

Topological Data Analysis of Electroencephalogram Signals for Pediatric Obstructive Sleep Apnea

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Abstract—Topological data analysis (TDA) is an emerging technique for biological signal processing. TDA leverages the invariant topological features of signals in a metric space for robust analysis of signals even in the presence of noise. In this paper, we leverage TDA on brain connectivity networks derived from electroencephalogram (EEG) signals to identify statistical differences between pediatric patients with obstructive sleep apnea (OSA) and pediatric patients without OSA. We leverage a large corpus of data, and show that TDA enables us to see a statistical difference between the brain dynamics of the two groups.

Clinical relevance— This establishes the potential of topological data analysis as a tool to identify obstructive sleep apnea without requiring a full polysomnogram study, and provides an initial investigation towards easier and more scalable obstructive sleep apnea diagnosis.

I. INTRODUCTION

Recent advancements in signal processing technology, including topological data analysis (TDA), provide a powerful method for analysis of EEG signals. TDA leverages ideas from the mathematical field of topology, and applies these ideas to the analysis of real-world signals[5]. Broadly, TDA allows us to exploit the topological and geometric structures inherent to data, and use these structures to study fundamental differences between the EEG signals of obstructive sleep apnea (OSA) positive and OSA negative patients. In this work, we present TDA techniques which allows identification of OSA using only EEG signals.

The key assumption in this work is that the brain connectivity network for OSA positive patients has a fundamentally different topological structure than the brain connectivity network for OSA negative patients.

Existing techniques for the identification of OSA in children involve a full overnight sleep study called a polysomnogram (PSG). This requires the patient to either go to a sleep lab in a medical facility or schedule an overnight sleep test at home, both of which can take months to schedule.

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A successful application of TDA to differentiating OSA positive and OSA negative patients directly through EEG signals, as we present in this paper, is a strong step towards easier and less invasive diagnosis of OSA in children, leading to better patient outcomes.

II. BACKGROUND

A. Obstructive Sleep Apnea

Pediatric Obstructive Sleep Apnea (OSA) is difficult to diagnose and leads to significant complications, including poor rest and heart disease[3]. In children especially, OSA can lead to behavioral changes and further reduction in quality of life. Identification of OSA in patients typically requires an overnight PSG study [11]. PSG studies include a number of sensors, such as EEG sensors, pulse oximetry, and respiratory rate sensors, among others. Acquisition of this information is difficult for the patient, as they must sleep in an unknown environment while being observed by medical personnel, as well as sleep with multiple sensors connected to their body. PSG studies are considered the gold standard for OSA diagnosis, and attempts to use other clinical methods such as questionnaires lead to poor diagnostic accuracy[11].

Snoring is relatively common in children, with about 10% incidence in children ages 1-9. Obstructive sleep apnea occurs in 2-3% of children with habitual snoring [3]. Further complication is caused by recent increases in pediatric obesity. Pediatric OSA is typically caused by adenotonsillary hypertrophy, and has been correlated with poor growth and failure to thrive [4]. Furthermore, daytime drowsiness (somnolence) is much less common in children with OSA as compared to adults with OSA, leading to further difficulties with the diagnosis of OSA.

B. Topological Data Analysis

TDA aims to identify quantitative information about the structure of data, and leverage this information for downstream data analysis tasks [5]. Data is assumed to exist in a metric space, allowing us to compute distances between different observations, which are typically viewed as points in an ambient space — i.e., forming a point cloud. From the data, one builds a shape on the input point cloud by varying the distance with which we consider two points as connected (further discussed in Section III-C).

TDA has previously been used to understand the underlying dynamics of the brain in work by [1], [8]. However, these papers focus on identification of attention-deficit/hyperactivity disorder (ADHD) in the case of Bourakna et. al. [1], and the classification of specific EEG

signal morphology during sleep in the case of Lella et. al. [8]. Work by Lella et. al. shows the strength of TDA as an analysis technique for single EEG signals, as this work outperforms previous state-of-the-art classification techniques with greater robustness to inherent noise than previous work; however, this work is focused on the specific morphology of EEG signals. While specific EEG morphology is useful, in the case of OSA we require a deeper understanding of brain dynamics in order to appropriately identify OSA-specific features. Additionally, we require a coherent analysis, involving all collected EEG channels, in order to fully exploit EEG data for TDA analysis. Work by Bourakna et. al. provides a strong foundation for leveraging TDA for analysis of ADHD data, while also using TDA to understand underlying brain network dynamics. This work performed a coherent analysis of multiple EEG channels in order to understand brain dynamics of ADHD patients rather than specific morphologies of individual EEG signals, and is therefore more robust to noise. We aim to apply these ideas to the identification of OSA.

OSA is not currently diagnosed by EEG signals in isolation, but rather in combination with other sensors collected during PSG studies. In this work, we leverage TDA applied to EEG signals without incorporating other sensors in order to identify statistically significant differences between OSA positive and OSA negative groups. This work provides a stepping stone towards improving scalability and ease of OSA diagnosis for both the clinician and the patient.

III. METHODS

A. Data

In this work, we leverage the Nationwide Children's Hospital (NCH) Sleep DataBank (SDB)[13], [7]. This dataset includes 3,984 PSG studies from 3,673 pediatric patients. This dataset contains a significant volume of data from a number of sensors, including EEG, electromyogram, electrooculogram, electrocardiogram, nasal and oral sensors to measure airflow, and pulse oximetry, among others. This large volume of data and sensors allows us to study pediatric OSA across a wide variety of pediatric patients. The dataset additionally contains annotations of sleep events, such as NREM1 sleep, NREM2 sleep, NREM3 sleep, and REM sleep, in 30 second intervals. We focus on EEG signals collected at 256 Hz using 7-channel EEG studies, which restricts us to 2,883 sleep studies. We identify patients as being in the "Apnea" or "No Apnea" based on the presence of an Apnea event string annotation in the patient's respective polysomnogram in the dataset. If an apnea-related string annotation is in the patient's polysomnogram, at any point during the sleep study, the whole study is placed into the "Apnea" group. If no apnea-related string annotation is found, the study is placed into the "No Apnea" group.

B. Signal Preprocessing

First, we perform basic signal processing on our EEG signals. We filter out 60Hz and 120Hz power noise using a 3rd order Butterworth bandstop filter.

We then leverage techniques from Bourakna et al. to create distance matrices from our EEG data[1]. We process data on a per-study basis. For a single PSG study we first extract all available EEG channels, leading to a set of time-series signals $X_i(t)$ for channel $i \in V$ and time $t \in \{1, \dots, T\}$. We break these signals into 30-second chunks to correlate with labels of Awake, NREM1, NREM2, NREM3, and REM sleep. We then create a smoothed periodogram from each channel for each 30-second chunk. A smoothed periodogram allows us to understand the frequency spectrum of our signal, while smoothing the frequency spectrum slightly using a small smoothing kernel to eliminate noise.

To calculate the smoothed periodogram, we use the following equations:

$$d(\omega_k) = \frac{1}{\sqrt{T}} \sum_{t=1}^T X_i(t) \exp(-i2\pi t\omega_k) \quad (1)$$

$$\hat{f}(\omega_k) = \sum_{\omega} k_h(\omega - \omega_k) d(\omega_k) d(\omega_k)^* \quad (2)$$

$k_h(\omega - \omega_k)$ indicates a smoothing kernel centered around ω_k and h is a bandwidth parameter. From these smoothed periodograms, we calculate the squared signal coherence between EEG channels i and j :

$$\mathcal{C}(X_i, X_j, \omega) = \frac{|f_{i,j}(\omega)|^2}{f_{i,i}(\omega) f_{j,j}(\omega)} \quad (3)$$

We then leverage a decreasing function to create a distance between coherence values for a given frequency ω :

$$\mathcal{D}(X_i, X_j, \omega) = 1 - \mathcal{C}(X_i, X_j, \omega) \quad (4)$$

The matrix $\mathcal{D}$ is indexed by both channel i , channel j , and frequency ω . EEG data is clinically analyzed using the following frequency bands: Delta band from 0.5 Hz to 4 Hz, Theta band from 4 Hz to 8 Hz, Alpha band from 8 Hz to 12 Hz, Beta band from 12 Hz to 30 Hz, and Gamma band from 30 Hz to 50 Hz.

We average across frequency all matrices which occur within a frequency band in order to obtain a single 7×7 matrix for that frequency band. Therefore, for a single 30-second EEG signal from 7 channels, we obtain 5 separate distance matrices, one for each frequency band. We treat distance matrices separately for each frequency band and sleep state. These distance matrices serve as the input for our TDA methods.

C. Topological Data Analysis Methods

In order to identify if the brain connectivity networks are fundamentally topologically different for OSA positive and OSA negative patients, we must understand how to differentiate topological spaces. We first begin with the definition of a simplicial complex.

Definition III.1 (Simplicial Complex [6]). A simplicial complex is a collection K of nonempty finite sets $\sigma \in K$, called simplices, such that $\sigma \in K$ and $\emptyset \neq \tau \subseteq \sigma$ always

imply that $\tau \in K$. If $\sigma \in K$ has $n + 1$ elements (i.e., $\sigma = \{x_0, \dots, x_n\}$) then we call it an n -simplex.

Intuitively, a 0-simplex $\{x_0\}$ represents a vertex, a 1-simplex $\{x_0, x_1\}$ an edge between x_0 and x_1 , a 2-simplex $\{x_0, x_1, x_2\}$ encodes a triangle spanned by these vertices, and so on in higher dimensions. Homology, which we define next, is a descriptor of the topology of simplicial complexes. It measures properties such as the number of connected components, the existence of holes, voids, and their higher dimensional analogs. Indeed, fix a field $\mathbb{F}$ (e.g., the rationals or the integers modulo a prime) and let $C_n(K)$ denote the $\mathbb{F}$ -vector space generated by the n -simplices of K . That is, each $\gamma \in C_n(K)$ can be written uniquely as $\gamma = \sum_{j=0}^J a_j \sigma_j$ for scalars $a_j \in \mathbb{F}$ and n -simplices $\sigma_j \in K$. Define the boundary of an n -simplex $\sigma = \{x_0, \dots, x_n\} \in K$ as $\partial_n(\sigma) = \sum_{i=0}^n (-1)^i (\sigma \setminus \{x_i\}) \in C_{n-1}(K)$. Since the n -simplices of K form a basis for $C_n(K)$, then we obtain linear transformations $\partial_n : C_n(K) \rightarrow C_{n-1}(K)$ which, as one can check, satisfy $\partial_n \circ \partial_{n+1} = 0$ for every $n \in \mathbb{N}$.

Definition III.2 (Simplicial Homology Group [5]). The n th homology group of a simplicial complex K is the quotient vector space

$$H_n(K) = \text{Ker}(\partial_n) / \text{Im}(\partial_{n+1})$$

The homology of K can be interpreted as follows: the dimension of $H_0(K)$ is exactly the number of connected components of K , $\dim(H_1(K))$ counts the essentially distinct closed loops in K bounding an empty region, $\dim(H_2(K))$ counts cavities, and similarly for $\dim(H_n(K))$, $n \geq 1$.

In this work, we attempt to understand the topological features of each homology group (e.g., connected components, holes, cavities, etc.) for the brain connectivity network of OSA positive and OSA negative patients. Further treatment of topological concepts can be found in [6].

In order to calculate homological features on sets of discrete data points, we apply persistent homology to the distance matrices described in Section III-B. Indeed, once we have obtained a distance matrix for a 30-second signal, we build a simplicial complex, in particular a *Rips Complex*, and furthermore the *Rips filtration* using the Python package `Ripser.py`[12].

Let $\mathcal{S}$ be our point cloud, with pairwise distances described by our previous distance matrix $\mathcal{D}$. The Rips complex $\mathcal{R}$ of $\mathcal{S}$ and distance r is given by the simplicial complex consisting of all subsets of $\mathcal{S}$ with diameter at most r , i.e.

$$\mathcal{R}(\mathcal{S}, r) = \{\sigma \subset \mathcal{S} | \text{diam}(\sigma) \leq r\}$$

Note that $\mathcal{R}$ is inherently tied to a given r value. We construct a *filtration*, called a Rips filtration, which is the set of Rips complexes created by varying the free parameter r . We start with a small r ($r = 0$), and increase r continuously to obtain a set of subcomplexes of our simplicial complex[6]:

$$\emptyset = K_0 \subseteq K_1 \subseteq K_2 \subseteq \dots \subseteq K_m$$

We leverage `Ripser.py` to compute our Rips filtration. As we vary r from K_{i-1} to K_i , our topological features change, and we gain or lose topological features[9]. We quantify the r at which a particular topological feature in a particular homology group appears and the r at which the same topological feature disappears as a (birth, death) pair, which exists in $\mathbb{R}^2$. We can plot these points on a graph to create a *persistence diagram*, as exemplified in Figure 1.

While the persistence diagram allows us to visualize the topological features of a particular sample, it does not extend to separable Banach spaces, and therefore does not lend itself to analysis through the lens of random variables[2]. However, we can transform our persistence diagram into a *persistence landscape*, which is a function and does exist in a separable Banach space. We construct persistence landscapes by drawing an isosceles triangle centered on the points of our persistence diagrams. When intersections occur the highest function is defined as the persistence landscape.

D. Permutation Testing

While we now have usable TDA features in the form of persistence landscapes, we need to identify a test which can indicate whether our TDA features from OSA positive pediatric patients is different than our TDA features from OSA negative patients. To this end, we leverage a permutation test, as described in [10]. In this methodology, our null hypothesis is that there is no impact of OSA on the brain connectivity network. To perform the test, we implement the following algorithm:

- 1) Calculate the average OSA positive persistence landscapes $\{\lambda_1^{(1)}, \dots, \lambda_{n_1}^{(1)}\}$ and the average OSA negative persistence landscapes $\{\lambda_1^{(2)}, \dots, \lambda_{n_2}^{(2)}\}$.
- 2) Calculate the difference between the average OSA positive persistence landscape and the average OSA negative persistence landscape, and find the most significant value of this difference landscape $\hat{F}_T$.
- 3) Permute the labels of our two groups such that we have n_1 randomly chosen positively labeled persistence landscapes and the remaining n_2 landscapes are negatively labeled.
- 4) Calculate the difference between the permuted positive group and the permuted negative group, and find the most significant value of this difference landscapes $\hat{F}_p$.
- 5) If $\hat{F}_p \geq \hat{F}_T$, count a significant permutation.
- 6) Repeat steps 3 and 4 B times. For this analysis we repeat these steps $B = 1000$ times.
- 7) Compute the p -value as the number of significant permutations S divided by the total number of permutations B .

We specifically perform this permutation test on the 0th homology group (H_0) persistence landscapes generated as described in Section III-C.

IV. RESULTS

We show example persistence diagrams and persistence landscapes for an OSA positive patient in Figure 1 and Figure 2.

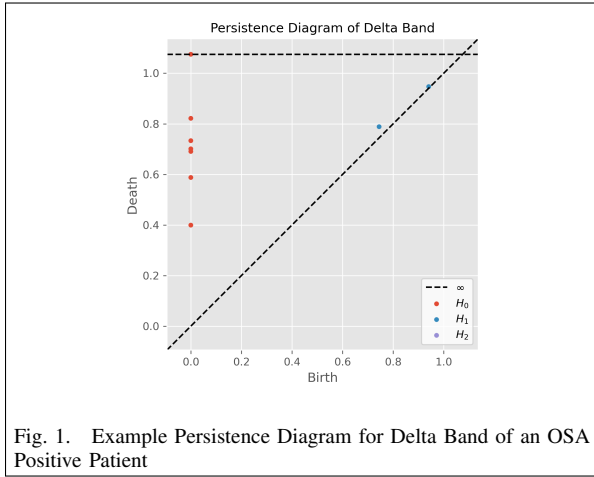


Fig. 1. Example Persistence Diagram for Delta Band of an OSA Positive Patient

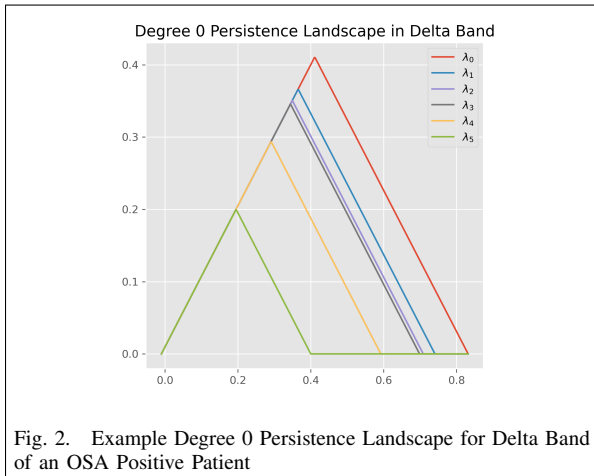


Fig. 2. Example Degree 0 Persistence Landscape for Delta Band of an OSA Positive Patient

We present p -values for our TDA permutation tests discussed in Section III-D in Table I. We consider p -values with $p < 0.05$ to be significant. We separate our distance matrices into signals which occur during NREM1 sleep, NREM2 sleep, NREM3 sleep, and REM sleep.

TABLE I
 p -VALUES FOR SLEEP STATES USING TDA

EEG Band	NREM1	NREM2	NREM3	REM
Delta Band	0.000	0.001	0.000	0.040
Theta Band	0.000	0.000	0.000	0.000
Alpha Band	0.000	0.000	0.000	0.000
Beta Band	0.000	0.000	0.000	0.000
Gamma Band	0.000	0.000	0.000	0.000

V. DISCUSSION

Our results indicate that TDA can identify statistically significant differences between OSA positive and OSA negative cohorts. Statistical significance is achieved in all bands for

all phases of sleep. These results are a strong indication that EEG signals alone can be used to help diagnose OSA in pediatric patients, allowing for less invasive diagnosis of OSA.

VI. CONCLUSIONS

In this work, we show that the brain dependence networks of OSA positive pediatric patients show a statistically significant difference than the brain dependence networks of OSA negative pediatric patients when analyzed using topological data analysis. Our work lays the foundation for using TDA for cohort-level OSA classification, and takes the first step towards being able to make an OSA diagnosis with a single EEG. Future work is needed to achieve clinical utility and to determine how the brain dynamics TDA identifies relate to respiration measures more typically used for OSA diagnosis.

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