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SUPPLEMENT ARTICLE

# Detection of convulsive seizures using surface electromyography

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**Summary**

Bilateral (generalized) tonic–clonic seizures (TCS) increase the risk of sudden unexpected death in epilepsy (SUDEP), especially when patients are unattended. In sleep, TCS often remain unnoticed, which can result in suboptimal treatment decisions. There is a need for automated detection of these major epileptic seizures, using wearable devices. Quantitative surface electromyography (EMG) changes are specific for TCS and characterized by a dynamic evolution of low- and high-frequency signal components. Algorithms targeting increase in high-frequency EMG signals constitute biomarkers of TCS; they can be used both for seizure detection and for differentiating TCS from convulsive nonepileptic seizures. Two large-scale, blinded, prospective studies demonstrated the accuracy of wearable EMG devices for detecting TCS with high sensitivity (76%-100%). The rate of false alarms (0.7-2.5/24 h) needs further improvement. This article summarizes the pathophysiology of muscle activation during convulsive seizures and reviews the published evidence on the accuracy of EMG-based seizure detection.

**KEYWORDS**

biomarkers, electromyography, Seizure detection, tonic, tonic–clonic seizures

## 1 | INTRODUCTION

Unpredictability and unawareness of seizure occurrence is distressing and disabling for patients with epilepsy and their caregivers.[1] This contributes to social isolation and decreased quality of life. There is a well-documented need for wearable seizure detection devices.[2–8] Bilateral or generalized tonic–clonic seizures (TCS) may lead to injuries, and they constitute a primary risk factor for sudden unexpected death in epilepsy (SUDEP), especially when patients are unattended.[9,10] Wearable seizure detection devices have been suggested as a tool to prevent SUDEP,[1,9,10] and a statement of research need on epilepsy deaths from UK research teams in 2014 encouraged development of seizure detection devices that may prevent SUDEP.[11]

Treatment as well as clinical trials of antiepileptic drugs (AEDs) typically use self-reporting of seizures. However, this is unreliable: in a video–electroencephalography (EEG) monitoring unit, 61% of the seizures remained unnoticed by the patients, especially focal to bilateral TCS (or secondarily generalized tonic–clonic seizures [GTCS]).[12] Patients who appear to be seizure-free and not needing of further antiepileptic drug (AED) adjustments may have undetected seizures, especially in sleep where, in addition, they are exposed to an increased risk of SUDEP.[9,10] Hence, there is a need for wearable seizure-detection devices that can both provide better, objective data about when and how often seizures occur, and alert assistance.

Wearable devices and gadgets are becoming widely used. Their impact is already being felt in education, communication, navigating, and entertainment, and this trend is now reaching healthcare applications, including seizure detection. There are thousands of wearable devices on the market that measure health parameters and signs. Market research reports have predicted an exponential growth in this field: the number of wearable devices shipped will rise from about 13 million in 2013 to 130 million in 2018, and market size will jump from $1.4 billion in 2013 to $19 billion in 2018.[13,14] Companies developing wearable healthcare devices include key players like Apple, Fitbit, Google,

and Samsung.[14] In contrast with the rapid technological development, the scientific evidence for the diagnostic accuracy of these wearable healthcare devices is disappointingly scarce, which limits their integration into rational medical decision making and reimbursement by healthcare providers.

Signals from muscles recorded with surface electromyography (sEMG) seem to be a promising modality for detection of motor seizures. In this review, we summarize the quantitative sEMG changes during convulsive seizures that constitute the basis for seizure detection, and we review the published evidence on EMG-based, wearable seizure-detection devices.

**Key Points**

- There are specific changes in quantitative surface EMG parameters during TCS
- High-frequency components of the EMG signal increase at onset of TCS
- The ratio of high- and low-frequency EMG wavelet components provides an objective measure for determining duration of the TCS phases
- Wearable devices using EMG-based algorithms accurately detect TCS
- EMG signals differentiate between TCS and psychogenic nonepileptic convulsive seizures

## 2 | PATHOPHYSIOLOGY OF MUSCLE ACTIVATION DURING CONVULSIVE SEIZURES

Motor neurons communicate with muscles via the neuromuscular junction. Thus, electric signals from muscles (EMG) directly reflect the activation of motor neurons by seizure activity. Recording EMG with surface electrodes is easy, and qualitative (visual) analysis of these, particularly in polygraphic registrations, helps characterize motor seizures.[15,16] However, until recently, quantitative analysis of ictal EMG signals received little attention. Besides gaining further insight into the pathophysiology of motor seizures, quantitative ictal EMG changes seem to represent excellent electrophysiological biomarkers of these.

### 2.1 | Muscle activation during tonic and tonic–clonic seizures differs from physiological muscle activation

Quantitative analysis of the entire duration of tonic seizures (TS) and TCS demonstrated that muscle activation was significantly different during these motor seizures compared to physiological muscle activation—maximal voluntary contraction (MVC) during seizures acted by healthy volunteers.[17] Furthermore, quantitative EMG during TS was different from the tonic phase of the TCS.[17] TS had EMG signals in higher frequency domains (expressed as higher median frequency and as increase in the relative power in the frequency domain above 100 Hz) compared to MVC and to TCS.[17] The amplitude of the EMG signals (expressed as root mean square of the signal) was significantly higher during the tonic phase of TCS, compared to TS and to MVC.[17] The EMG-EMG coherence between muscles on the left and right sides was significantly higher during motor seizures (TS and TCS) compared to the acted ones (MVC).[17]

### 2.2 | Dynamic changes of quantitative EMG during TCS

Wavelet analysis showed specific dynamic evolution (Figure 1) of the TCS, characterized by changes in the low-frequency (LF) wavelet component (2-8 Hz) and the high-frequency (HF) wavelet component (64-256 Hz).[18] These changes determined the following phases: tonic-onset, tonic-maintenance and clonic.[18]

There was a gradual amplitude increase in the tonic-onset phase (Figure 1), and both LF and HF components increased.[18] In the tonic-maintenance phase, the gradual amplitude increase continued, and there was a specific dissociation between HF and LF components; whereas the HF component increased dramatically, there was a total suppression of the LF component. A similar dissociation was not observed between HF and LF in physiological muscle activation (MVC during seizures acted by healthy volunteers) or convulsive psychogenic nonepileptic seizures (PNES): in these subjects, both wavelet components were constantly present throughout the episode, and the onset of muscle activation was not gradual.[18,19] The transition from the tonic to the clonic phase was characterized by a marked increase in the LF component (clinically corresponding to the "vibratory" movements observed in video recordings of TCS) and decrease of the HF component (Figure 1).[18]

The ratio between HF and LF components constituted a reliable quantitative parameter, both for objective determination of the seizure duration, and for the segmentation of the TCS into the phases described above (Figure 1).[18] The median duration of TCS was 85 s (73-93 s).[18]

### 2.3 | Silent periods and the clonic phase

As TCS evolved, tonic muscle activity became interrupted for longer and longer periods by suppression of the muscle activity (silent periods) characterizing the clonic phase

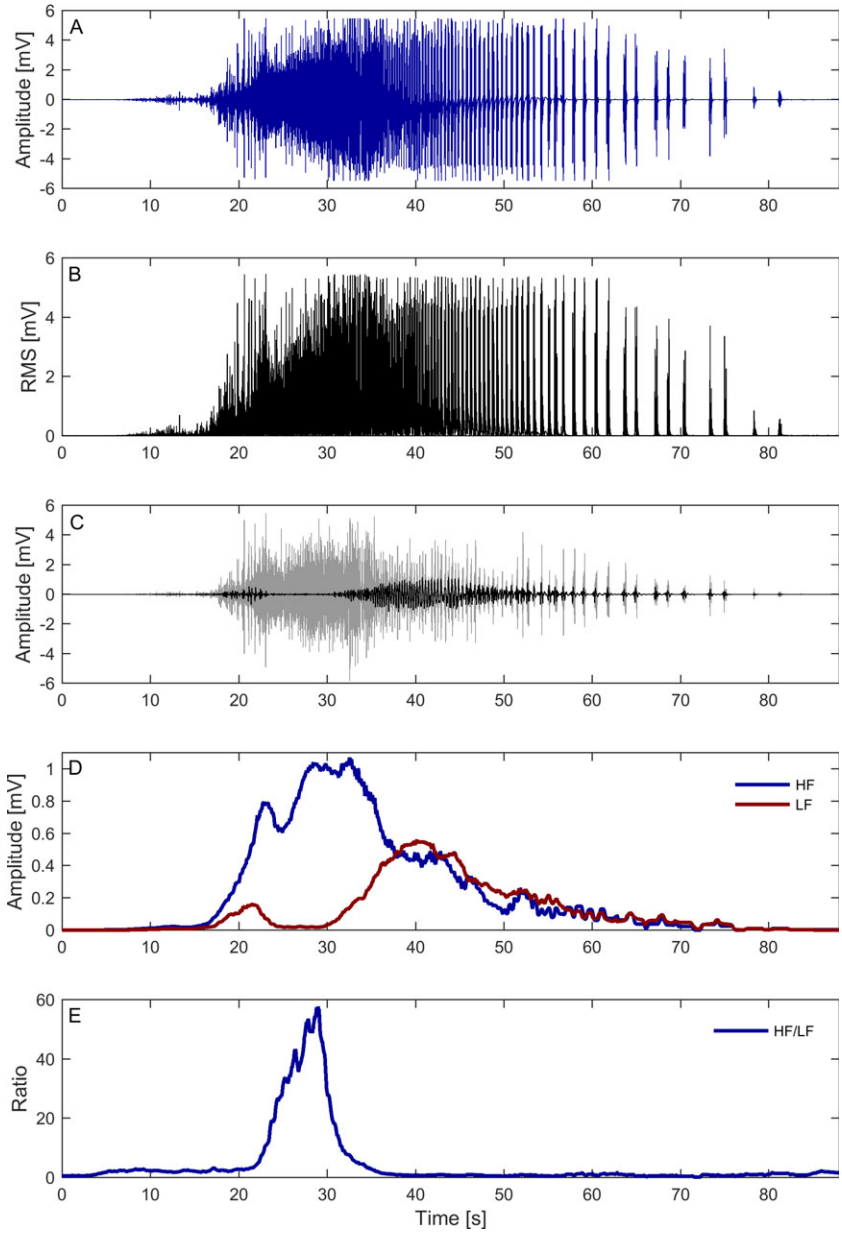


**FIGURE 1** Quantitative surface EMG of a TCS. A, EMG signal of the TCS. B, Root mean square (RMS) throughout the TCS. C, Signals corresponding to the high-frequency (black traces) and low-frequency (gray traces) of the EMG signal. D, Normalized amplitudes corresponding to the low-frequency (red) and high-frequency (blue) components. E, High-frequency/low-frequency ratio

(Figure 2).[18] The increase in duration of the silent periods was exponential (Figure 2), which further differentiated them both from acted seizures (by healthy volunteers) and from convulsive PNES.[18,19]

Duration of the clonic discharges between the silent periods was remarkably stable, around a median value of 209 msec (183-67 msec).[18] Such short contractions could not be reproduced by acting seizures in healthy volunteers: duration of the acted jerks was 1326 (817; 1749) msec. The energy of the clonic discharges peaked after the vibratory period of the clonic phase and the right-left EMG coherence also continued to increase and peaked during the clonic phase.[18] Thus, both intramuscular and intermuscular synchronization peaked during the clonic phase.[18]

## 2.4 | Electrophysiological biomarker of TCS

The specific quantitative EMG changes during the onset phase of the TCS constitute the pathophysiological background for a seizure biomarker: there is a marked increase in the HF component (Figure 1), and an increase in the amplitude of the signal.[18] However, to be implemented as an algorithm that runs real-time, the electrophysiological biomarker needs to use little computational power. By filtering EMG signals with a high-pass filter of 150 Hz (Figure 3A), the algorithm operates in the frequency domain in which seizure-specific signals occur.[20] The increase in frequency is easily monitored using the automated counting of the number of zero-crossings (per second) of the filtered EMG signal (Figure 3B).[20] To emphasize the need for an

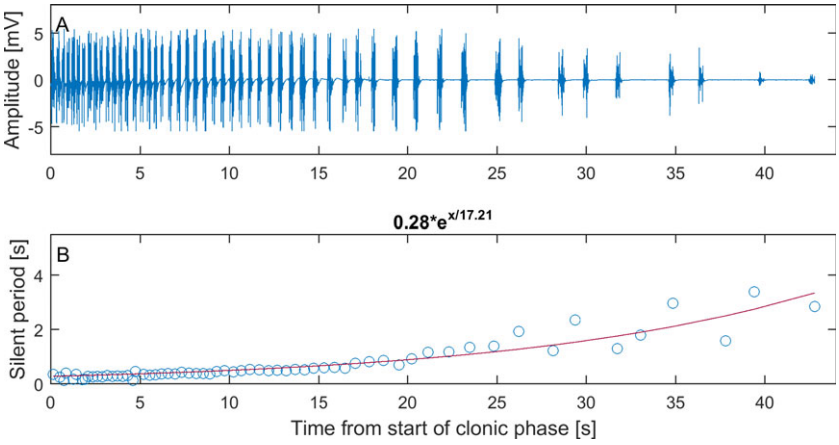


**FIGURE 2** Clonic phase of the TCS. A, EMG signal during the clonic phase of the TCS. B, The vertical axis shows the duration of the silent periods, and the horizontal axis shows the time from the start of the clonic phase. Note the exponential increase in the duration of the silent periods, as the seizure evolves

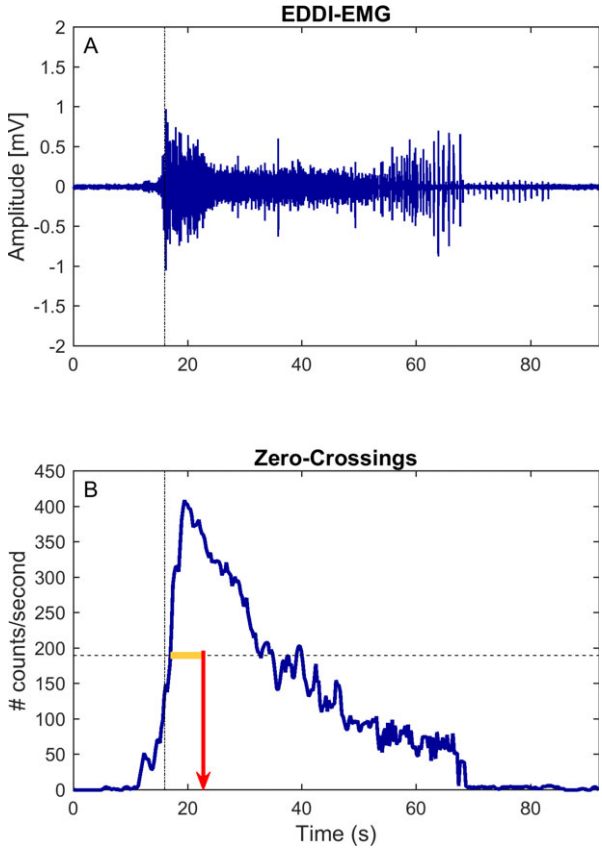


**FIGURE 3** The generic EMG-based seizure detection algorithm. A, EMG signal after high-pass filtration (150 Hz). B, Evolution of the number of zero-crossings (vertical axis) throughout the TCS; the stippled horizontal line marks the threshold for identifying the seizure activity; the yellow horizontal line depicts the minimum number of time-periods with zero-crossings exceeding the threshold, necessary for triggering seizure alarm (red arrow)

increase in amplitude, a hysteresis of ±50 μV is added to the algorithm.[20] When the number of zero-crossings / s exceeds the threshold for a predefined period of time, the seizure is identified and an alarm is triggered (Figure 3B).[20] For more details about quantitative analysis of surface EMG and electrophysiological biomarkers of convulsive seizures, the reader is referred to a recently published review.[21]

### 2.5 | Seizure severity assessed by quantitative EMG

Using the objective method, based on the HF/LF ratio for segmentation of the TCS, the tonic-maintenance phase appeared to have a remarkably constant duration (median = 11s; 8-13 s).[18] However, duration of both tonic-onset and clonic phases was highly variable, and correlated to seizure severity (duration of the TCS and frequency of their occurrence). Short tonic-onset phases were correlated with long clonic phases and with higher seizure occurrence, whereas longer tonic-onset phases were correlated with short clonic phases and low seizure occurrence.[18]

Because both in the tonic-onset phase and the start of the clonic phase there is an increase in the LF component, it is reasonable to assume that these are manifestations of intrinsic inhibitory mechanisms counteracting the development of seizures (at onset) and eventually terminating them (in the clonic phase). When inhibitory mechanisms are strong, it takes longer for the ictal processes to overcome them (long tonic-onset phase), and shorter for the inhibitory mechanisms to stop the seizure (shorter clonic phase). For weaker inhibitory mechanism it is the other way around (ie, short tonic-onset and long clonic phase).[18]

## 3 | EMG-BASED AUTOMATED DETECTION OF CONVULSIVE SEIZURES

We searched PubMed for relevant articles published since 1950. We used 2 separate search strings: (1) "(EMG OR electromyography) AND (seizure AND detection)"; (2) "(wearable OR mobile) AND (seizure AND detection),"

and did not use any language restrictions. We found 81 PubMed entries. In addition, we identified 22 articles in the reference lists. After reading the abstracts, 44 studies were discarded because they were not related to seizure detection using noninvasive wearable devices in humans. Eight articles were reviews, 5 articles were surveys on the needs and expectations of patients concerning wearable seizure detection devices, and 4 papers were on differentiating between epileptic and nonepileptic seizures. Thus, we identified 42 original research papers on wearable seizure detection devices. Detection of TCS or TS has been addressed in 12 papers, 7 using EMG alone and 5 using EMG in combination with other modalities.

A seizure-detection algorithm using the electrophysiological biomarker described earlier[20] has been optimized in a phase-2 study,[22] using conventional amplifiers in epilepsy monitoring units (EMUs).[23] Based on EMG data from 22 TCS recorded from 11 consecutive patients, using receiver-operating characteristic curve analysis, the detection-threshold (number of zero-crossings / s), and the time-period above, the threshold necessary to trigger an alarm were determined, so that the generic (not patient-tailored) algorithm achieved a sensitivity of 100%, false alarm rate (FAR) of 1/24 h, and mean detection latency of 13.7 s.[23]

Another phase-2 study[22] that retrospectively analyzed EMG signals recorded with conventional amplifiers in EMU, reported a different approach.[24] The algorithm used Hotelling's T-squared power analysis of compound muscle action potentials, and it was patient-tailored: MVC was initially measured in each patient to establish the baseline (physiologic) threshold.[24] In offline (retrospective) analysis, the algorithm detected 20 of the 21 TCS, in 11 patients (sensitivity: 95%).[24] The average detection-latency was 20 s, which was longer than for the generic algorithm. This is probably explained by the seizure phase that triggers the alarm (onset phase for the generic algorithm and early transition to clonic phase in the patient-tailored algorithm).

Both algorithms have been implemented into dedicated, wearable devices (Figure 4). The algorithm based on the number of zero-crossings was implemented into EDDI (Epileptic seizure Detector Developed by IctalCare), placed on the belly of the biceps muscle via a hypoallergenic patch that contained the EMG electrodes (Figure 4B). EDDI obtained CE (Conformité Européenne) marking registration in 2013. Initially EDDI was tested in a pilot study.[25] Successively, a modified version of the algorithm was validated in a prospective, multicenter study of real-time seizure detection, using the wearable EMG-device and predefined cutoff values for triggering seizure-alarms,[26] thus qualifying as a phase-3 trial.[22] The gold standard was identification (by trained experts) of seizures recorded during video-EEG monitoring. Seventy-one consecutive patients were recruited in 3 centers (20 patients with TCS). The sensitivity of EDDI for detecting TCS was 94% (30 of 32 TCS were detected; 95% confidence interval [CI] 86%-100%). Median seizure detection latency was 9 s (range: −4 to 48 s). The FAR was 0.67/day. No adverse events occurred, and none of the patients withdrew from the study.

The algorithm based on Hotelling's T-squared power analysis was implemented into a SPEAC (Sensing Portable sEMG Analysis Characterization system) developed by Brain Sentinel (Figure 4A). The device was granted de novo clearance by the U.S. Food and Drug Administration (FDA) in 2017. The performance of the device was tested in a large multicenter study: 37 of the recruited 199 patients (in 11 EMUs) had TCS.[27] EMG was recorded prospectively, using the dedicated, wearable device.[27] However, data analysis and seizure detection was not real-time: it was done offline (after seizure occurrence time, ie, retrospectively) from data archived at a central site.[27] There was no single, predefined cutoff value, but the performance of the algorithm was tested across a wide range of thresholds (95-255). Therefore, according to the standards proposed for seizure detection trials,[22] this study qualifies as phase-2. At the threshold setting of 145, the algorithm had a sensitivity of 76%: it detected 35 of the 46 TCS (95% CI 61%-87%). The FAR was 2.52/ 24 h.[27] The electrodes in the SPEAC system were placed perpendicular to the muscle fibers. Therefore, when the device was placed more than 45 degrees from the midline of the belly of the biceps (this was the case in 29 patients included into the study), crosstalk between biceps and triceps muscles caused a reduction in the signal amplitude (in-phase cancellation), greatly reducing sensitivity of the algorithm.[27] In the EDDI system, electrodes were placed parallel to the muscle fibers, which prevented crosstalk and in-phase cancellation.[26] In a subgroup of patients in whom the device was placed over the midline of the biceps muscle (n = 149), the sensitivity reached 100%: the device detected 29 of 29 TCS (95% CI 88%-100%) that occurred in the 24 patients with a properly placed device, with a latency of 7.7 s and FAR of 1.44/ 24 h.[27] Mild to moderate adverse events were reported in 28%, and 15% of the recruited patients withdrew from the study during EMG monitoring.[27] An important feature of the SPEAC system is that EMG data are stored for offline review by practitioners.

EMG was included into multimodal systems for detection of TCS, along with accelerometers in wearable devices,[28–31] and along with EEG and ECG signals in the EMUs.[32] Although the small-scale studies reported an improved performance in the multimodal setting as compared to the unimodal one, the sensitivity and FAR of these multimodal TCS detectors was not superior to what the 2 large-scale multicenter studies reported on the unimodal, EMG-based devices. For a detailed review on multimodal seizure detection, the reader is referred to another paper of this supplement of *Epilepsia*.[33]

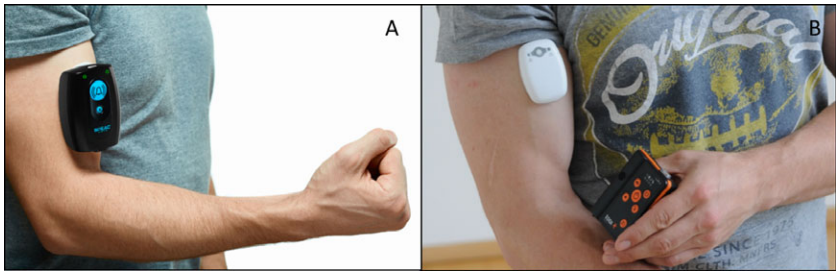


**FIGURE 4** Wearable seizure detection devices based on surface EMG. A, SPEAC; B, EDDI

We have found 2 patents corresponding to the commercially available EMG-based seizure-detection devices.[34,35]

Our PubMed search identified 4 prospective studies on clinical validation of accelerometry-based, wearable devices using real-time detection of TCS.[36–39] Sensitivity and FAR of these devices were inferior or similar to the EMG-based ones (see the detailed review on accelerometry-based seizure detection devices in this supplement of *Epilepsia*[40]). Compared with EMG-based detection, detection latencies of these devices are longer, since alarms are triggered during the vibratory or the clonic phase. However, they are easy to use because they do not require self-adhesive electrode patches.

Detection of TS using surface EMG proved to be less successful than detection of the TCS. TS have significant intra- and intersubject variability. Hence, a generic algorithm was not feasible.[41] With patient-specific algorithms, complete seizure detection was achieved in a retrospective analysis of EMG signals, in a small number of patients, yet with a much higher FAR (between 0.08 and 7.9).[41] Detection of TS is challenging due to the low amplitude, which makes TS similar to the patterns given by high-frequency noise (induction artifacts). The other challenge encountered in this study was physiologic muscle activation at a smaller intensity than MVC. This submaximal muscle activation had higher frequency content than MVC, bringing it closer to the features of TS.[41]

In addition to seizure detection, EMG can be used for differentiating epileptic from nonepileptic (psychogenic) convulsive seizures. Inspection of the quantitative EMG features specific for TCS (temporal dynamics of the HF/LF ratio and the evolution of the silent periods) accurately differentiated between epileptic and nonepileptic convulsive seizures.[19] Using the EMG-based biomarker of TCS, an automated algorithm differentiated between TCS and convulsive PNES with an accuracy of 95%.[42]

## 4 | CONCLUSION

Specific, quantitative EMG changes characterize TCS. Besides giving insight into the dynamics of TCS, these quantitative changes constitute an excellent basis of electrophysiological biomarkers for TCS. Two large-scale, multicenter, prospective trials on 2 different wearable seizure-detection devices demonstrated that EMG-based algorithms detect TCS with a sensitivity of 94% and FAR of 0.7/24 h (EDDI) and, respectively, a sensitivity of 76% and FAR of 2.5/24 h (SPEAC). In the subgroup of patients with optimized placement of EMG electrodes, sensitivity achieved 100%, with a FAR of 1.4/24 h (SPEAC). These studies provide robust evidence on the accuracy of EMG-based seizure detection using wearable devices. However, the FAR in a subgroup of patients is still higher than acceptable, and this needs further improvement. Besides seizure detection, the algorithms can differentiate between TCS and nonepileptic convulsive episodes. For the detection of tonic seizures, the EMG-based approach appears at present to be less robust. The major limitation of EMG-based seizure detection is that this approach detects only convulsive seizures. A comprehensive seizure detection system for clinical use will need additional components.

## DISCLOSURE OF CONFLICTS OF INTEREST

Dr. Conradsen was an employee and is currently consultant for IctalCare. The remaining authors have no conflicts of interest to disclose. 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.

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