Continuous Monitoring of Electrodermal Activity During Epileptic Seizures Using a Wearable Sensor

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Abstract—We present a novel method for monitoring sympathetic nervous system activity during epileptic seizures using a wearable sensor measuring electrodermal activity (EDA). The wearable sensor enables long-term, continuous EDA recordings from patients. Preliminary results from our pilot study suggest that epileptic seizures induce a surge in EDA. These changes are greater in generalized tonic-clonic seizures and reflect a massive sympathetic discharge. This paper offers a new approach for investigating the relationship between epileptic seizures and autonomic alterations.

I. INTRODUCTION

EPILEPSY is a common neurological disorder that involves repeated and spontaneous seizures. These seizures are the manifestation of abnormal, excessive or synchronous neuronal activity in the brain. Epileptic seizures are often associated with significant changes in autonomic nervous system (ANS) functioning [1]. Autonomic signatures such as flushing, sweating and piloerection often accompany partial seizures and auras [2-4]. In contrast, generalized tonic-clonic seizures (GTCS) are associated with severe increases in blood pressure and changes in heart rate and cardiac conduction [5]. Seizure-induced autonomic dysfunction can have serious clinical consequences and potentially fatal effects when the cardiovascular or respiratory systems are involved [6].

There are distinct differences between the sympathetic and parasympathetic divisions of the ANS. While parasympathetic discharges produce responses to promote restoration and conservation of energy, the sympathetic nervous system increases metabolic output to adjust to external challenges. So far, autonomic alterations in epilepsy have mostly been studied using indirect parameters such as heart rate, respiratory rate and blood pressure changes that are dually modulated by both divisions of the ANS [5, 7-9]. Spectral analysis of heart rate variability (HRV) can provide a sensitive index of cardiac parasympathetic control via the

vagus nerve [10], but its utility for uncoupling sympathetic activity remains controversial [11, 12]. On the other hand, sympathetic postganglionic fibers consisting of non-myelinated class C nerve fibers surround eccrine sweat glands and their activity modulates sweat secretion [13]. Thus, modulation in skin conductance, referred to as electrodermal activity (EDA), is a unique parameter that reflects purely sympathetic activity [14, 15].

Several cortical structures with recognized seizure potential have direct or indirect connections with autonomic centers from the medulla oblongata [16]. Studies have shown that electrical stimulation of such structures can induce changes in EDA [17, 18]. For example, stimulation of the cingulate gyrus in humans produced strong palmar skin conductance responses. Increases in skin conductance were also observed when the frontal cortex was stimulated. As such, we decided to perform long-term monitoring of EDA activity in patients with epilepsy to investigate ictal-related changes in sympathetic activity. Our working hypothesis was that epileptic seizures, GTCS in particular, might induce large changes in EDA reflecting strong ictal sympathetic discharges. To this end, we designed a wearable EDA sensor suitable for long-term monitoring [19].

In this pilot study, we investigate changes in EDA during epileptic seizures. A wearable wristband EDA sensor is described and used to collect long-term, continuous recordings from patients with epilepsy. Preliminary observations of ictal changes in EDA parameters are presented. Since GTCS is reported as the most consistent risk factor for sudden, unexpected death in epilepsy (SUDEP) [20], we evaluated the difference in EDA changes between GTCS and complex partial seizures (CPS). Finally, we discuss the findings and future work.

II. METHODS

A. Description of the wearable EDA sensor

Our sensor measures exosomatic EDA (skin conductance) by applying direct current to the stratum corneum of the epidermis beneath measuring electrodes. To achieve a wide dynamic range of skin conductance measurements, the analog conditioning circuitry utilizes non-linear feedback automatic bias control with low-power operational amplifiers. In addition, the sensor module also contains a triaxis accelerometer for measurements of physical activity (actigraphy). A microcontroller digitizes the analog signals via a 12-bit A-D at a sampling frequency of 20 Hz. The data
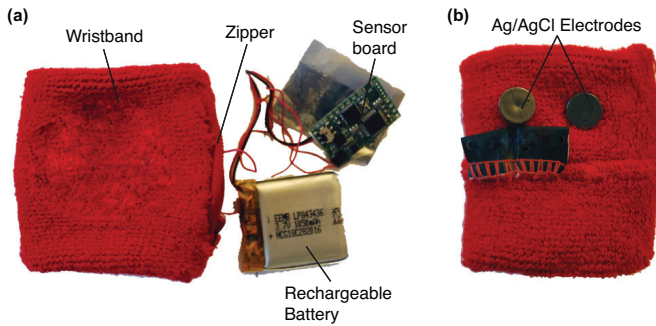
Fig. 1. Overview of the wearable EDA sensor. (a) The sensor board and rechargeable battery are packaged into a regular terry cloth wristband and can be accessed via a zipper. (b) The inside surface of the wristband contains two Ag/AgCl electrodes used to measure skin conductance.

is then written to an onboard microSD card.

We integrated the sensor module into a regular wristband made out of terrycloth, resulting in a comfortable, attractive and lightweight wearable sensor (Fig. 1). Since all electronics and wiring are concealed within the wristband, the resulting device is inconspicuous, non-stigmatizing and allows for discrete monitoring of EDA. Furthermore, the electronic module can be easily detached when the user desires to wash the wristband. We used Ag/AgCl disc electrodes with contact areas of 1.0 cm^2 for our recordings as recommended in the literature [21]. These electrodes are disposable and can be snapped onto or removed from the wristband with ease. Although the electrodes are commonly placed on the palmar surface of the hand (e.g. medial and distal phalanges of the fingers and the thenar and hypothenar eminences), we use the ventral side of the distal forearms as recording sites. Placement of electrodes on the forearm are less susceptible to motion artifacts and highly correlated to palmar recordings [22]. A 3.7 V lithium polymer battery with a capacity of 1100 mAh provides around 40 hours of operation; the battery can be recharged via a micro-USB cable.

B. Data collection

This study was approved by the Institutional Review Boards (IRB) of the Massachusetts Institute of Technology and Children's Hospital Boston. We enrolled patients with epilepsy who were admitted to the hospital's long-term video-telemetry monitoring (LTM) unit for assessment. EEG recordings were performed using conventional scalp electrodes (10-20 system) at a sampling rate of 256 Hz. Two modified lead-I ECG (adhesive electrodes placed below the clavicles of both sides) was recorded simultaneously with EEG. EDA sensor wristbands were placed on the wrists such that the electrodes were in contact with the ventral side of the forearms. EDA recordings were sampled at 20 Hz and synchronized with the video/EEG/ECG recordings by generating technical artifacts at the beginning and end of each session for offline realignment. Each recording session lasted approximately 24 hours and batteries were replaced on a daily basis.

TABLE I
CLINICAL DETAILS

Patient	Sex/age/ epilepsy duration (yrs)	Recording duration	Seizure focus	Seizure type
1	M/20/4	4 days	Left frontotemporal	GTCS
2	F/11/7	1 day	Multifocal	GTCS
3	M/9/9	4 days	Left frontotemporal	GTCS
4	M/16/1	3 days	Bifrontocentral	GTCS
5	F/18/1	2 days	Right frontal	CPS
6	F/13/4	1 day	Right occipital	CPS
7	M/19/9	3 days	Right frontal	CPS

GTCS = generalized tonic-clonic seizure; CPS = complex partial seizure

C. Data analysis

Ictal video-EEG recordings were retrospectively reviewed by a team of epileptologists blinded to the EDA data. Each EEG seizure was reviewed for ictal onset and offset times, EEG location and seizure semiology on video recordings. EDA recordings were analyzed using custom written software in MATLAB (The Mathworks, Inc.). EDA recordings were low-pass filtered (1024 points, Hamming window, cut-off frequency of 3 Hz) to reduce motion artifacts. For each seizure, the resulting change in EDA amplitude (difference between response peak and ictal onset baseline) and recovery time was calculated. Recovery time was determined as the time from the response peak to point where EDA fell below 37% of the EDA response peak [14].

D. Statistics

Comparison between EDA parameters during CPS and GTCS were performed using the Wilcoxon rank sum test. p values < 0.05 were regarded as significant.

III. RESULTS

The study group consisted of seven patients (four boys, three girls, age: 15.1 ± 4.2 years) with epilepsy of varying etiology. Table 1 shows a summary of the clinical characteristics. A total of 13 seizures were analyzed, including nine GTCS and four CPS.
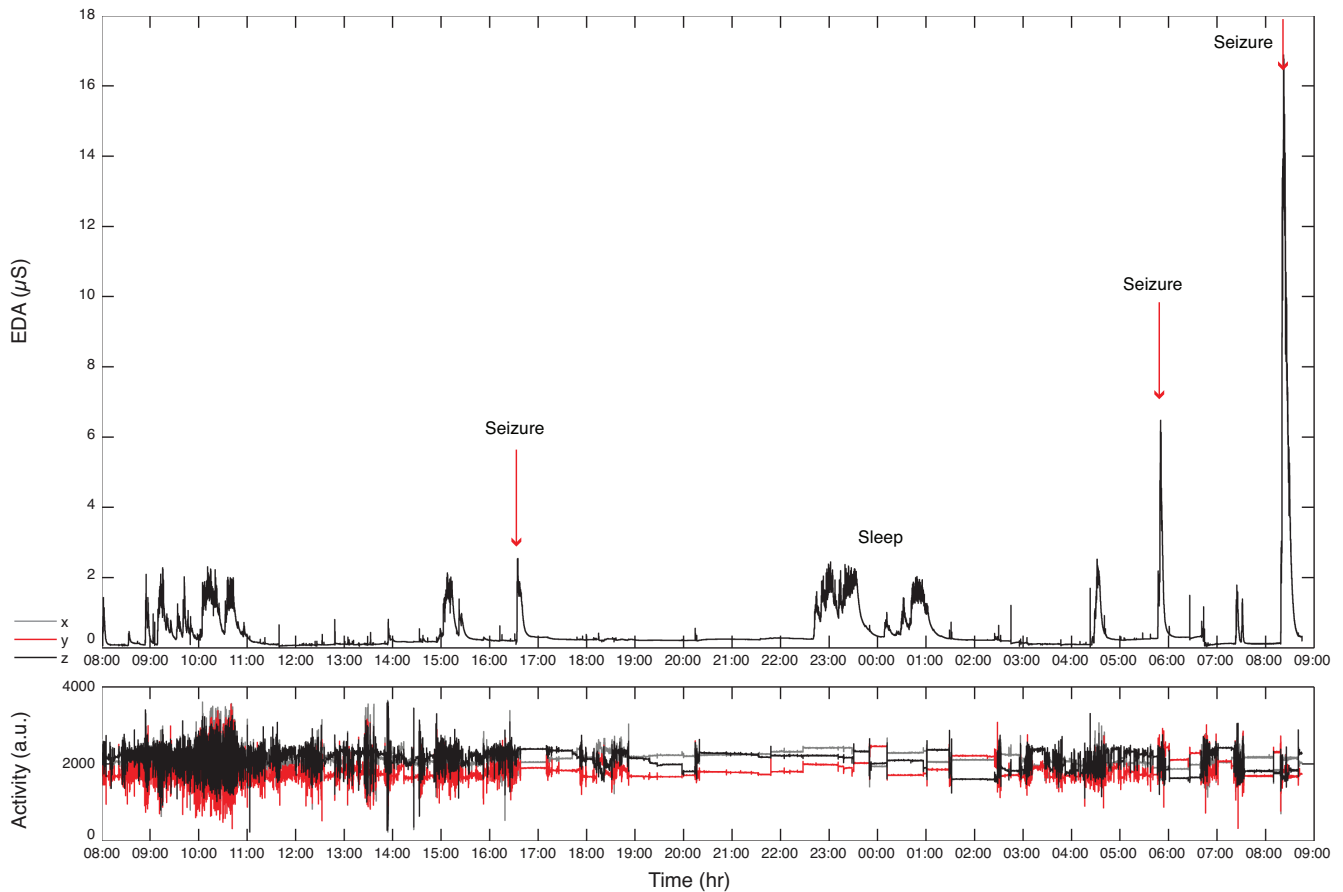
Fig. 3. Example of a 24 hour EDA recording from an 11 y.o. girl with intractable epilepsy. Elevations in EDA can be seen during three EEG seizures.
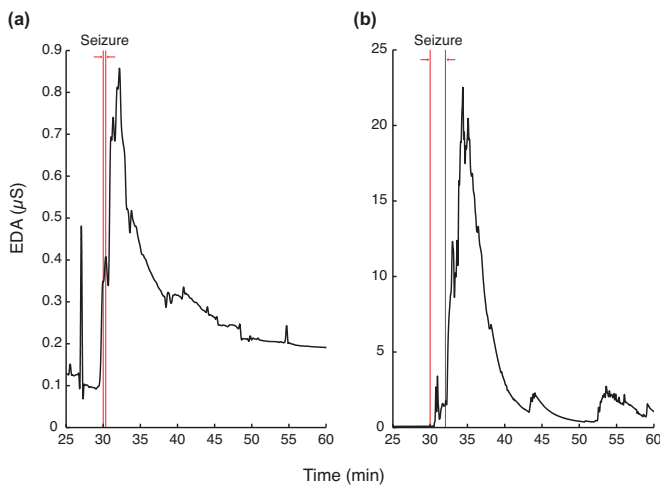

Fig. 4. Example of EDA changes during a (a) CPS and (b) GTCS

Fig. 5. Periictal EDA parameters (a) Amplitude of EDA (b) Recovery time

We successfully recorded long-term, continuous EDA in patients with epilepsy. The total recording time from all included patients was 18 days. Figure 3 shows an example of a 24-hour EDA recording from a patient along with the actigraphy signals that reflect physical activity. The EDA changed dynamically over the recording period and was higher before noon (09:00 to 11:00) and also during sleep (around 19:00 to 02:30). 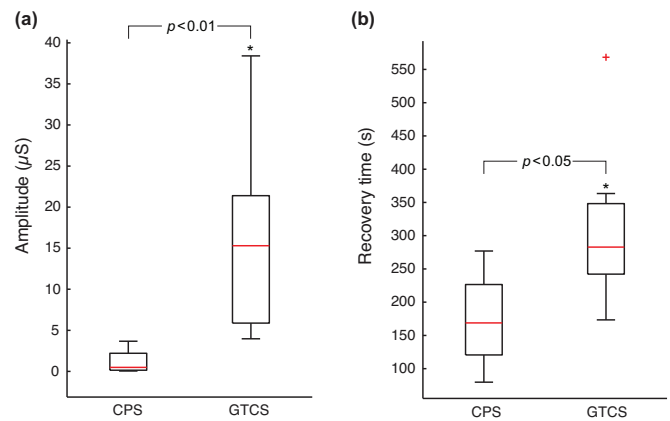Three EEG seizures (indicated by red arrows) occurred during this 24-hour period. EDA was significantly elevated immediately after the onset of each EEG seizure.

Figure 4 shows a close-up of EDA changes during two different seizures types (red lines mark the ictal onset and offset times). The example of a CPS (Fig. 4a) shows that EDA increased around 0.7 µS whereas during the GTCS (Fig. 4b), EDA increased by over 20 µS. Both seizures induced a rapid surge in EDA that continued into the postictal period, and a period of sustained elevation followed

by a slow decay.

We evaluated the differences between EDA parameters during GTCS and CPS. The change in EDA amplitude (Fig. 5a) was significantly higher after GTCS seizures compared to CPS ($p < 0.01$). In addition, EDA remained elevated (Fig. 5b) for a longer time during GTCS compared to CPS ($p < 0.05$).

IV. DISCUSSION

Based on the results from our pilot study, spontaneous epileptic seizures were associated with large increases in EDA. In 1958, Van Buren reported a decrease in skin resistance during seizures [23], which is consistent with our observations. However, an important distinction is that the aforementioned study was performed on induced seizures and the procedures used to precipitate seizures alone often resulted in lowered skin resistance. Furthermore, our measurements were obtained using a new wristband sensor that can record all day and night in a person's natural environment. EDA increases during GTCS were greater than during CPS and were sustained for a longer duration. These findings suggest that a massive sympathetic outflow occurs during GTCS and continues postictally. Reports of elevated plasma catecholamines following GTCS support this indication of a sympathetic surge [24, 25]. In future work, we will perform a full characterization of changes in EDA parameters and study the role of seizure type and ictal onset location.

This is the first long-term EDA study in patients with epilepsy and we presented our initial findings. Seizure-induced EDA elevation is a possible sign of autonomic instability that could play a role in the pathophysiology of SUDEP. We predict that the periods of postictal sympathetic EDA elevation are associated with periods of impaired vagal reactivation. Studies investigating the relationship between EDA and HRV, a risk factor for sudden cardiac death [12], are underway.

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