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ORIGINAL ARTICLE

Spike detection in the wild: Screening of suspected temporal lobe epilepsy cases using a tailored 2-channel wearable EEG

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Abstract

Objective: To clinically validate the contribution of a custom-built EEG wearable device (waEEG) compared to a full 10–20 electrode array ambulatory EEG (aEEG) for screening epilepsy cases in patients with suspected temporal lobe epilepsy (TLE) but negative routine EEGs.

Methods: Patients (aged 16–91 years) with clinically suspected TLE who were referred for a 24 h aEEG were fitted with an additional 2-channel bipolar waEEG device and prospectively enrolled in the study until 20 TLE diagnoses were confirmed by aEEG. 41 patients were included and their waEEG was blindly reviewed by two experienced clinical neurophysiologists and a semi-automated spike detection software to categorize patients into TLE (spikes present) and non-TLE (no spikes) groups.

Results: The experts achieved good sensitivity (95%–100%) and accuracy (98%–93%) with excellent interrater agreement (kappa>0.80) in patient labelling. The semi-automated software performed poorly (40% sensitivity, 68% accuracy) and failed to classify TLE in more than half the cases. Classification was not affected by restricting spike detection to the evening and night time, which reduced the average length of the analyzed EEG from 23.4 to 10.4 h. Three false-positive spike detections were thoroughly analyzed and reclassified as artifacts due to eye and body movements and electrocardiographic contamination. To better control cardiac artifacts, the addition of an ECG channel to the waEEG is recommended.

Significance: Detection of spikes with waEEG allows accurate detection of epilepsy in suspected TLE cases, with less technical and professional effort and improved acceptance. This screening tool could improve the yield of follow-up with a conventional aEEG and provide an accessible method for monitoring interictal epileptiform activity in TLE.

Plain Language Summary: Epilepsy is a chronic short circuit in the brain. In adults, it most often affects the temporal lobes, resulting in temporal lobe epilepsy

(TLE). Seizures are infrequent but difficult to treat. Electroencephalography (EEG) is the best method to detect the electrical disturbances and is crucial to distinguish epilepsy from other non-epileptic disorders. Developing simple, inexpensive and easily accessible portable EEG methods that complement in-hospital assessment could significantly impact patient care. Our study aims to clinically validate a wearable epilepsy screening device to aid in TLE management, reduce delays in diagnosis and enable straightforward assessment of epileptic activity.

KEYWORDS

electroencephalography, epilepsy, neurotechnology, temporal lobe epilepsy, wearable devices

1 | INTRODUCTION

Temporal lobe epilepsy (TLE) is one of the most common forms of epilepsy in the general population[1,2] and has a significant impact on a wide age range from childhood to the geriatric population.[3] The clinical diagnosis of TLE can be challenging[4–8] as the characteristic focal aware and focal impaired awareness seizures are not always easily recognized as manifestations of epilepsy,[9] occur infrequently and also coexist with competing behavioral and cognitive disorders.[10–12] In this context, electroencephalography (EEG) plays a fundamental role as it can detect characteristic interictal epileptiform discharges (IEDs), namely spikes in a typical scalp topography[13] as well as ictal patterns that can be detected over the temporal lobe,[14,15] essentially the anterior area.[16] Nonetheless, the scarcity of these elements may limit the ability of standard clinical sleep EEG to make a diagnosis.[17] Current clinical practise in this scenario is to refer the patient for long-term EEG recordings, either 24 h ambulatory EEG (aEEG) or inpatient video-EEG monitoring (vEEG) to improve yield,[18,19] as it has been recognized that prolonging recordings is more effective than increasing the number of electrodes to detect missed IEDs.[20] As in most cases where TLE is suspected, seizures are occasional, aEEG recordings are more cost-effective than the vEEG technique, as the additional cost of obtaining video recordings rarely makes a useful additional contribution (e.g. recording of seizures) to the features found on EEG, which are similar for both methods.[21]

The consistent observation that the epileptiform activity seen in the EEG of TLE is found over the temporal lobe electrodes, with little or no contribution from extratemporal lobe electrodes,[13,22,23] opens the possibility for further technical simplification of the standard aEEG, by limiting the electrode map to the temporal areas and the use of EEG-based wearable devices (waEEG). An important goal of these home studies is to achieve a level of technical simplification that allows the recordings to be set up and maintained by the patient and/or family, which would pave the way for long-term and cost-effective recordings. The combination with Internet of Things in Healthcare (IoTh) technology for remote recording and processing of the EEG would then enable long-term, non-invasive and individualized monitoring of epileptiform activity. In this study, we aimed to use a prototype waEEG device, previously used by our group[24,25] and referred to as Neury to explore the capabilities of a simple recording system to reliably detect temporal lobe spikes in undiagnosed patients with suspected TLE, which could support a solid diagnosis of this condition. In this way, we sought to answer the following questions:

Key points

- TLE is the most common focal epilepsy in adults and shows typical but poorly specific symptoms.
- Scalp spikes in the EEG, together with the typical clinical picture, enable a robust differentiation of epileptic cases, e.g. TLE, from non-epileptic conditions.
- Spikes in the temporal lobe can be reliably detected in aEEG but can easily be overlooked in short-term routine clinical EEG.
- Temporal lobe spikes can be identified in the nocturnal waEEG by experienced clinical neurophysiologists with similar consistency as in the full array aEEG.
- Reduced spatial EEG sampling with a wearable device can consistently detect epilepsy cases in patients with clinical suspicion of TLE.

1. What is the clinical usefulness of waEEG compared to aEEG in the management of suspected cases of TLE?
2. Can an accessible waEEG screening and/or follow-up method be established for use in the home environment in clinical cases of suspected TLE?

2 | METHODS

2.1 | Patients and clinical data

We conducted a prospective cross-sectional study with a consecutive sample of adult patients clinically suspected of TLE and referred for complementary neurophysiological assessment with an aEEG at the Department of Neurophysiology of Hospital Júlio de Matos, from June 2020 to December 2022. Patients were invited to participate in the current study by wearing a small recording device alongside the medical aEEG device. They were informed that the aim of the study was to investigate simplified techniques of epilepsy diagnosis compared to standard aEEG methods. All participants signed an informed consent form. The study was approved by the institutional ethics committee (approval number 00482020) and is in accordance with the Declaration of Helsinki. Anonymity was maintained and all results obtained were used exclusively for academic purposes and without financial interest. Patients derived from the hospital's outpatient neurology and psychiatry clinic and from other public hospitals in the vicinity. Recordings were prospectively collected and anonymously labeled so that they were blind to the subsequent diagnosis resulting from the associated aEEG (which was chosen as the gold standard). Patient enrolment continued until 20 clinical aEEG confirmations for TLE were available, resulting in a total of 43 EEG recordings, of which 2 were excluded due to significant electrode artifacts (Table 1).

2.2 | EEG recordings

The aEEG recordings were performed with genuine Grass® gold cup electrodes applied to the scalp with collodion and conductive gel. An array of 31 EEG channels (Fp1/2, F3/4, C3/4, P3/4, O1/2, F7/8, T7/8, P7/8, Fz, Cz, Pz, F9/10, FT9/10, FC1/2, FC5/6, CP1/2 and CP5/6) was used, (Figure 1A), as well as an ECG lead. An ambulatory medical grade electroencephalography device (Siesta™, Compumedics Inc.®) was used to record the previous 32-channel setup at 256 Hz, using a bandwidth of 0.5–70 Hz and a 50 Hz notch filter.

The 2-channel (bipolar) waEEG was recorded continuously with the above-mentioned Neury device. We also placed Grass® gold cup electrodes with collodion at positions T1-TP7 and T2-TP8 (Figure 1B),[13,26] with the grounded snap electrode in the chest, which also supported the device. After acquisition, the file was transferred to the laptop and converted to EDF format. The cross-platform open-source software EDFbrowser version 2.04 (available at https://www.teuniz.net/edfbrowser/) was used for this purpose.

The aEEG reports were generated after a standard visual assessment of the entire recording by the senior clinical neurophysiologist (AL). The subjective impression was that the vast majority of spikes that ultimately supported the diagnosis of TLE occurred in the evening and at night, partly due to the better signal-to-noise ratio of the recording during these periods as there are fewer overall artifacts. This observation provided the opportunity to shorten the processing time of the study recordings, and accordingly all waEEG recordings were trimmed to the nocturnal sleep period. The final 41 remastered wearable EEG recordings were blindly and randomly reviewed by two experts (AL and DFB) at the end of the study inclusion period. Patients with aEEG recordings without spikes in the temporal lobe electrodes were categorized as non-TLE and included non-epileptic and extra-TLE patients (only one case with parietal spikes, Table S1). Those with spikes in the temporal lobe electrodes were classified as TLE patients.

2.3 | Spike detection and analysis

The spikes were recognized by both experts by visual inspection, and the spikes used for diagnosis were marked in each patient (Figure 2A). Spike detection was also performed with an automated algorithm used by the Reveal software in Persyst®14. The software was installed on an HP® ENVY×360 Convertible 15-dr0008np with an Intel® Core™ i7-8565U CPU @ 1.80GHz processor (OS Microsoft™ Windows 11, 64 bit), (RAM 16 GB DDR4), followed by visual post-processing to exclude artifactual detections. Some patients had very few spikes (cases N131, N122, N121, N109, N80, N57, Figure 2), which prevented analysis of hemispheric lateralisation.

2.4 | Statistical analysis

The statistical analysis was performed with IBM® SPSS® Statistics, Version 29 (IBM Corp). A combination of visual inspection of histograms with kurtosis and skewness and the Kolmogorov–Smirnov normality test was used to test

TABLE 1 Clinical, EEG and MRI data from TLE and non-TLE patients.

	Patient	Age (y)	Sex	Routine EEG	aEEG	MRI	Clinical manifestations
TLE	N15	42	F	Left temporal lobe IED	Left temporal lobe IED	*n/a*	Recurrent impairment of consciousness. Septic endocarditis.
	N26	49	F	Normal	Fronto-temporal IED	*n/a*	Recurrent impairment of consciousness.
	N45	18	F	Normal	Right temporal lobe IED	Hippocampal asymmetry	Nocturnal hypermotor episodes with impaired consciousness.
	N57	82	F	Left temporal lobe SW	Left temporal lobe SW and ShWD	*n/a*	Recurrent impairment of consciousness.
	N78	18	F	Normal	Bilateral temporal lobe IED	*n/a*	Recurrent impairment of consciousness.
	N80	91	F	Left temporal lobe SW and IED	Left temporal lobe SW and IED	Leukoencephalopathy	Language impairment, impaired consciousness and tremor in the right face.
	N87	78	F	Normal	Right temporal lobe SW and IED	*n/a*	Post stroke epilepsy.
	N91	32	F	*n/a*	Left temporal lobe SW and IED	Normal	Language impairment and evolution to GTCS
	N92	77	F	Normal	Bilateral temporal lobe IED	*n/a*	Post traumatic epilepsy with FIAS.
	N97	49	M	Normal	Left temporal lobe IED	*n/a*	Fainting episodes.
	N102	47	F	Normal	Left temporal lobe IED	Bilateral mesial temporal lobe sclerosis	FIAS starting at 11y.
	N103	78	M	Normal	Bilateral temporal lobe IED	Right caudate nucleus stroke	Episodes of impaired consciousness preceded by difficulties in speaking.
	N106	64	F	Left temporal lobe SW	Left temporal lobe SW and IED	Normal	Episodes of memory impairment lasting for 24 h.
	N109	79	M	Left temporal lobe SW	Left temporal lobe SW and IED	*n/a*	Sincopal episodes.
	N111	20	F	Normal	Bilateral temporal lobe IED	Normal.	FIAS starting at age 16 years.
	N113	38	F	Normal	Bilateral temporal lobe IED	*n/a*	FIAS starting at age 17 years.
	N121	50	M	Normal	Bilateral temporal lobe IED	*n/a*	Schizophrenia and epilepsy. Syncopal episodes lasting about 1 min.
	N122	55	M	Normal	Right temporal lobe SW and IED	Neocortical right temporal lobe lesion	Post-traumatic epilepsy.
	N130	44	F	Normal	Left temporal lobe SW and IED	*n/a*	FIAS with automatisms.
	N131	29	M	Left temporal lobe IED	Left temporal IED	*n/a*	Visual and auditory halucinations.

TABLE 2 (Continued)

	Patient	Age (y)	Sex	Routine EEG	aEEG	MRI	Clinical manifestations
non-TLE	N29	45	F	*n/a*	Normal.	*n/a*	Consciousness impairment episodes.
	N55	60	F	*n/a*	Normal.	Right frontal and Temporal Cavernomas	Daily fainting spells and reduced motor tone.
	N66	16	F	Normal	Normal.	*n/a*	FIAS and psychogenic seizures.
	N67	64	M	*n/a*	Normal.	*n/a*	Two tonic seizures in the night sleep.
	N69	46	F	Normal	Normal.	*n/a*	Psicosis with episodes of impaired consciousness.
	N72	76	M	Normal	Normal.	*n/a*	Intermitent confusional episodes.
	N88	20	M	Left temporal lobe SW	Normal.	*n/a*	Temporary left hemiparesis. High vascular risk
	N94	19	M	Normal	Left parietal IED	Left frontal cyst	Episode of loss of consciousness.
	N95	23	F	Normal	Normal.	*n/a*	Bipolar disorder. Episodes of impaired consciousness.
	N98	28	F	Normal	Normal.	*n/a*	Seizure episode.
	N99	38	F	*n/a*	Normal.	*n/a*	Refractory epilepsy.
	N101	55	F	*n/a*	Normal.	*n/a*	Bipolar disorder with confusing episodes and loss of consciousness.
	N107	60	F	Normal	Normal.	*n/a*	Episodes of impaired consciousness
	N114	25	M	Normal	Normal.	*n/a*	Episodes of mental confusion.
	N116	64	F	*n/a*	Normal.	*n/a*	Episodes of memory impairment. Generalized epilepsy and meningitis in childhood.
	N117	61	M	*n/a*	Normal.	Cerebelar atrophy	Behavioral episodes interpreted as epilepsy. Not treated with medication.
	N123	49	F	Normal	Normal.	Normal.	Two episodes of transient global amnesia.
	N124	74	F	Normal	Normal.	*n/a*	Three episodes of transient global amnesia.
	N126	40	M	Normal	Normal.	Neocortical left temporal lobe lesion	Brain trauma at the age of 21, with posterior seizures and psychotic symptoms.
	N128	59	M	Normal	Normal.	*n/a*	Episodes of transient global amnesia.
	N129	17	M	Normal	Normal.	*n/a*	Sonambulism/FIAS? Behavioral abnormalities.

Abbreviations: aEEG, 24 h ambulatory EEG; EEG, electroencephalography; FIAS, focal impaired awareness seizures; GTCS, generalized tonic-–clonic seizures; MRI, magnetic resonance imaging; n/a, not available; ShWD, sharp-wave discharge; SW, slow waves; TLE, temporal lobe epilepsy.

all quantitative variables for normality. For descriptive purposes, normally distributed variables were reported with mean ± standard-deviation, while non-normally distributed quantitative variables and ordinal variables were described with median (Me) and interquartile range (IQR). Absolute and relative frequencies were used to describe qualitative variables. The number of correct and incorrect classifications for each user of waEEG was

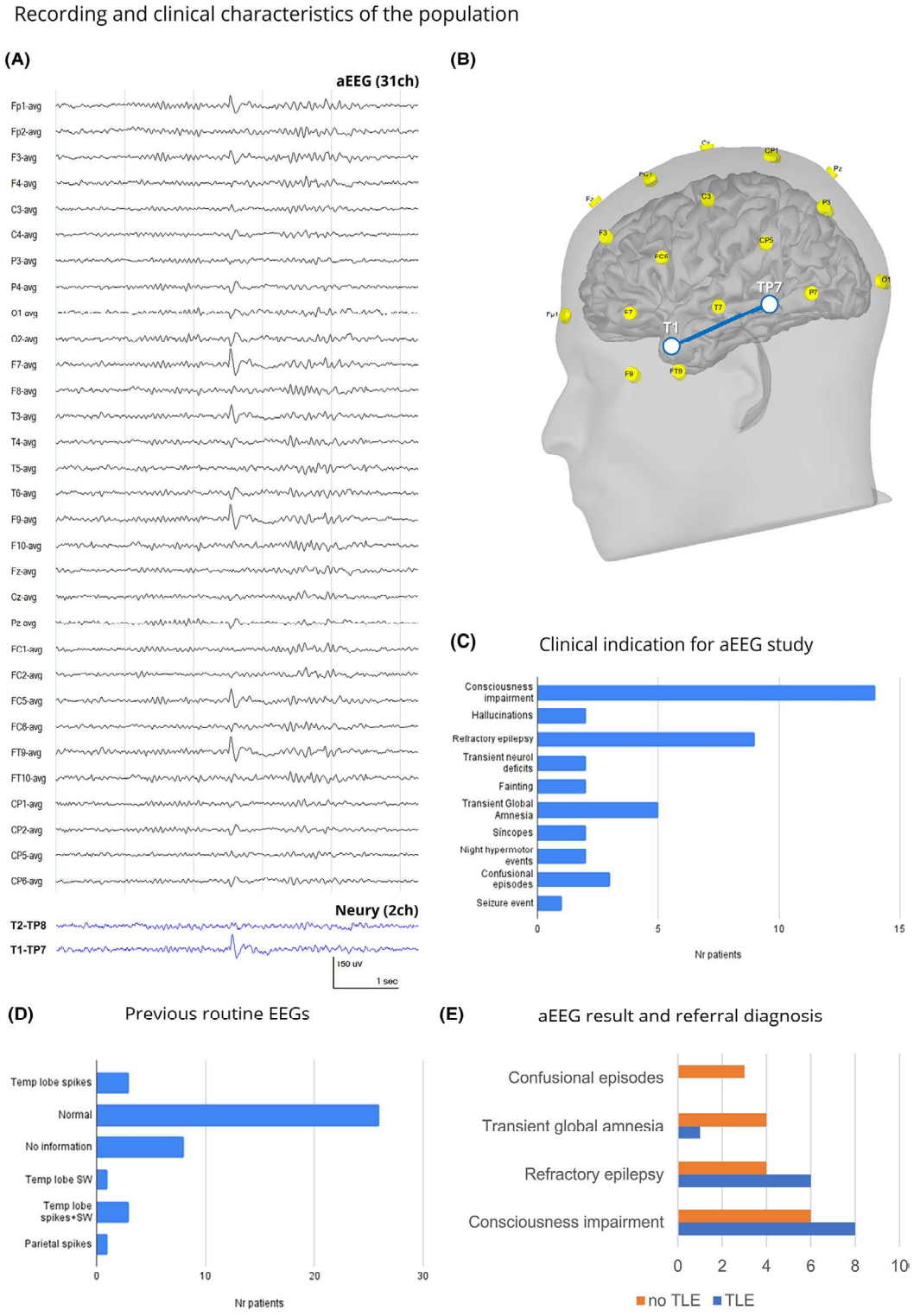

FIGURE 1 Recording and clinical characteristics of the population. (A) Example of a spike detected by aEEG and waEEG, simultaneously. (B) Schematic representation of the wearable EEG montage and the anatomical relationship to the temporal lobe. (C) Clinical motivation for the prescribed aEEG prolonged study. (D) Results of previous routine EEG studies. (E) Final classification of patients by principal diagnosis prior to referral. AEEG, Ambulatory EEG; waEEG, Wearable EEG.

described. Sensitivity was determined as the true-positive rate in patients with TLE (classified by the gold standard method); specificity was determined as the true-negative rate in patients without TLE; accuracy was determined as the ratio of the true classification to the total number of patients. For inferential analysis, differences in

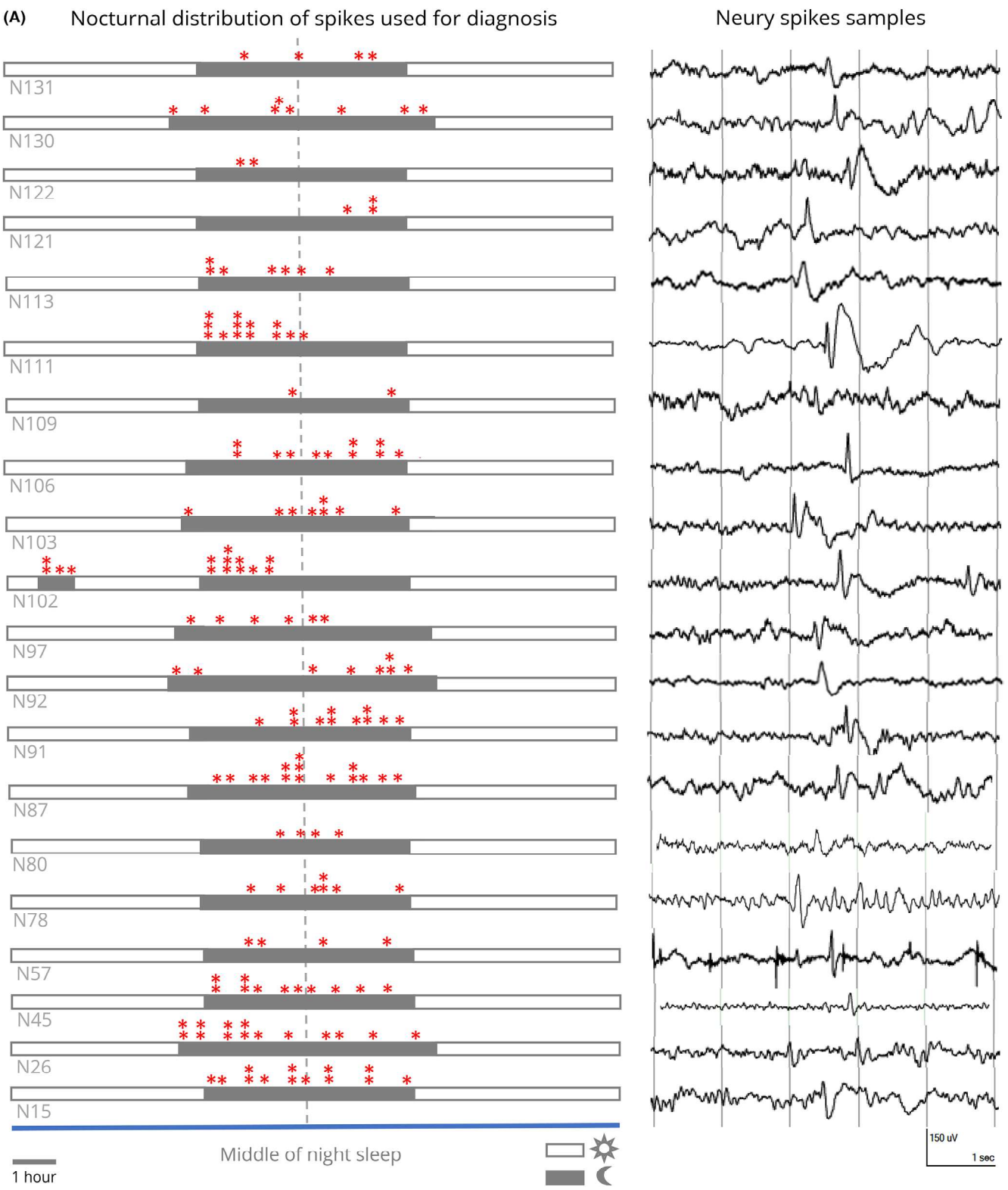

FIGURE 2 Neury (waEEG) findings in TLE patients. Nocturnal distribution of spikes used for diagnosis, in each individual patient (left). Different examples of individual spikes (right).

assessment time between methods were assessed using the non-parametric Wilcoxon signed rank test for paired samples. Agreement in the identification of patients with TLE between the gold standard and waEEG, between different observers using waEEG, and between manual and automated analysis of waEEG was assessed using Cohen's kappa statistic[27] with 95% confidence intervals (95% CI). Kappa scores were interpreted as follows: <0 as less than chance agreement, 0.01–0.20 as slight agreement, 0.21–0.40 as fair agreement, 0.41–0.60 as moderate agreement, 0.61–0.80 as substantial agreement, and 0.81–1.00 as near perfect agreement.[28]

A type I error of .05 was considered for all comparisons.

3 | RESULTS

3.1 | Patients demographics and clinical characterization

Forty-one patients were included in this study (15 men, 26 women), with a mean age of 48.3 ± 21.3 years. Women predominated in both the TLE (70%) and non-TLE (57%) groups. The most common clinical condition leading to an EEG was recurrent episodes of impaired consciousness (34.1%), which was differently distributed in patients with and without TLE (40.0% and 28.6%, respectively). The second most common was transient global amnesia (12.2%), which occurred in 2% of TLE and 19% of non-TLE patients (Figure 1C).

Information on previous routine EEGs performed prior to inclusion in the study was available for 33 patients (19 in the TLE and 14 in the non-TLE group), with 26 findings being normal (13 in each patient group) (Figure 1D). The one abnormal report in the non-TLE group mentioned 'slow waves in the temporal lobe', which was not confirmed by a follow-up EEG. Of the 6 abnormal reports in the TLE group, 3 found spike–wave complexes in the temporal lobe, while the remaining 3 found spikes in the temporal lobe. Overall, in 19 patients in the TLE group, aEEG provided diagnostic information in 13 cases that had previously been reported as normal and supplemented abnormal, but non-diagnostic previous reports in 6 cases, showing a significant clinical impact on this group of patients. Magnetic resonance imaging (MRI) reports were available for 13 patients (8 in the TLE and 6 in the non-TLE group), which showed abnormal findings in both groups (5 in the TLE and 4 in the non-TLE group), (Table 1). The structural lesions were heterogeneous and mainly involved the temporal lobes (5 of 9) and occurred in both the TLE group (3) and the non-TLE group (2). These clinical, electroencephalographic and imaging data are summarized in Table 1.

3.2 | TLE diagnosis in waEEG and inter-rater agreement

Visual inspection of the data was performed independently by DFB and AL, taking into account only the evening and nighttime sleep periods. It revealed a single stable morphological spike or a spike-and-slow-wave complex, more than twice, in each patient throughout the entire recording (Figure 2, right). The length of the reviewed EEG ($z = -5.44$, $p < 0.001$) for the waEEG (Me 10.4 h, IQR 1.4 h) was significantly shorter for waEEG (Me 10.4 h, IQR 1.4 h) than for aEEG (Me 23.4 h, IQR .9 h). The sensitivity for the detection of TLE was 95.0% and 100% and the specificity was 100% and 85.7%. This resulted in an accuracy of 97.6% and 92.7%. The automated analysis with Persyst®14 showed a lower sensitivity and accuracy (40.0% and 68.3%), with a specificity of 95.2%. The agreement between waEEG and the gold standard is summarized in Table 2. Our results show that this wearable can provide sufficient data for a user to correctly classify a patient as having TLE. Indeed, the kappa scores showed an agreement with aEEG of over 0.80 for both raters ($\kappa = 0.951$, 95%CI [0.857; 1.00] and $\kappa = 0.854$, 95%CI [0.697; 1.00]). Inter-rater agreement was also favorable when using waEEG ($\kappa = 0.807$, 95%CI [0.631; 0.983]). On the other hand, automatic classification using wearable data showed lower agreement with both the gold standard method ($\kappa = 0.357$, 95%CI [0.116; 0.598]) and the expert analysis of waEEG ($\kappa = 0.288$, 95%CI [0.029; 0.547] and $\kappa = 0.269$, 95%CI [0.051; 0.487]).

3.3 | False positive analysis

Preliminary data analysis of the waEEG recordings by the lead clinical neurophysiologist (AL) revealed 3 incorrect TLE diagnoses based on inaccurate spike detections (Figure 3A). A more detailed analysis of these events with simultaneous aEEG and ECG allowed their reclassification as artifacts. The events in one patient were generated by cardiac potentials due to ventricular extrasystoles (Figure 3B). The events in another patient were reclassified as eye movement artifacts. The events in the third case were reclassified as head movement artifacts.

4 | DISCUSSION

We conducted a real-world study on the ability of a waEEG device to detect epilepsy in patients who were clinically suspected of having TLE but whose routine EEGs were inconclusive. The promising results in terms of sensitivity, with strict control of the false positive (FP) rate, open up interesting possibilities for effective screening of epilepsy

TABLE 2 Statistical assessment of waEEG-based classification between experts and the semi-automatic algorithm.

		Rater 1		Rater 2		Persyst 14		
		Non-TLE	*TLE*	*Non-TLE*	*TLE*	*Non-TLE*	*TLE*	**Total**
aEEG	*Non-TLE*	21	0	18	3	20	1	21
	TLE	1	19	0	20	12	8	20
	Kappa	0.951 [0.857; 1.00]		0.854 [0.697; 1.00]		0.357 [0.116; 0.598]		–
	Sensitivity	95.0%		100.0%		40.0%		–
	Specificity	100.0%		85.7%		95.2%		–
	Accuracy	97.6%		92.7%		68.3%		–
Persyst 14	*Non-TLE*	20	12	17	15	–	–	32
	TLE	2	7	1	8	–	–	9
	Kappa	0.288 [0.029; 0.547]		0.269 [0.051; 0.487]		–		–
Rater 2	*Non-TLE*	18	0	–	–	–	–	18
	TLE	4	19	–	–	–	–	23
	Kappa	0.807 [0.631; 0.983]		–		–		–
Total		22	19	18	23	32	9	41

Note: 95% confidence intervals are represented for Cohen's kappa.

cases prior to the traditional aEEG-based methodology, with noteworthy improvements in cost, tolerability and availability. Such reliable screening may contribute to an optimized use of resource-limited long-term diagnostic techniques and allow easy point-of-care assessment of interictal epileptiform activity, both of which may improve clinical decision making in TLE patients.

4.1 | Previous clinical and EEG data and diagnosis of TLE

In our patient cohort, previous routine EEG examinations revealed either no spike activity (26 of 36) or rare temporal lobe spikes (3 of 36) (Figure 1D), which prompted the treating physicians to perform further neurophysiological examinations with an aEEG before the final diagnosis of TLE was accepted. Clinical assessment in this group of patients proved remarkably effective in suspecting TLE among the various types of possible epilepsies, as only one case of extratemporal lobe epilepsy occurred in the total of 21 epilepsy cases in our cohort (patient N94, Table 1). The fact that TLE is the most common form of focal epilepsy in adulthood was certainly an important contributing factor. The aEEG allowed a clear distinction between epilepsy cases with spikes, which are probably epileptic, and cases without spikes, which are probably not epileptic, and compensated for the low specificity of the clinical assessment. The fact that 20 of 21 patients in our series with spikes were localized in the temporal lobe further supports the diagnosis of TLE compared to other forms of epilepsy. Overall, in our cohort of patients referred for aEEG recordings with the clinical suspicion of TLE, but with little or no support from routine EEGs, this diagnostic hypothesis was confirmed by the detection of spikes over the temporal lobe in 49% of cases, with only one case of extratemporal epilepsy. This suggests that referring physicians do well in distinguishing TLE from other epilepsies but cannot rule out non-epileptic disorders in about 50% of cases. The additional detection of spikes over the temporal lobe is particularly effective in later differentiation and complements the clinical assessment very well. Patients with TLE and spike activity limited to the posterior areas may escape detection by the 2-channel waEEG recording, as only the middle and anterior temporal areas are sampled. No such patient could be found in our series, leading us to believe that they are rare compared to the typical TLE patients with anterior spikes and may have different clinical manifestations than those used by clinicians to suspect TLE.

4.2 | Statistical performance of waEEG spike detection

The high sensitivity in detecting spikes in the waEEG (95% and 100%) by the experts supports our hypothesis

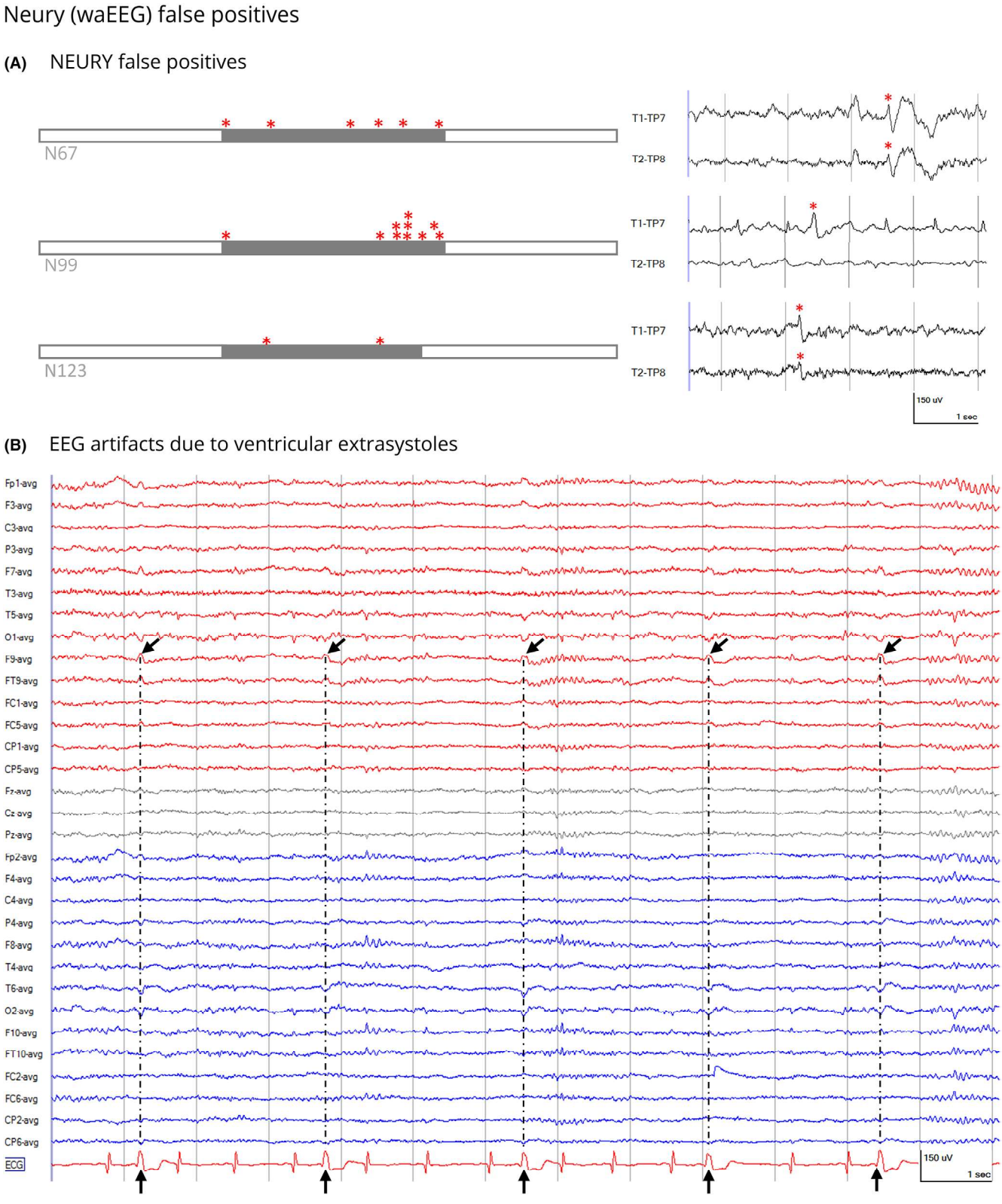

FIGURE 3 Neury (waEEG) false positives. (A) Individual representation of the 3 false-positive classifications in the waEEG data set and the corresponding EEG traces. (B) Extended 10/20 electrode array aEEG epoch showing the EEG artifact induced by ventricular extrasystoles in patient N99. WaEEG, Wearable EEG.

that most of the spikes contributing to the diagnosis of TLE in the aEEG can also be detected in the simplified recordings, leading to the same supportive evidence for the diagnosis of epilepsy. The savings in preparation and review time are considerable, even more so if recording is restricted to nighttime when most spikes are more consistently recognized. This leads to an improvement in cost-effectiveness, tolerability and availability, which could have a positive impact on clinical practise. The inter-rater concordance for TLE classification based on waEEG was 0.81 (Table 2), demonstrating a robust discrimination between patients with and without spikes. This result supports the interpretation that the greatest diagnostic yield of long-term aEEG for epilepsy diagnosis can be achieved with waEEG restricted to the nocturnal period. Previously published attempts to simplify the use of aEEG for TLE diagnosis were evaluated by Asif et al.[22] using automatic seizure detection in a publicly available dataset. They found a gradual deterioration in the ability to detect seizure events in the temporal lobes when the number of electrodes used was gradually reduced. They did not study spikes, but several reports have shown that subtemporal electrodes are superior in detecting spikes originating in the temporal lobe,[13,29] as they typically have a much higher potential than the extratemporal electrodes (Figure 1A). The bipolar montage of electrodes surrounding only the temporal lobe used in the present study was good at detecting the more frequent anterior discharges while maintaining sensitivity for the posterior ones, and the signal-to-noise ratio was quite similar to that of aEEG.

4.3 | Automatic vs. visual classification

The performance of automatic spike detection followed by visual post-processing of the waEEG datasets was worse for TLE classification than that of visual spike detection, mainly due to low sensitivity (Table 2). The further observation that no FP classifications were obtained with the semi-automated method suggests that the limiting factor with this technique is the ability of the spike detection software to identify good spike candidates, while post-processing appears to effectively remove the FP. The poor performance of semi-automated spike detection software is consistent with recent data on the subjective opinion of professionals involved in EEG processing of epilepsy cases. These report a widespread lack of confidence in currently available software options for fully automated spike and seizure detection.[30] Several factors may contribute to the poor performance in our dataset, including very few spikes in the entire recording of some patients, noisier and more frequent artifacts in outpatient recordings compared to inpatient settings used for training the algorithm. We were unable to identify a single factor explaining these results, which will likely require much more thorough analysis with larger cohorts.

The possibility that the spikes detected in the waEEG originate outside the temporal lobes, despite the clinical picture suggestive of TLE, cannot be excluded due to the limited spatial sampling of such recordings. However, we believe that such a scenario is relatively rare, as evidenced by the fact that all cases with waEEG spikes in our series showed the typical spatial distribution for TLE in an extended electrode array (31 channels) of the accompanying aEEG. To avoid the above-mentioned limitation, we believe that patients with spikes on waEEG should undergo a more extensive EEG examination to better map the spikes and detect possible concurrent spikes elsewhere in the scalp. The isolated spikes we saw in TLE patients were not mistaken for physiological variants, such as wicket spikes, but the ECG produced more insidious artifacts that led to the misclassification of a patient with extrasystoles (Figure 3B) and suggested to us the usefulness of adding an ECG recording channel for waEEG montage in future recordings. In our study, expert visual analysis is a prerequisite for analyzing waEEG data to achieve the same performance as aEEG analysis in identifying the critical spikes for reliable TLE diagnosis. The limitation of the reviewed waEEG to the evening-night period and the small number of channels that need to be visually monitored allows the clinical neurophysiologist a faster workflow than with the aEEG. In the authors' limited experience, this could correspond to a quarter of the time required for a complete 24-hour aEEG.

4.4 | Proposal for a waEEG-based protocol of TLE diagnosis

The results of the previous analysis demonstrate the ability of nocturnal waEEG recordings with visual inspection to detect interictal phenomena leading to an epilepsy diagnosis with high sensitivity and a low number of FP. Misclassification of non-TLE cases as TLE cases in waEEG is a potentially serious problem that could ultimately render the methodology worthless if a robust strategy to overcome this problem is not found. It results from the detection of false spikes due to unrecognized artifacts. Identified artifacts that lead to false spike detections include heart, eye and head movements. Knowledge of these artifacts can further improve FP rate control. We propose the following strategies to improve the robustness of waEEG spike detection:

1. Mapping an ECG channel to the setup is crucial for the detection of cardiac artifacts.
2. The detection of eye movements can be improved by comparison with the artifacts caused by such movements during the calibration procedures at the beginning of the recording.
3. Movement artifacts can be minimized by limiting the analysis of the recordings to the evening and night sleep period.

Overall, our study suggests that in cases with clinical suspicion of TLE, neurophysiological diagnosis of epilepsy (spike detection) can be confirmed in a nocturnal waEEG recording with a comparable accuracy as with a standard 24 h aEEG with full 10–20 electrode array, offering an attractive cost–benefit profile and improved acceptability. The limited spatial sampling of waEEG is a major limitation of its ability to properly map TLE spikes and renders it incapable of detecting independent spikes at extratemporal locations. Both can be reliably performed with conventional long-term EEGs and are of fundamental importance for a neurophysiological diagnosis of TLE. In this context, waEEG has the potential to complement, but not replace, aEEG.

In current clinical practise in patients with a clinical suspicion of TLE, waEEG may have a role as a complementary screening method for epilepsy when routine EEG shows little or no support for this diagnosis, as the much larger temporal sampling can provide additional valuable data on spike consistency and hemispheric laterality if enough events are detected. The strong support for epilepsy underlying the clinical picture provides a useful basis for complementary investigations aimed at a more accurate diagnosis of the individual condition. In such cases, early referral to specialist epilepsy centres with expertise in imaging protocols for TLE and an existing epilepsy surgery programme could optimize resources, avoid gaps and delays in treatment and thus improve clinical decision making.

5 | CLINICAL RELEVANCE OR FUTURE DIRECTIONS

The good performance of waEEG in classifying patients with suspected TLE shows its potential as an intermediate test for epilepsy, outperforming routine EEG but requiring much fewer resources than standard aEEG. The shorter preparation time, better tolerability and lower social burden are further advantages. A clear limitation of the waEEG is the inability to detect extratemporal lobe epilepsies, for which the extended spatial mapping of the is a clear advantage.

AUTHOR CONTRIBUTIONS

DFB performed the data analysis and wrote the manuscript. JIS performed the statistical analysis and prepared the corresponding tables and the manuscript part. DD and HC collected EEG data and clinical data. AL contributed to the concept and design of the study and revised the manuscript for intellectual content.

CONFLICT OF INTEREST STATEMENT

The authors declare that they have no known competing financial interests or personal relationships that could have influenced the work on this article. 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.

DATA AVAILABILITY STATEMENT

Deidentified data is available upon reasonable request.

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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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