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RESEARCH ARTICLE

Distinct spike-and-wave EEG profiles reveal susceptibility to fleeting/almost loss of consciousness (so-called blips) in generalized epilepsy

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Abstract

Objective: To characterize electroencephalographic (EEG) profiles of short spike-and-wave bursts (SWBs) in patients with idiopathic generalized epilepsy reporting sensations of fleeting/almost loss of consciousness, described as “a blip on the screen”—a phenomenon first termed “blips” by J.W. Lance.

Methods: Among 176 consecutive patients, 19 were included based on a diagnosis of idiopathic generalized epilepsy and the presence of SWBs on EEG recordings. Patients were classified as “blippers” (yes) or “non-blippers” (no) based on their response to whether they had ever experienced “blip”-like sensations. A total of 624 SWBs extracted from EEG traces were compared between groups. The analysis focused on the spike (10–125 Hz) and wave (2.5–4 Hz) frequency band components across predefined spatial regions of interests.

Results: SWBs were significantly longer in blippers than in non-blippers (median: 1.2 s, interquartile range [IQR]: (Q1–Q3): 753–1796 vs 0.9 s, 599–1541; $p < .001$, $r = .14$). EEG analyses revealed two distinct spatial profiles, with higher fractional change in wave amplitude over anterior frontal (39.73, 12.56–81.27 vs 17.34, 5.18–56.95; $p < .01$, $r = .13$), frontal (46.94, 20.55–100.28 vs 31.08, 9.51–88.81; $p < .05$, $r = .10$), and occipital (25.25, 11.51–50.97 vs 14.99, 4.94–43.80; $p < .01$, $r = .13$) regions, but not parietal ($p > .05$). Similarly, spikes showed increased amplitude in anterior frontal (5.32, 2.50–15.27 vs 4.20, 2.58–7.60; $p < .05$, $r = .09$) and central (3.95, 2.81–6.20 vs 3.07, 1.82–5.08; $p < .01$, $r = .14$) regions, with lower occipital power in blippers (1.93, 1.36–3.19 vs 2.53, 1.52–4.33; $p < .05$, $r = -.10$). Overall, spike and wave amplitudes were higher in blippers, particularly at SWB onset. All p -values were false discover rate (FDR)-corrected and analyses were conducted at the SWB level.

Significance: Distinct EEG profiles of SWBs may be associated with self-reported blip experiences.

Hal Blumenfeld and Laurent Vercueil shared senior authorships.

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KEYWORDS

blip syndrome, spike-and-wave bursts, epileptiform activity, absence seizures, consciousness

1 | INTRODUCTION

The blip syndrome, first described by James W. Lance,¹ was characterized in a case series involving 12 non-epileptic patients reporting momentary sensations of impending loss of consciousness, lasting less than a second, with occurrence frequencies ranging from one episode per month to 15 per day. The term “blip” was coined to illustrate an instantaneous subjective sensation, “like a blip on a screen,” which vanishes as soon as it appears. It is intriguing that Lance believed that these blips could be “quasi-epileptic phenomenon,” suggesting that epileptic discharges could mimic these experiences.

Recently, we reported on a 15-year-old patient with juvenile myoclonic epilepsy who described blip-like sensations during video electroencephalography (EEG) recording. Remarkably, each self-reported sensation was time-locked to brief (1–3 s) spike-and-wave bursts (SWBs).² Building on this observation we proposed defining an epileptic “blip” as characterized by four key features: (1) a sensation of fleeting/almost loss of consciousness; (2) an extremely brief duration, perceived as lasting less than 1 s; (3) a phenomenological localization within the interior of the head; and (4) strictly time-locked to a generalized SWB.

It is important to note that these phenomena are described as purely internal, devoid of any external sensory component. Patients do not report visual, auditory, or somatosensory hallucinations. This unique feature raises important questions about how SWBs influence the stream of consciousness, expanding the field of transitory cognitive impairments due to interictal discharges.³ It is striking that patients with epileptic blips seem capable of detecting their own EEG discharges, pointing to a direct introspective link between epileptic activity and transient disruptions in conscious awareness.

Epileptic blips represent a novel phenomenon that is likely undetected and unrecognized. In order to further investigate their presence among patients with SWBs, we conducted a survey among a cohort of consecutive patients in our epileptology department to identify individuals who had experienced blip-like sensations. Our goal was to compare SWBs in patients reporting a history of blips (so-called “blippers”) to those without (“non-blippers”) and to determine whether blippers exhibited distinctive SWB characteristics, similar to those seen in spared vs impaired consciousness during absences.^{4,5} Specifically, we aimed to identify different

Key points

- Epileptic blips are described as brief events in which patients behave as if they perceive their own electroencephalographic (EEG) spike-and-wave bursts (SWBs).
- At the burst level, distinct spike-and-wave EEG profiles were observed in patients with a history of blips (blippers), suggesting candidate physiological correlates.
- A specific combination of spike-and-wave physiological changes may contribute to the perception of fleeting or almost impairments of consciousness.
- SWBs from blippers show increased wave amplitude in the frontal/occipital areas and higher spike amplitude over anterior frontal and central EEG sites.

electroencephalographic SWB profiles to better understand the neurophysiological underpinnings of the occurrence of blips.

2 | MATERIALS AND METHODS

2.1 | Participants

The initial cohort consisted of 176 consecutive patients screened at the epilepsy-specialized neurology department of Grenoble-Alpes University Hospital (CHUGA) over a 4-month period (February to May 2005). These data had not been analyzed previously; this study marks their first scientific use. Patients were included based on the following criteria: (1) a diagnosis of idiopathic generalized epilepsy (IGE), a condition associated with a higher propensity for generalized spike-and-wave bursts^{6,7}; and (2) the presence of epileptiform generalized SWBs identified in EEG recordings.

At the time the cohort was established in 2005, a total of 19 participants meeting the inclusion criteria were asked the following questions: “Do you sometimes feel like there’s a brief moment of disconnection? As if a flash is passing through your head? Like a blip on a screen?”. Those who responded affirmatively were assigned to the “blippers” group, whereas those who responded negatively

were classified as “non-blippers”. This retrospective study included only EEG recordings predating blip-related clinical investigation, as group classification relied on patients’ recollection of the phenomenon at the time of inclusion. No standardized assessment was conducted between 2006 and 2022.

In 2023, upon resuming scientific investigation of the blip phenomenon, two additional patients were included, both of whom spontaneously reported blip-like experiences during clinical interviews and met the original inclusion criteria. As this study is not epidemiological, their inclusion aimed to enhance statistical power. It is important to note that the diagnostic criteria for IGE have remained largely consistent over the years,^{8,9} ensuring the continued relevance and reliability of the dataset for present analyses. In total, 21 participants were included in the study (8 blippers, 13 non-blippers). However, due to partial EEG file corruption, only 19 participants from the original cohort were retained for EEG-based analyses (6 blippers, 13 non-blippers).

EEG data were recorded during wakefulness from 19 scalp electrodes arranged according to the international 10–20 system, using the Deltamed Coherence EEG system at a sampling rate of 256 Hz. The cumulative median recording time was 70 min per participant, across an average of two EEG sessions. [Table 1](#) summarizes the participants’ demographic and clinical characteristics. A total of 745 SWBs were initially identified, but due to file corruption and additional selection criteria (described later), 624 SWBs were ultimately retained for EEG analysis.

2.2 | Data analysis

The methodology for identifying and analyzing SWBs was based on established protocols (see Guo et al.⁴; Springer et al.⁵). EEG data were acquired at CHUGA, France, and analyzed at Yale, USA, using MATLAB 2024b (MathWorks) and the EEGLAB toolbox (v2024; Delorme & Makeig¹⁰).

2.2.1 | Spike-and-wave burst identification

Generalized SWBs were visually identified and marked using a customized version of the EEGLAB software, developed in previous work.¹¹ An epilepsy specialist (L.V.), blinded to group assignments, manually annotated the onset and offset of each SWB (see [Supporting Information](#) for details). Only interictal bursts were analyzed, based on the absence of any reported clinical manifestations or ictal semiology during the recordings.

TABLE 1 Group demographics, clinical profiles, and electrophysiological characteristics.

	<i>n</i>	Female	Age, years (med [Q1–Q3])	ASO, years (med [Q1–Q3])	Epilepsy duration (med [Q1–Q3])	AS only	GTCS only	AS + GTCS	MS + GTCS	AS + MS + GTCS	SWB
Blippers	8	6	29 [24–38.5]	12.5 [8–15]	13.6 [6.7–31.8]	1	1	4	1	1	132
Non-blippers	13	9	18 [16–26]	14 [11–19]	7 [4–9]	–	6	5	2	–	613
Total	21	15	–	–	–	–	–	–	–	–	745

Note: This table presents the median age at EEG sessions, age at seizure onset (ASO), and the number of patients with a history of myoclonic seizures (MS), absence seizures (AS), and/or generalized tonic-clonic seizures (GTCS) for each group. The table also reports the total number of generalized spike-and-wave bursts (SWBs) identified in EEG recordings. Abbreviations: AS, absence seizures; ASO, age at seizure onset; GTCS, generalized tonic-clonic seizures; med, median; MS, myoclonic seizures; Q1, first quartile (25th percentile); Q3, third quartile (75th percentile); SWB, spike-and-wave burst.

2.2.2 | EEG preprocessing

Preprocessing steps were adapted from previous work⁵ (see [Supporting Information](#) for further details). EEG data were high-pass filtered with a finite impulse response (FIR) with a 1 Hz corner frequency to remove low-frequency drift, and a 50 Hz notch filter was applied to attenuate power line noise. For artifact rejection, any 1 s epoch of the signal was rejected if its amplitude exceeded 150% of the power mean in each SWB, averaging voltage across channels. All electrodes were re-referenced to a common average reference to enhance signal comparability.

SWBs and surrounding time periods were segmented into distinct windows: pre-SWB (20 s before onset), SWB (epileptiform burst period), and post-SWB (20 s after offset). The 20-s pre-SWB window was selected in line with previous work.^{4,5,12} Although early pre-burst changes have been described,¹³ major spectral shifts typically emerge at SWB onset. Overlapping SWBs were managed by equally splitting overlapping peri-SWB windows or by rejecting events occurring within 4 s of another SWB. The remaining signal served as the global baseline. Finally, the global baseline mean voltage per channel was subtracted from the SWB and peri-SWB windows. This was done to ensure full control over potential residual voltage offsets.

2.2.3 | EEG analysis

Power spectral analysis was employed to explore differences across spatial regions of interest (ROIs), following methodologies previously applied in the analysis of spike-and-wave discharges during absence seizures (see Guo et al.⁴). Typical spike-and-wave discharges consist of low-frequency 3–4 Hz waves and high-frequency (>10 Hz) spikes; thus, our analysis focused on the corresponding 2.5–4 and 10–125 Hz frequency ranges. For each range, EEG power was calculated for each SWB and normalized by dividing it by the mean power during the pre-SWB period of the same channel, yielding fractional power. The results were visualized using head maps, which were quantified across predefined scalp regions of interest. In addition, time courses of fractional EEG amplitude were plotted for blippers vs non-blippers, calculated across all electrodes together.

Given the subtler nature of blips compared to absence seizures, potentially associated with power differences across the entire scalp, we adopted a more granular division of the scalp into five ROIs, rather than the broader three regions (frontal, central, and posterior) used in prior studies. The selected ROIs included frontal anterior (Fp1–Fp2), frontal (F3, Fz, F4), middle (C3, Cz, C4), parietal (P3, Pz, P4), and occipital (O1, O2) electrodes. Regional scalp

data were obtained by averaging the fractional power within each ROI during SWB periods.⁴

2.2.4 | Statistical analysis

Statistical analyses were performed in MATLAB using non-parametric Wilcoxon rank-sum tests, consistent with prior work,⁵ to compare demographic, clinical, and electrophysiological variables between blippers and non-blippers (significance threshold: .05). False discovery rate (FDR) correction was applied to account for multiple comparisons.¹⁴ Fisher's exact test assessed associations between blippers and specific IGE subtypes (e.g., absence).

Statistical analyses were focused on SWB periods, with the 20 s preceding each SWB event used as a reference for signal change measurements. Wilcoxon rank-sum tests were employed to compare median fractional power amplitude in scalp ROIs during SWBs between blippers and non-blippers. FDR correction was applied to adjust for multiple comparisons. Analyses were performed separately for frequency bands associated with spike and wave components. Similar analyses in the time–frequency domain were conducted to capture temporal variations in fractional power between SWBs from blippers and non-blippers. Periods spanning –20 s to +20 s around each discharge were compared (Springer et al.⁵), and fractional power within each window was calculated with 50% overlap. To ensure accurate frequency estimation, the window size was adjusted to include at least two full cycles within each frequency band. Consequently, different window sizes were used: 256 sampling points (1 s) for the 2.5–4 Hz band and 51 (0.2 s) for the 10–125 Hz band.

Statistical analyses were primarily conducted at the individual SWB level, in line with prior discharge-level studies,^{4,5,12,15} with the specific aim of identifying potential biomarkers at the burst level. However, to account for the nested structure of the data, complementary subject-level analyses were also performed, as detailed in the [Supporting Information S1](#).

3 | RESULTS

Of the 19 participants retained for EEG analyses, 6 reported experiencing “blips.” No blips were annotated during EEG recordings. However, given the fleeting and underrecognized nature of the phenomenon, it is possible that some events were overlooked or under-evaluated in routine clinical settings. As previously detailed, two additional patients, included in 2023, met the inclusion criteria and were added to the clinical and demographic analyses only (bringing the total to 8 blippers and 13

non-blippers for these analyses). Although their EEG studies were annotated, file corruption prevented their inclusion in quantitative analyses. EEG-based results thus rely on 6 blippers and 13 non-blippers.

3.1 | Demographics and clinical characteristics

All statistical analyses were performed using non-parametric rank-sum tests. Results are described using medians and interquartile ranges (IQRs; Q1–Q3), with corrections for multiple comparisons applied via the FDR method for EEG-related analyses. Detailed clinical and demographic data, including antiseizure medication (ASM) regimens at inclusion, are provided in the [Table S1](#).

Blippers were significantly older (median: 29 years, IQR (Q1–Q3): 24–38.5) than non-blippers (18 years, 16–26), based on participants' mean age across their EEG sessions ($p < .05$). No significant group differences were found in age at seizure onset (blippers: 12.5 years, 8–15; non-blippers: 14 years, 11–19). However, epilepsy duration—calculated as the difference between participants' mean age across their EEG sessions and age at seizure onset—was significantly older in blippers (median: 13.6 years, IQR: 6.75–31.8) than in non-blippers (median: 7 years, 4–9; $p < .05$). In addition, no significant differences were observed in the frequency of SWBs per hour, calculated as the ratio of SWBs to EEG session duration (non-blippers: 21, 7.59–31.9; blippers: 8.5, 2.09–25.5; $p > .05$).

To assess whether blippers were more prone to certain seizure types within the idiopathic generalized epilepsy group (i.e., absence, myoclonic and/or generalized tonic-clonic seizures), we compared the proportion of patients with a history of each seizure type between blippers and non-blippers. For each seizure type, a 2×2 contingency table (presence vs absence × group) was constructed, and Fisher's exact test was applied due to small sample sizes. No significant group differences were observed in the prevalence of absence seizures (blippers: 6/8; non-blippers: 5/13), myoclonic seizures (blippers: 2/8; non-blippers: 2/13), or generalized tonic-clonic seizures (blippers: 7/8; non-blippers: 5/13), all p 's $> .05$.

3.2 | EEG profile of spike-and-wave burst

This section describes the analysis of EEG data focusing on SWBs in blippers and non-blippers. To ensure accurate results, SWBs occurring within 4 s of each other were excluded to avoid overlap and to provide sufficiently long pre-ictal periods that (1) do not interfere with post-ictal periods and (2) enable reliable channel mean estimation.

Two corrupted data files were also excluded, resulting in 109 SWBs in the blipper group and 515 SWBs in the non-blipper group, for a total of 624 SWBs.

SWBs in blippers were significantly longer in duration (median: 1288 ms, IQR (Q1–Q3): 753–1796) compared to non-blippers (928 ms, 599–1541; $p < .001$, $r = .14$). EEG recordings revealed broadly distributed SWBs with frontal amplitude predominance ([Figure 1A,B,D,E](#)). However, blippers showed greater EEG power across widespread regions, particularly in the anterior frontal and posterior areas, compared to non-blippers ([Figure 1C,F](#)).

In the wave frequency range (2.5–4 Hz), blippers exhibited significantly higher power in anterior frontal, frontal, and occipital regions. In the spike frequency range (10–125 Hz), blippers demonstrated greater power in anterior frontal and central regions. Of interest, non-blippers displayed higher occipital power in the spike frequency range but lower occipital power in the wave frequency range compared to blippers.

In greater detail, median differences in fractional EEG power for waves were as follows: anterior frontal (blippers: 39.73, 12.56–81.27; non-blippers: 17.34, 5.18–56.95; $p < .01$, $r = .13$); frontal (blippers: 46.94, 20.55–100.28; non-blippers: 31.08, 9.51–88.81; $p < .05$, $r = .10$); and occipital (blippers: 25.25, 11.51–50.97; non-blippers: 14.99, 4.94–43.80; $p < .01$, $r = .13$). Central (blippers: 12.15, 8.14–23.68; non-blippers: 13.11, 4.95–28.12) and parietal (blippers: 16.73, 7.62–32.19; non-blippers: 16.42, 5.46–33.32) regions were not significant ($p > .05$). For spikes, differences were observed in anterior frontal regions (blippers: 5.32, 2.50–15.27; non-blippers: 4.20, 2.58–7.60; $p < .05$, $r = .09$), and central regions (blippers: 3.95, 2.81–6.20; non-blippers: 3.07, 1.82–5.08; $p < .01$, $r = .14$). Finally, in the occipital region, non-blippers showed higher power compared to blippers (blippers: 1.93, 1.36–3.19; non-blippers: 2.53, 1.52–4.33; $p < .05$, $r = -.10$). All p -values were corrected using the FDR. Frontal (blippers: 4.46, 2.95–8.39; non-blippers: 4.99, 2.91–8.89) and parietal (blippers: 3.61, 2.34–5.05; non-blippers: 3.16, 1.93–5.00) areas were not significant ($p > .05$).

Complementary per-subject summary metrics are provided in [Table S2](#). Group comparisons using Wilcoxon rank-sum tests at the subject level did not yield statistically significant differences ([Table S3](#)). However, a group-level generalized linear mixed-effects model analysis revealed a similar trend-level difference (uncorrected) between blippers and non-blippers in the spike frequency band within the occipital ROI ([Table S4](#)).

Time course analysis revealed overall greater amplitudes from the onset of SWBs in blippers compared to non-blippers ([Figure 2A,B](#)). In the 2.5–4 Hz band (associated with wave components), group differences emerged prior to burst onset. In the 10–125 Hz range (associated

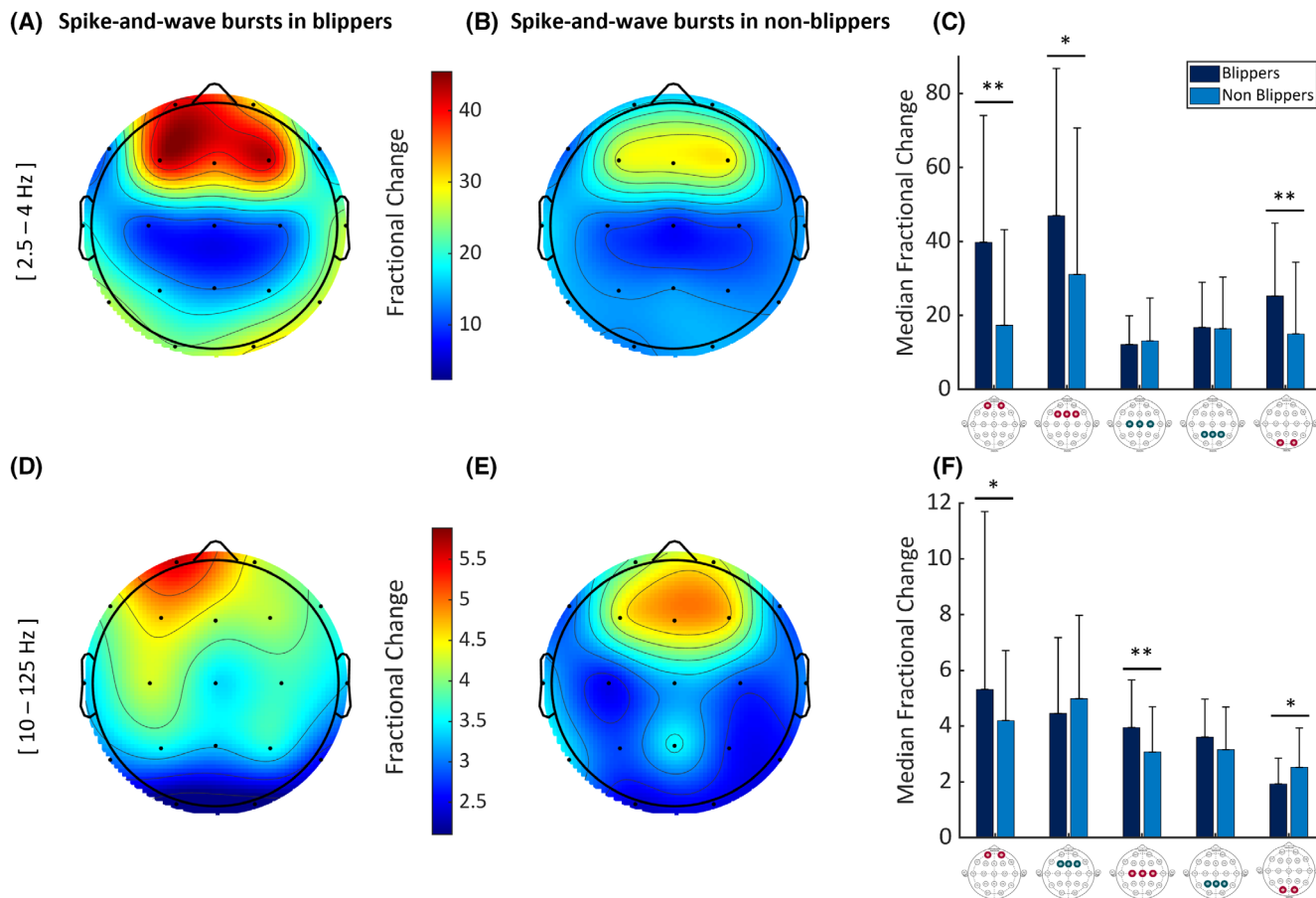


FIGURE 1 Electroencephalographic (EEG) power of spike-and-wave bursts (SWBs) at the burst level in blippers compared to non-blippers. (A, B) Topographic maps of EEG power across 19 channels in the 2.5–4 Hz frequency range, representing the wave components of spike–wave bursts for blippers (A) and non-blippers (B). (C) Median fractional EEG power is displayed for each scalp region of interest (ROI) for blippers and non-blippers (anterior frontal: Fp1, Fp2; frontal: F3, Fz, F4; central: C3, Cz, C4; parietal: P3, Pz, P4; and occipital: O1, O2) in the 2.5–4 Hz frequency range, comparing blippers and non-blippers. (D, E) Topographic maps of EEG power in the 10–125 Hz frequency range, corresponding to the spike components of spike–wave bursts, are shown for blippers (D) and non-blippers (E). (F) Median fractional EEG power in the 10–125 Hz frequency range for spike–wave bursts in blippers vs non-blippers for each scalp ROI. Color scale bars represent EEG power during SWBs normalized to pre-SWB power prior to seizures (fractional change). The top color bar applies to A and B, whereas the bottom color bar applies to D and E. In C–F, dark blue represents blippers, and sky blue represents non-blippers. Interquartile range (IQR): 75th percentile (third quartile, Q3) to 25th percentile (first quartile, Q1) is centered around the median (second quartile, Q2). For clarity, only the upper half (IQR/2) is displayed as error bars above the median. ROIs on the *x*-axis are highlighted in red when statistical significance is reached after false discovery rate (FDR) correction (* $p < .05$; ** $p < .01$). $n = 624$ SWBs, including 109 in 6 blipper patients and 515 in 13 non-blipper patients.

with spike components), amplitude differences became significant shortly after seizure onset. This suggests that SWBs in blippers are associated with more pronounced physiological changes on EEG. Of interest, blippers also exhibited a period of higher pre-ictal power in the 10–125 Hz range and periods of higher post-ictal power in both the 10–125 and 2.5–4 Hz frequency ranges compared to non-blippers.

Individual subject time courses of fractional EEG power are shown in Figure S1B–D. Subject-level rank-sum comparisons are presented in A and B, yielding longer periods of significance. However, given the limited statistical power at the subject level—particularly for some blippers

contributing only 1 SWB—these results should be interpreted with caution.

4 | DISCUSSION

The objective of this study was to compare the characteristics of EEG spike-and-wave bursts (or SWBs) between patients with IGE who had previously experienced blips and those who had not. The goal was to identify EEG features that distinguish SWBs recorded in individuals susceptible to epileptic blips. Apart from a significantly higher age in the blip group—which

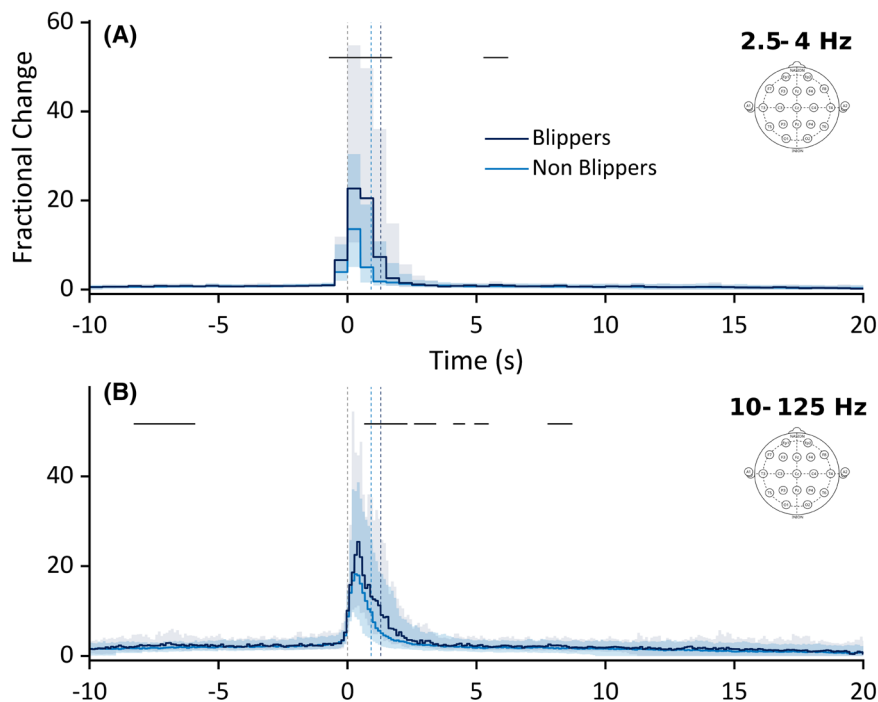


FIGURE 2 Time course of electroencephalographic (EEG) fractional power during spike-and-wave bursts (SWBs) in blippers versus non-blippers across all electrodes at burst level. (A, B) Time course of EEG amplitude fractional change (signal divided to baseline) for low (2.5–4 Hz, A) and high (10–125 Hz, B) frequency ranges, representing the waves and spikes characteristic of SWBs. Bold segments indicate statistically significant differences ($p < .05$) following false discovery rate (FDR) correction for multiple comparisons. Time courses are aligned to SWB onset (time = 0 s) and display the median (second quartile, Q2; bold color lines) and interquartile range (IQR; shaded area between 25th (first quartile, Q1) and 75th (third quartile, Q3) percentiles) across all electrodes. Dashed vertical lines mark burst onset (gray) and median offset (dark blue for SWBs in blippers; sky blue for non-blippers). Fractional power was computed using overlapping windows with a 50% overlap. The initial window size was 51 sampling points (0.2 s) for the 10–125 Hz band and 256 (1 s) for the 2–4 Hz band, which accounts for the visual differences observed in the curves. $n = 624$ SWBs, including 109 in 6 blipper patients and 515 in 13 non-blipper patients. Longer window size in the 2.5–4 Hz band reduces temporal precision compared to the 10–125 Hz band.

was also associated with longer epilepsy duration—no notable differences were found in demographic or epilepsy-related clinical variables. Notably, the duration of SWBs was slightly longer in blippers compared to non-blippers, although their duration remained remarkably brief (~1.2 s). Across all SWBs, the spike and slow wave components consistently exhibited maximal amplitude in the frontal cortex (Figure 1), a pattern commonly reported in absence seizures^{4,16,17}—suggesting potentially shared mechanisms between absences and blips.

Indeed, typical absences are non-motor seizures characterized by abrupt and transient episodes of staring and unresponsiveness, typically lasting ~10 s, and associated with generalized spike-and-wave discharges at 3–5 Hz.¹⁸ The spike and slow wave components of these discharges exhibit maximal amplitude in the bi-frontal convexity, with optimal identification in the fronto-polar regions.^{16,17,19} Absences are widely recognized as the most distinct and pure form of consciousness impairment in epilepsy²⁰ and show strong electrophysiological (SWBs) and semiological (consciousness impairment) parallels with blips.

A large EEG/functional magnetic resonance imaging (fMRI) cohort study analyzing 810 absence seizures demonstrated that varying degrees of consciousness impairment are driven by widespread physiological changes across brain networks, including the default mode, task-positive, and frontoparietal networks.⁴ This continuum suggests that spike-and-wave discharge characteristics—such as duration and spike-and-wave power across frontal, central, and parietal regions—are closely linked to the severity of consciousness impairment, and may serve as a framework for characterizing blips.

Nevertheless, shorter paroxysmal epileptiform EEG abnormalities are conventionally classified as interictal bursts (<3 s) and considered clinically silent.²¹ However, even brief epileptiform discharges have been shown to disturb behavior²² and cognition,^{3,23} highlighting that duration alone does not strictly correlate with the extent of impairment. This is further supported by the existence of very short absences (<4 s),²⁴ as well as by reports in photosensitive patients, where one subjective sensation of impaired consciousness was already noted during a 2 s

photic stimulation-induced discharge.²⁵ Taken together, these findings suggest that the duration of epileptiform activity is not the sole determinant of consciousness alteration—a perspective that directly informs how blips should be interpreted, as they may lie on a continuum with absence seizures.

The variability of absence seizures, combined with the morphological similarities in spike-and-wave bursts between absences and blips, as well as their shared association with consciousness impairment—complete interruption in absence seizures vs brief, self-detected disruptions in blips—raises the possibility that blips may reflect a minimal expression of absence seizures or, at the very least, a “micro-absence” or an “almost-absence.” It is notable that many blippers had a comorbid diagnosis of absence seizures, which may partly explain the electrophysiological patterns observed. However, interpreting such short bursts as absence seizures would require a reconsideration of the current International League Against Epilepsy (ILAE) definition of absences, which typically involve longer, clinically observable ictal events. Notably, however, not all patients experiencing blips had absence seizures.

The precise electrophysiological mechanisms underlying the specific phenomenological experience associated with blips remain unclear. It thus remains to be determined whether the blip-like phenomenon arises from an inefficient or hyper-efficient spike component, a wave component, or an interplay of both.

Our results suggest that both the spike and wave components of SWBs exhibit higher amplitudes in blippers compared to non-blippers, with distinct regional differences (see Figure 1). The wave component shows greater amplitude in the frontal cortex, consistent with the stronger involvement of the cortico-thalamic network in absence seizures,^{26,27} which is proposed to specifically impair cognitive functions.²⁰ Of note, the maximum amplitude of the wave component for both blippers and non-blippers appears to be in the same frontal region, but of larger magnitude for the blippers (see Figure 1A–C). The spike component also has maximal amplitude in the anterior regions for both blippers and non-blippers; however, for blippers this component appears to be more anteriorly displaced into prefrontal electrode contacts, whereas for non-blippers the maximum appears in frontal contacts (Figure 1D–F). Another regional difference between the wave and spike component is seen in the occipital area, where blippers have significantly larger magnitude for waves but smaller magnitude for spikes. Such regional differences were not seen between behaviorally spared and impaired absence seizures, suggesting a possible difference in physiology between absence seizures and blips.^{4,5,28} To better understand the altered consciousness

in both absence seizures and blips, it will be necessary in future work to investigate the potential involvement of anterior and posterior brain networks in greater detail. Global theories of consciousness emphasize the prefrontal cortex's crucial role within a frontoparietal network supporting conscious access on a timescale of a few hundred milliseconds.^{29,30} However, posterior brain networks are also postulated to play a key role in conscious processes,^{31–33} including self-representation³⁴ and metacognitive processing.³⁵

In the blippers group, all patients had a history of absence seizures and/or tonic-clonic seizures (see Table 1). The classification of blips remains complex, as their characteristics do not fit neatly within the ictal-interictal dichotomy. Although the electrophysiological amplitude of SWBs in blippers suggests ictal epileptic discharges, their brief duration aligns more closely with interictal activity (see Figure 2). However, as discussed, duration alone does not strictly determine the extent of impairment.

This intermediate duration—~300 ms longer in SWBs from blippers (~1.2s) compared to those from non-blippers (~0.9s)—raises important questions about its clinical relevance. This difference may be a first step in defining a permissive temporal window, sufficient to transiently disrupt consciousness, yet still short enough to avoid the complete memory suppression typically observed in absence seizures. In this context, such SWBs could reflect a borderline state in which consciousness is briefly impaired but the interruption remains accessible to introspection and, consequently, to encoding and recall. However, the duration does not strictly match the phenomenological experience reported by patients—as “a fraction of a second”—suggesting that only part of the burst may suffice to generate a blip experience, or that subjective and objective temporalities may not align.

It is interesting that auras—often described as sensorial experiences preceding seizures³⁶—were classically thought to be associated with focal rather than generalized epilepsy; however, more recent work has shown that auras occur commonly in generalized epilepsy as well.³⁷ The subjective nature of blips suggests that they may represent a distinct and underrecognized type of aura in patients with generalized epilepsy. However, unlike classical auras, which primarily involve sensory or perceptual systems, blips appear to reflect a transient disruption in the stream of consciousness itself. Described as purely internal and fleeting,^{1,2} these phenomena could be conceptualized as an “aura of consciousness,” providing a window into how brief epileptic bursts interfere with neural networks critical for conscious experience and how such disruptions are subjectively detected by the patient.

4.1 | Limitations and perspectives

In our study, the comparison of SWBs between blippers and non-blippers did not include information about the occurrence of blips or not in the individual SWB episodes. Therefore, we were only able to investigate potential overall physiological differences between patients with vs without blips rather than studying the pathophysiology of individual blips themselves. Ideally, this study would have included patients experiencing epileptic blips during EEG recordings, allowing for a robust characterization of the relationship between SWBs and simultaneous subjective phenomena. However, such cases appear to be rare.² Instead, we assumed that the recollection of previous blip sensations could be linked to a distinct SWB profile. This serves as a first step toward a larger study directly contrasting blips and non-blips to examine their timing and neurophenomenological correlates.³⁸ Moreover, the spatial resolution of our data is limited, underscoring the need for more advanced electrophysiology and neuroimaging to better map cortical and subcortical networks. Large-scale studies including epileptic and non-epileptic participants are needed to assess the prevalence and characteristics of blip-like phenomena, including their potential association with different seizure types.

In addition, because patients were taking various ASMs, a potential influence on SWB characteristics or the subjective reporting of blip-like sensations cannot be ruled out and should be investigated prospectively. Other potential confounding factors—age and epilepsy duration—should also be considered. Although previous studies have shown that SWB amplitude tends to decrease with age,³⁹ blippers in our sample—who were older and had higher epilepsy duration—exhibited increased amplitudes at burst level, suggesting that age alone is unlikely to account for the observed differences. Finally, since no direct associations between EEG discharges and phenomenological descriptions were assessed in this retrospective study, it remains possible that blips do not originate from epileptic activity and may instead be related to psychiatric comorbidities such as anxiety or to associated medications.

Although differences were clearly observed at the SWB level, complementary analyses revealed that these effects did not consistently generalize at the subject level. This discrepancy likely reflects a combination of high intra-subject variability, unequal numbers of events per participant, and limited statistical power for such analyses—particularly for subjects contributing only one or a few SWBs. These findings underscore the importance of interpreting our results as event-level phenomena rather than as stable, subject-level traits. They also point to the potential of SWB-specific EEG features as promising event-level biomarkers, and highlight the need for future

studies with greater statistical power and more balanced within-subject data.

5 | CONCLUSION

At the burst level, spike-and-wave bursts reveal distinct electroencephalographic patterns in patients susceptible to epileptic blips in generalized epilepsy. Although these findings do not systematically generalize at the subject level, they may point to candidate physiological features associated with the propensity to detect subtle impairments of consciousness. This line of investigation raises fundamental questions about how epileptic bursts may transiently disrupt the continuity of consciousness. Future research should further explore the clinical and theoretical dimensions of epileptic blips, refining their characterization and underlying mechanisms to enhance both scientific understanding and patient care.

AUTHOR CONTRIBUTIONS

Edgar Matringe contributed to Conceptualization, Funding Acquisition, Investigation, Data Curation, Formal Analysis, Methodology, Software, Visualization, and Writing – Original Draft Preparation. Aya Khalaf contributed to Conceptualization, Methodology, Formal Analysis, Resources, Software, Validation, and Writing – Review & Editing. Romain Granchamp contributed to Methodology, Software, Validation, and Writing – Review & Editing. Juan R. Vidal and Marcela Perrone-Bertolotti contributed to Writing – Review & Editing, Validation, and Supervision. Hal Blumenfeld contributed to Conceptualization, Methodology, Project Administration, Resources, Supervision, Validation, and Writing – Review & Editing. Laurent Vercueil contributed to Conceptualization, Funding Acquisition, Project Administration, Data Curation, Investigation, Methodology, Resources, Supervision, Validation, and Writing – Review & Editing.

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CONFLICT OF INTEREST STATEMENT

None of the authors has any conflict of interest to disclose.

DATA AVAILABILITY STATEMENT

The data that support the findings of this study are available from the corresponding author upon reasonable request.

ETHICS STATEMENT

This retrospective study, covering the period from 2004 to 2023, was approved by the Grenoble Institutional Ethics Committee for Research (approval number CERGA-Avis-2024-08). We confirm that we have read the Journal's position on issues involved in ethical publication and affirm that this report is consistent with those guidelines.

PATIENT CONSENT STATEMENT

Patients were informed of their rights—including the option to decline participation and the assurance of personal data protection—2 months before the study began. Given the retrospective nature of the study and its approval by the ethics committee, the need for informed consent was waived, in accordance with the principles of the Declaration of Helsinki.

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SUPPORTING INFORMATION

Additional supporting information can be found online in the Supporting Information section at the end of this article.

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