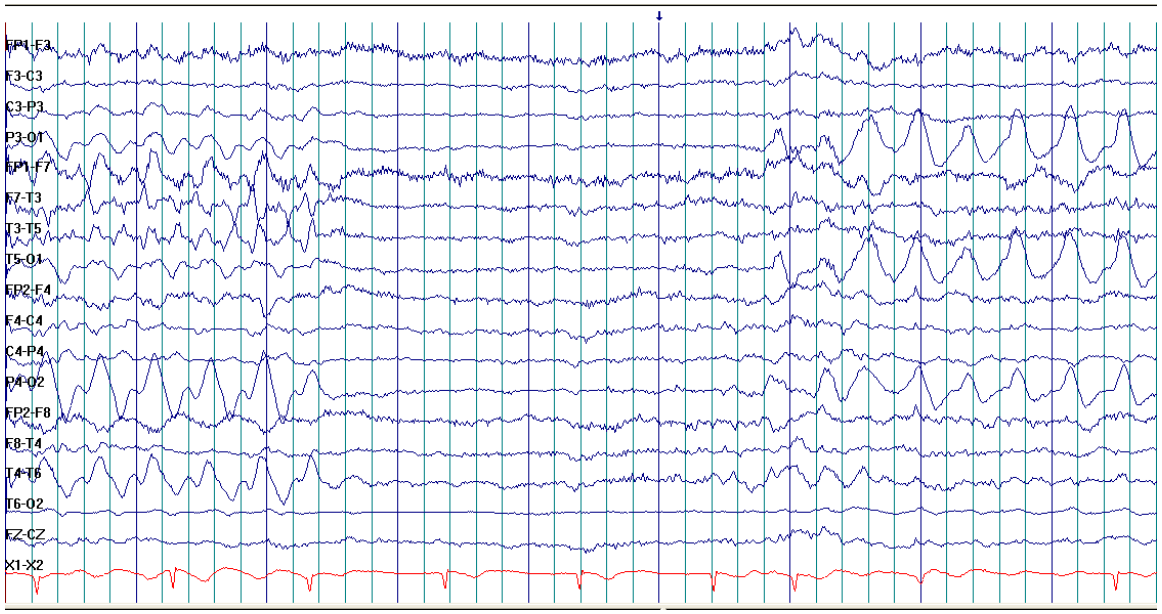
Persistence of electro cerebral rhythms on EEG, beginning of movement artifact as patient begins to move likely due to hypo perfusion.



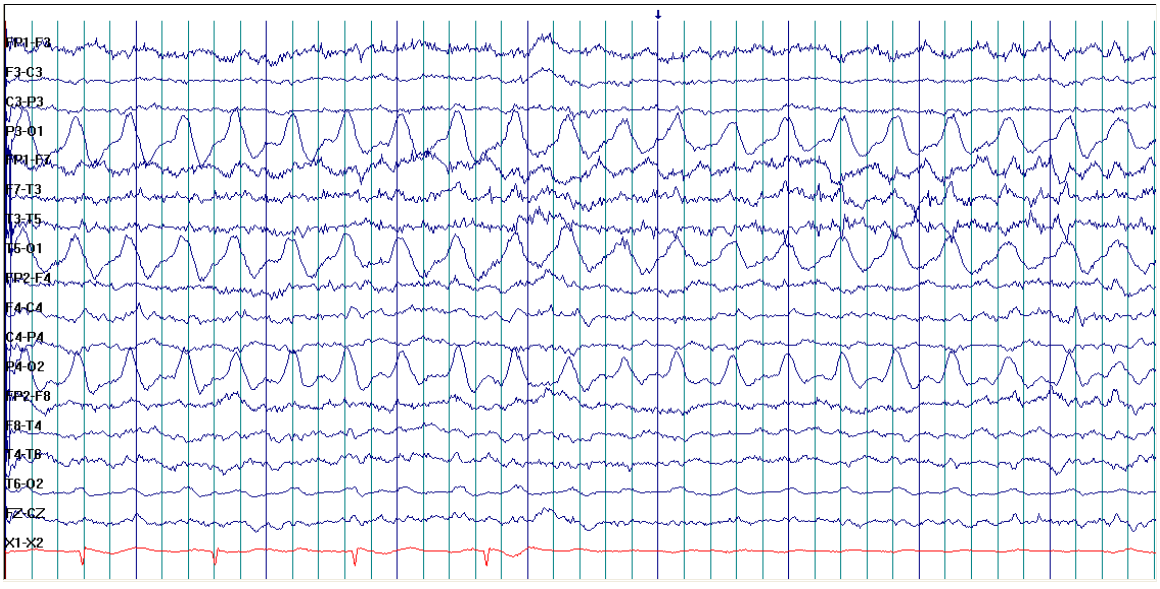
Relative attenuation of the background and presence of movement artifact, occasional irregular QRS complex but no sustained cardiac output is noted.



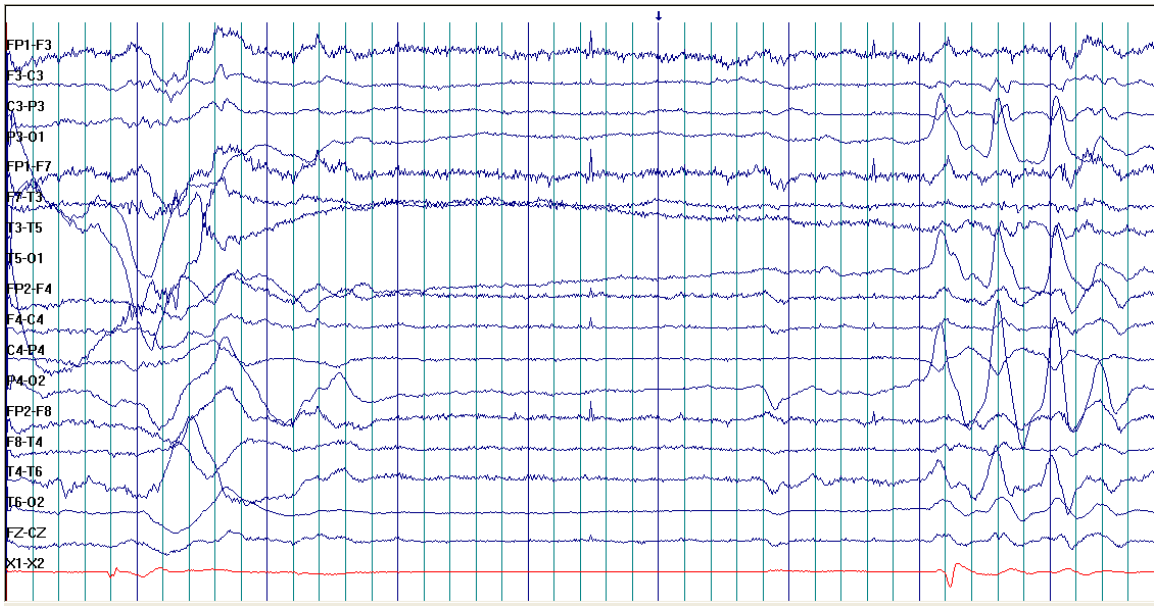
CPR is begun, EKG rhythm returns, electro cerebral rhythms return among movement artifact from chest compressions. These panels for the first and second cases were similar, however CPR was not performed in the first case and there was no subsequent seizure activity as in the second case(see below.)



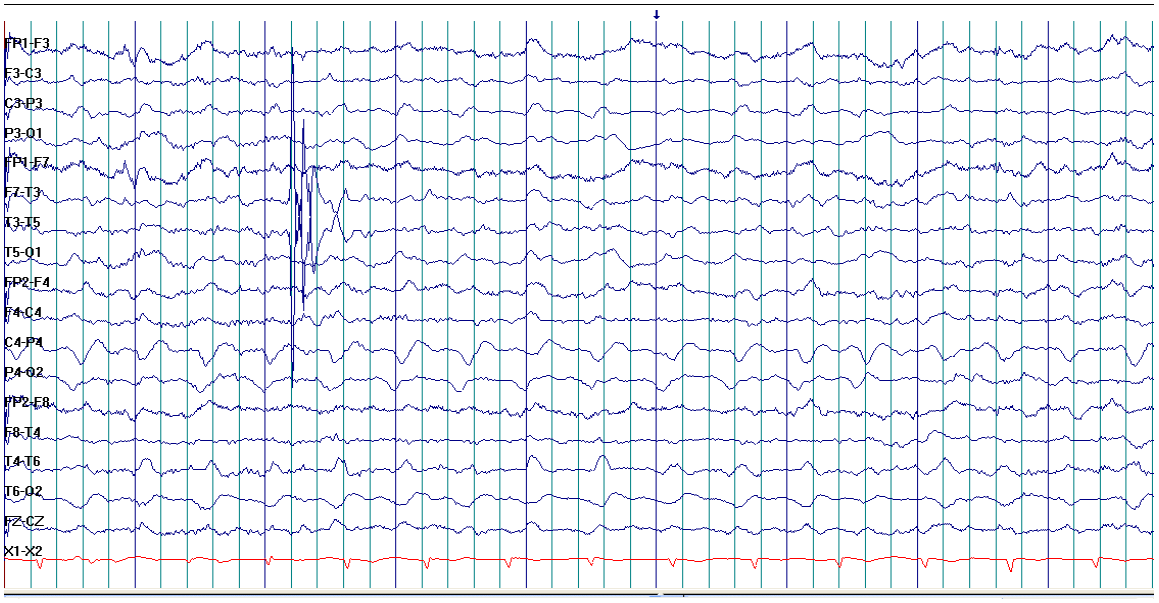
Intermittent chest compression and electro cerebral rhythms and EKG rhythms are noted.



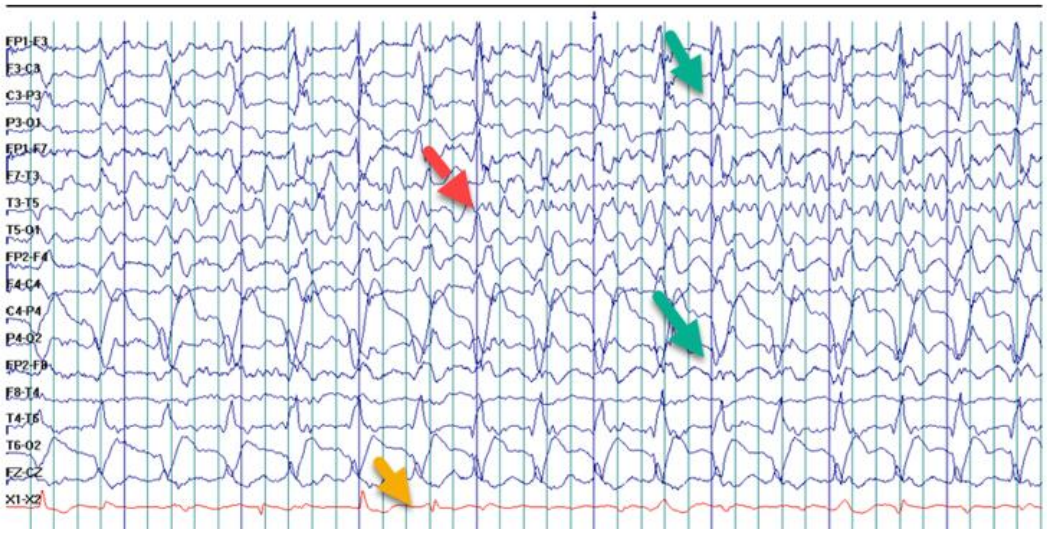
A second cardiac arrest/asystole occurs with persistence of electro cerebral rhythms and movement artifacts for about 24 seconds see next panel- followed by attenuation of the EEG background.



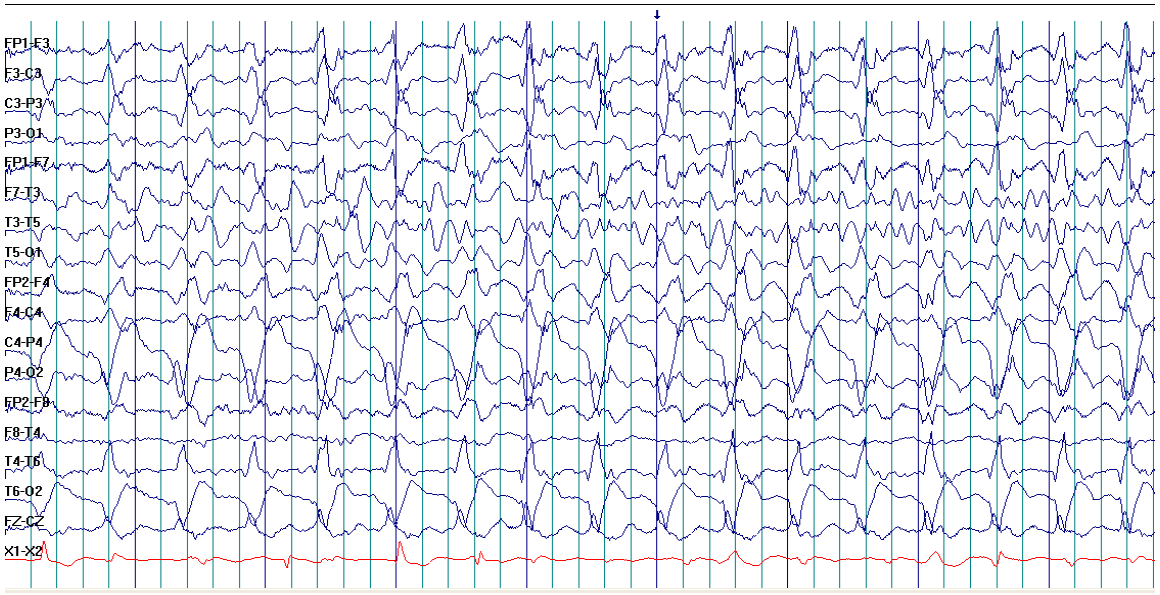
Note the asystole continues and there is diffuse attenuation of the EEG. Chest compressions are begun at the latter portion of this panel.



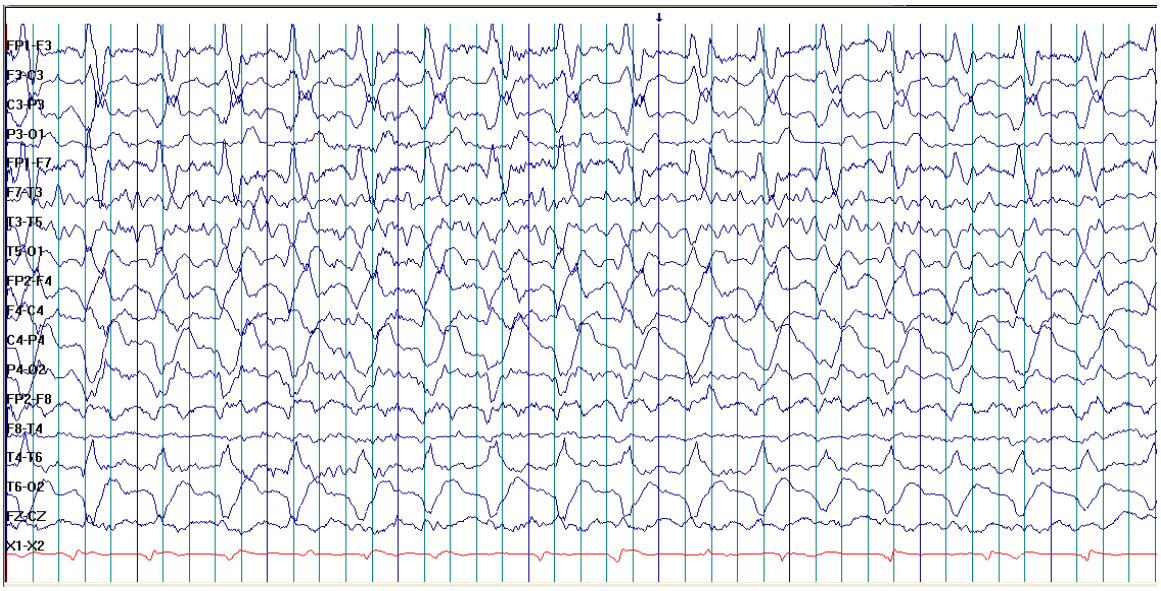
EKG rhythm returns again, EEG background noted- slowed compared to previously.



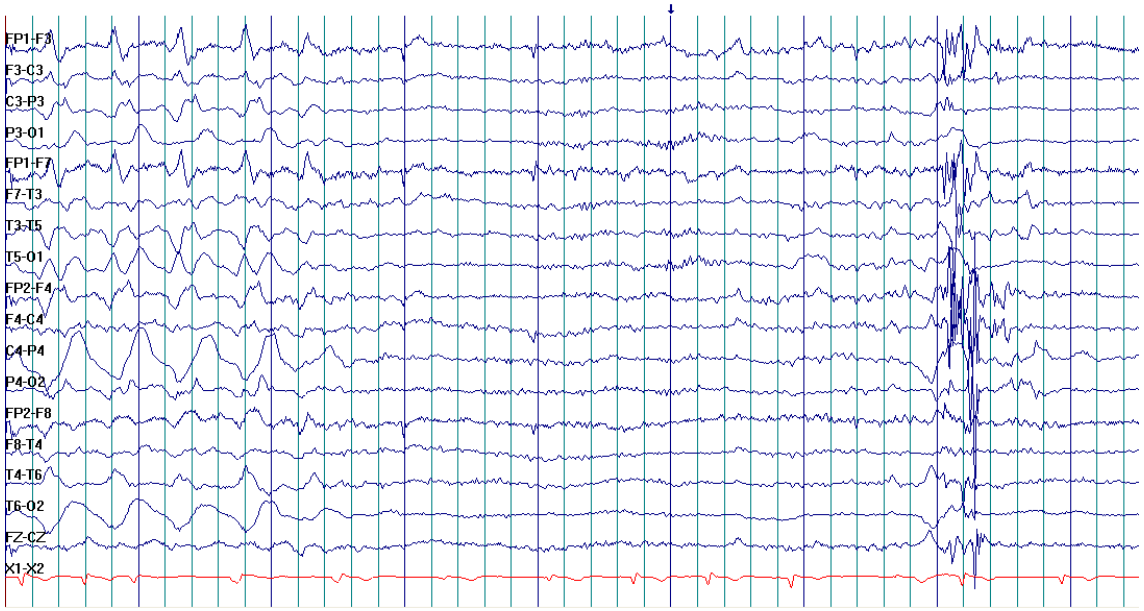
EKG channel(yellow arrow) persists in its irregularities as both more generalized cortical irritability (green arrows) organizes into ictal continuum with generalized periodic discharges reaching approximately 2 Hz as an additional focal seizure (red arrow) at T3-T5 also emerges



EKG channel persists in its irregularities and likely organized generalized epileptiform discharges organize into the beginning of a diffuse electrographic seizure.



Electrographic seizure activity continues, EKG channel shows irregular polymorphic QRS complexes.



Termination of Electrographic seizure, continuation of the irregular but present EKG rhythm is noted.

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