BRIEF COMMUNICATION

Electroencephalography Attenuations in Adults: Clinical Correlates

Gary Hunter, Ryan Verity

ABSTRACT: Spontaneous intermittent generalized attenuations (SIGAs) are defined as a transient decrease in amplitude of electroencephalography (EEG) activity in response to a physiologic process, external stimuli, or as a result of a pathologic condition. We seek to investigate their relationship to clinical outcomes. Demographic information, modified Rankin Scale (mRS), and clinical information were noted on 22 consecutive patients with SIGAs on their EEG. 12 of the 22 patients (54.5%) died, and 12 patients (54.5%) were admitted to the intensive care unit or coronary care unit. Future studies should attempt to prospectively compare outcomes among patients with SIGAs against a control group.

RÉSUMÉ : Atténuations de l'activité électrique du cerveau chez l'adulte : des corrélats cliniques. On peut définir les « atténuations spontanées intermittentes généralisées » (dites « SIGA ») comme une diminution transitoire de l'amplitude de l'activité électrique du cerveau en réponse à un processus physiologique ou à des stimulus externes ou bien encore en lien avec un état pathologique. Notre objectif est ici d'étudier la relation existante entre ce phénomène d'atténuation et l'évolution de l'état clinique d'un certain nombre de patients. Pour ce faire, nous avons colligé, dans le cas de 22 patients vus consécutivement et donnant à voir de telles atténuations lors d'un électroencéphalogramme (EEG), leurs caractéristiques démographiques, leurs scores à la *Modified Rankin Scale* et des renseignements de nature clinique. Au total, 12 patients sur ces 22 sont décédés (54,5 %) ; de plus, 12 patients sur ces mêmes 22 (54,5 %) avaient été admis à une unité de soins intensifs (USI). À ce sujet, de futures études devraient tenter de comparer de manière prospective les résultats obtenus chez des patients manifestant de telles atténuations aux résultats d'un groupe formé de témoins.

Keywords: EEG, Neurocritical care, Brain function, Cerebral metabolism, Clinical neurophysiology, COMA, Electroencephalography, Ion channels

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Spontaneous intermittent generalized attenuations (SIGAs) are defined as a transient decrease in amplitude of electroencephalography (EEG) activity in response to a physiologic process, external stimuli, or as a result of a pathologic condition[1]. However, these are infrequently described and are not included in standard EEG terminology[2]. These EEG attenuations have been previously described in children, mainly as an epileptic phenomenon in syndromic epilepsies (e.g. Lennox–Gastaut syndrome, West syndrome) also known as electrodecremental events (EDE)[3]. However, the significance of SIGAs has not been identified in adults[4]. Diffuse EEG attenuations in adults have been noted by electroencephalographers without definite clinical associations or formal study. Anecdotal evidence suggests there may be an association with the triphasic waves, generally seen in the setting of metabolic encephalopathy[5].

We have observed that severe metabolic encephalopathies appear to be associated with SIGAs in hospitalized patients. We, therefore, hypothesize that EEG attenuations in adults may be of prognostic value in the assessment of metabolic encephalopathies. We seek to further investigate their relationship to triphasic waves and other markers of metabolic encephalopathy, and to characterize underlying etiologies.

This is a prospective case series. We reviewed EEG and clinical features from 22 consecutive adult patients who underwent EEG as part of their diagnostic evaluation and demonstrated SIGAs. SIGAs were defined as transient, infrequent, and intermittently generalized decreases in EEG voltages lasting at least 500 msec, with at least 50% decrease from baseline amplitude, and were felt to clearly be discrepant from background activity. Patients taking anesthetic agents and those with preexisting epilepsy were excluded. All pertinent medications are listed in Table 1. Demographic information, baseline and discharge modified Rankin Scale (mRS), diagnosis at discharge, EEG altering medications, intensive care unit (ICU) admissions, relevant imaging, mental status, discharge destination, pertinent lab values, and metabolic abnormalities were noted. Each patient's EEG was then re-evaluated by an EEG-certified neurologist. The neurologist began by identifying if attenuations were indeed present, duration of the attenuation (msec), background amplitude and frequency, presence of spikes and/or triphasic waves, and temporal relationship to these discharges. Ethics approval was obtained from our local biomedical research ethics board.

The assessment includes 22 patients, 7 males, and 15 females. Examples of EEG attenuations from Patient 16 and Patient 18 (with triphasic waves) are shown in Figures 1 and 2, respectively. Mean patient age was 73.7 ($SD = 11.0$) at admission. Mean mRS score at baseline was 1.4 ($SD = 1.4$), and was 4.8 ($SD = 1.5$) at discharge (Table 1). Twelve of the 21 patients (54.5%) died

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Table 1: Relevant clinical information

ID	Baseline mRS	Discharge mRS	Discharge location	Diagnosis at discharge	Mental status	ICU (Yes/No)	EEG relevant medications	Pertinent investigations
Patient 1	2	4	Long-Term Care	Intracerebral Hemorrhage	Drowsy	N	Morphine, Clonidine	Left Intracerebral Hemorrhage
Patient 2	0	6	Death	Sepsis	Drowsy	Y	Piperacillin and Tazobactam Sodium	Septic, Elevated White Blood Cells
Patient 3	0	3	Long-Term Care	Cardiac Arrest	Confused	Y	Lorazepam	Post-Cardiac Arrest
Patient 4	0	6	Death	Subarachnoid Hemorrhage and Intraventricular Hemorrhage	Coma	Y	Phenytoin	Subarachnoid Hemorrhage, Intraventricular Hemorrhage
Patient 5	2	6	Death	Stroke	Drowsy	Y	Clonidine	Sepsis, Hypoxia, Brainstem Stroke
Patient 6	0	6	Death	Stroke	–	Y	–	Bilateral, Diffuse Strokes
Patient 7	1	6	Death	Brain Metastasis	Drowsy	N	Gabapentin	Widespread Metastasis
Patient 8	2	5	Home Hospital for End-of-Life Care	Stroke	Drowsy	Y	–	Stroke
Patient 9	0	1	Home	Encephalitis	Drowsy	N	–	Pleocytosis in CSF, DWI Left Temporal Region
Patient 10	1	1	Home	Drug Overdose	Drowsy	N	Baclofen	–
Patient 11	0	6	Death	Septic Shock	Drowsy	Y	–	Sodium 120, Elevated Liver Enzymes
Patient 12	4	4	Long-Term Care	Seizure	Drowsy	N	Phenytoin	–
Patient 13	1	5	ICU at local Hospital	Encephalopathy	Drowsy	Y	Lithium, Midazolam	–
Patient 14	2	6	Death	Seizures	Stupor	N	Clobazam	–
Patient 15	4	4	Long-Term Care	Encephalopathy	Drowsy	N	Phenytoin	Right Meningioma
Patient 16	3	6	Death	Cardiac Arrest	Coma	Y	–	Uremic
Patient 17	0	6	Death	Seizures and Paraneoplastic Syndrome	Drowsy	Y	Phenytoin	T2 Right Hippocampus
Patient 18	0	6	Death	Intracerebral Hemorrhage	Drowsy	Y	–	Small Intracerebral Hemorrhage
Patient 19	0	6	Death	Intracerebral Hemorrhage and Renal Failure	Drowsy	Y	–	Uremic, Intracerebral Hemorrhage Right Thalamus
Patient 20	4	4	Long-Term Care	Encephalopathy	Drowsy	N	–	Hypernatremia, Uremic
Patient 21	3	4	Long-Term Care	Seizures and Stroke	Drowsy	N	Phenytoin, Gabapentin	–
Patient 22	2	6	Death	Seizure	Drowsy	N	Lorazepam, Phenytoin	Uremic

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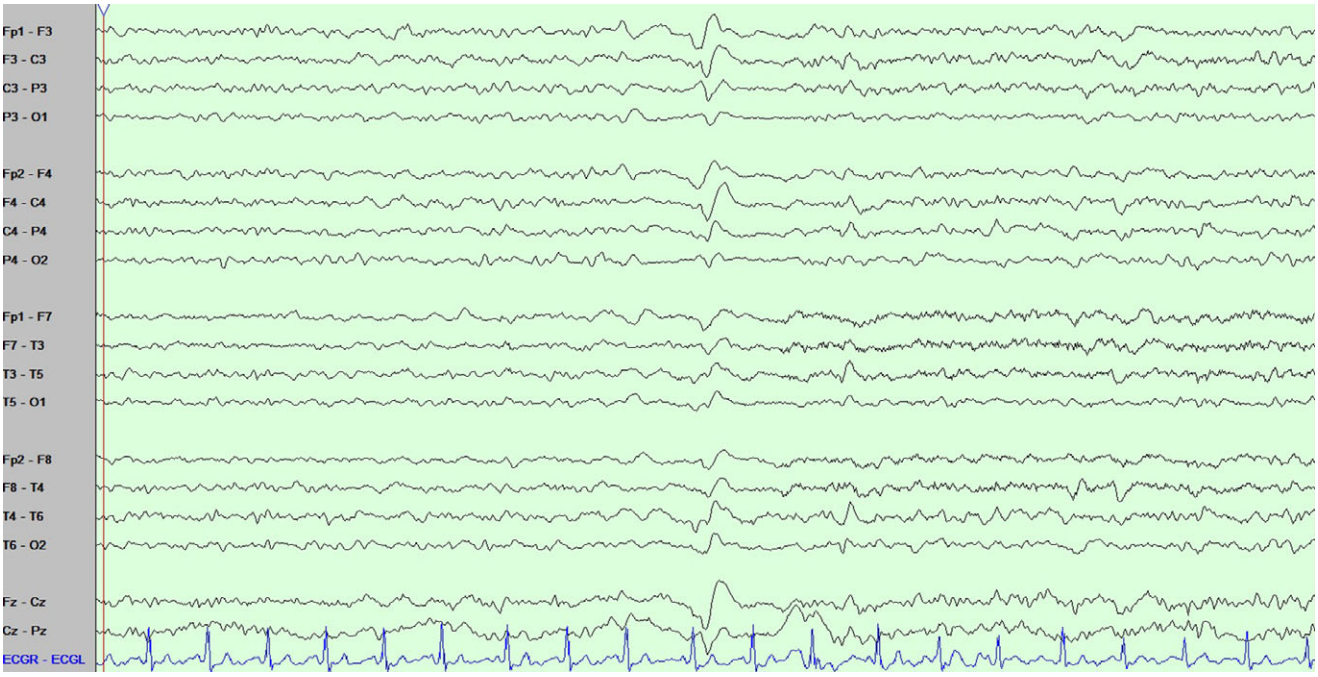

Figure 1: *EEG with attenuations and tiphasic waves from Patient 16 post-cardiac arrest.*

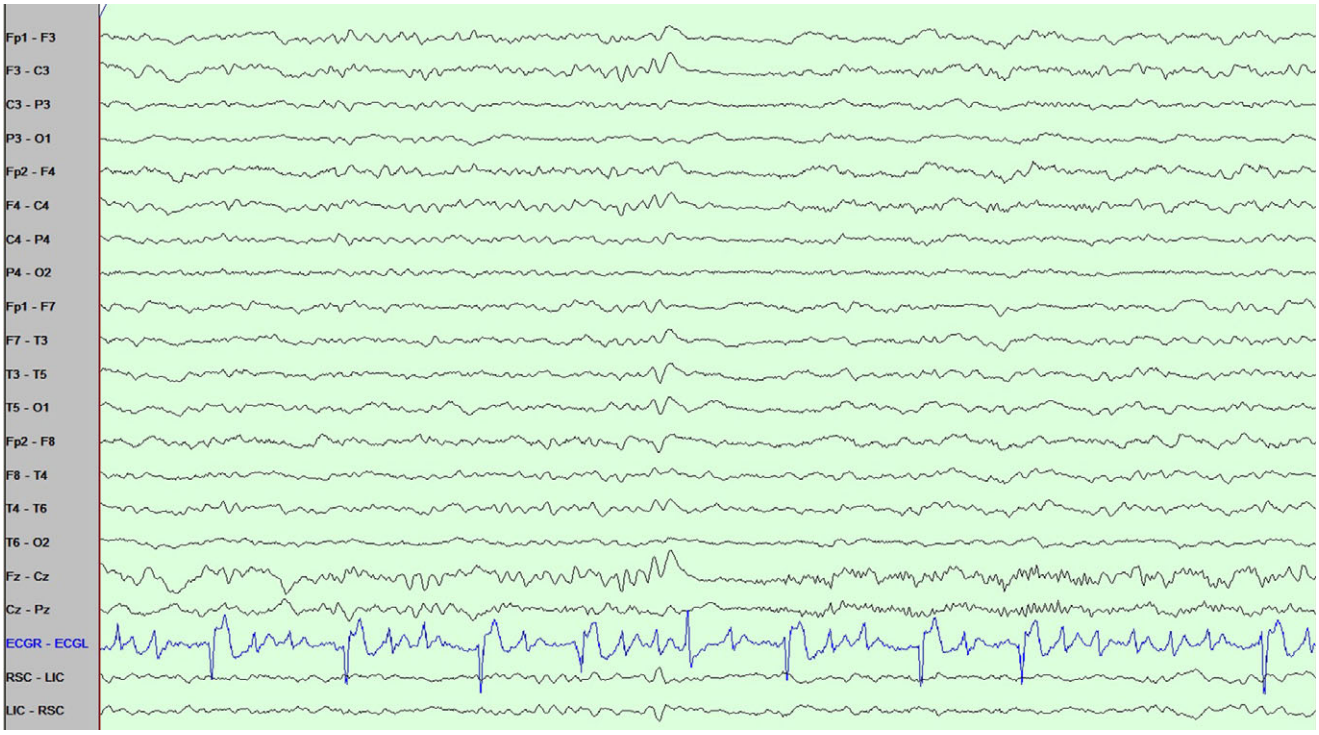

Figure 2: *EEG with attenuations and triphasic waves from Patient 18, who had an intracerebral hemorrhage.*

during their course in hospital as a result of their illness. Four patients (18.2%) did not have a change in mRS score from baseline to discharge. The mean discharge mRS was 4.8 (*SD* = 1.5). Eighteen patients were drowsy at the time of their EEG, 2 were in a coma, and the other 2 were described as nonspecifically encephalopathic. Twelve patients (54.5%) were admitted to the ICU or coronary care unit during their time in hospital. Only three patients were discharged home. Other clinical information is further described in Table 1. Mean SIGA duration was 1300 msec (*SD* = 403 msec). Fourteen of the 22 (63.6%) patients showed

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Table 2: EEG data

	Background (Hz)	Spikes (Yes/No)	Triphasics (Yes/No)	Triphasics preceding attenuation (Yes/No)	Duration of attenuation	EEG classification (Mayo clinic EEG scheme)
Patient 1	6	Y	Y	Y	660	V
Patient 2	5	N	Y	Y	1000	V
Patient 3	6	Y	N	N	1500	IV
Patient 4	Alpha coma	N	N	N	1000	V
Patient 5	Alpha coma	N	Y	N	1100	V
Patient 6	7	N	Y	Y	1200	V
Patient 7	6	N	Y	Y	1000	V
Patient 8	5	N	Y	N	2100	V
Patient 9	8	N	N	Y	1500	II
Patient 10	5	N	Y	Y	2200	V
Patient 11	6	N	Y	Y	1300	V
Patient 12	6	N	Y	Y	1800	V
Patient 13	6	N	Y	Y	1300	V
Patient 14	8	N	Y	N	1800	V
Patient 15	6	N	Y	Y	1600	V
Patient 16	5	N	Y	Y	1400	V, Theta coma
Patient 17	5	Y	N	N	1300	IV
Patient 18	6	N	Y	Y	1000	V
Patient 19	6	Y	Y	Y	1500	V
Patient 20	6	N	N	N	1200	V, Theta
Patient 21	6	Y	N	N	1000	V
Patient 22	7	N	Y	Y	600	V

IGAs immediately following a triphasic wave. All patients except one showed a slowing of background activity. Epileptiform activity was seen in four patients. Other EEG findings are detailed in Table 2. Underlying diagnoses were variable and included both primary brain pathologies (stroke, hemorrhage, seizures; $n = 14$) and systemic illness (metabolic encephalopathies $n = 8$). The primary brain pathology group had a mean discharge mRS of 5.1 ($SD = 1.5$) and the systemic illness group had a mean discharge mRS of 4.4 ($SD = 1.8$).

As EEG attenuations have previously not been well described in adults, we sought to understand their clinical associations and prognostic significance. The most striking finding from this review is the strong association with mortality and persistent disability at discharge, with a majority of survivors discharged to some form of supportive care rather than home. While these outcomes are difficult to distinguish from the underlying disease prognosis, the association appears to support this EEG finding as a marker of severe brain dysfunction.

The underlying pathophysiology of SIGAs remains unclear. Generalized attenuation is likely associated with transitory dysfunction in the setting of reduced cortical synchronicity[6]. The duration of the attenuations may support potassium channel dysfunction, secondary to a burst release of calcium[7]. The length of the attenuations was mostly around the 1300 msec mark, with variance as short as 600 msec and as long as 2200 msec. Length of attenuation did not seem to correlate to the prognostic outcome. In children, it is hypothesized that diffuse attenuations are triggered by the activation of the arousal circuit in the setting of diffuse brain dysfunction[8]. SIGAs often followed a triphasic waveform, and most patients showed triphasic waves at some point during the recording. The apparent association with triphasic waveforms supports the idea of an underlying metabolic encephalopathy, and triphasics with attenuations may represent a more advanced stage of encephalopathy.

Limitations of this study include a lack of control group and small sample size. The aim of the current study was to describe this EEG finding and the associated clinical and electrographic features, as well as outcomes. Causation and independent prognostic value cannot yet be determined from this study.

From this data, it seems probable that SIGAs is a meaningful EEG finding that should be noted and reported by electroencephalographers, and that they are likely associated with encephalopathies and poor outcomes.

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Statement of Authorship

GH: responsible for literature review, patient enrollment, ethics approval, EEG review and interpretation, and manuscript writing/editing.

RV: responsible for literature review, data collection, and manuscript writing/editing.

Disclosures

The authors have no conflicts of interest to declare.

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