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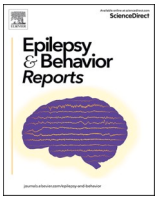

Safety and feasibility of exhaustive exercise testing for people with epilepsy☆

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ABSTRACT

People with epilepsy (PWE) are encouraged to participate in exercise and be physically active, but some PWE may report exercise-associated seizures (EAS). However, there is a lack of objective tools to inform individual recommendations for physical activity and exercise participation in PWE. This study investigated the feasibility and safety of exhaustive exercise testing in PWE. 29 patients underwent an objectively and subjectively exhaustive exercise test on a bicycle ergometer and resting state EEG was obtained before and after exercise. One patient with a history of EAS experienced a seizure immediately after exercising. In patients without EAS, an asymptomatic subclinical electrographic seizure was observed in one patient, and two patients revealed interictal epileptiform discharges only after exercise. All EEG changes occurred in the setting of non-REM sleep, while the respective pre-exercise EEG recordings revealed less sleep. No seizures or significant EEG changes after exercise were observed in any other patient. EEG investigations before and after exhaustive exercise were feasible in PWE, but safety protocols need to be established, especially in patients with EAS. Investigation of a higher number of PWE with and without EAS with repeat exercise-associated EEG may provide information about the clinical utility of exercise-associated EEGs when counseling PWE.

1. Introduction

Exercise-associated seizures (EAS) are rare in people with epilepsy (PWE). In prior studies, only 2 % experienced genuine EAS, defined as seizures occurring in more than 50 % of the exercise sessions [1]. Mechanisms potentially contributing to EAS may include lactic acidosis, hyperventilation, hyponatremia, hyperthermia, fatigue, stress due to competition or each combination thereof [2–5]. Studies investigating (positive or negative) effects of chronic or acute exercise in PWE often lack standardization of type, intensity, and duration of exercise. The clinical heterogeneity of epilepsy syndromes may also contribute to inaccuracies in the interpretation of those results. Previous investigations analyzing the effects of acute exercise excluded potentially significant influencing factors such as exercise-induced exhaustion and effects of sleep during prolonged recovery in the post exercise setting [6–9]. Currently, counseling PWE on sports and exercise activities relies solely on the clinical history of the patient. The lack of clinical predictors of EAS often leaves both clinicians and patients in doubt about whether and how PWE may engage in sports and exercise, although it is widely accepted that exercise provides positive effects and that PWE should generally be encouraged to be active [4]. Some authors even argue that exercise may even have the potential to reduce the risk for sudden unexpected death in epilepsy (SUDEP). Seizure frequency and cardiovascular health are known risk factors for SUDEP [10], which might be improved by regular sports and exercise.

Implementing standardized exercise tests in conjunction with electroencephalography (EEG) may be helpful in this clinical context. Although exercise-associated EEG has previously been investigated [8,9,11], there is still a lack of knowledge regarding the impact of acute exercise on epilepsy symptoms, EEG changes, and occurrence of EAS. Although a standardized application of exercise and (electro-)physiological measurements have been found to deliver clinically significant information and is already successfully integrated into patient care of other medical specialties (e.g., cardiology as exercise-associated electrocardiography (ECG) [12,13]), there is still no standardized protocol for exercise-associated EEG recordings in PWE despite the availability of portable, affordable, and relatively easy to use EEG systems [14]. Because of the high predisposition to movement-associated artifacts,

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EEG recording may only be feasible before and after an acute bout of exercise. EEG recordings in relation to exercise in normal controls have revealed that exercise-induced oscillatory brain changes persist even after exercise [7,15], suggesting that post-exercise EEG recordings may also be helpful in the management of PWE with EAS.

PWE often fear exercise-induced seizures and injuries [16,17], indicating the need for objective biomarkers to guide the management of PWE with or without a known history of EAS. The goal of this study is to assess the safety and feasibility of a standardized exercise protocol that leads to complete exhaustion for PWE and provide the basis for further standardized evaluations.

2. Methods

The study was conducted in accordance with the Declaration of Helsinki and approved by the ethics committee of the Westfalian Medical Board. The current analysis follows a previously published study protocol [18]. In brief, informed consent was obtained from the subjects. Participants were recruited through pharmacies, physicians, local hospitals, and direct communication during medical consultations in clinic. All types of epilepsy were included. PWE were excluded, if they had physical impairments that would prevent them from undergoing an exhaustive exercise test (n = 0).

Medical history was obtained by questionnaire and a clinical interview. Information on seizure frequency and experience of exercise-induced seizures was obtained in relation to the day of the delivery of the exercise test. Resting-state 128-channel EEG according to the International 10–20 System was obtained in a supine position in a darkened room. Data from 24 electrodes (actiCHamp, Brain Products GmbH) was clinically interpreted for this investigation. The recording lasted 10 min before exercise and 30 min after exercise. FPz was used as the ground electrode, and FCz was used as a reference electrode. Impedance was kept below 25 kΩ. Data were sampled at 1000 Hz, but for clinical analysis, the data were downsampled to 250 Hz and filtered using a Zero Phase Shift Butterworth Filter with a low cutoff of 1 Hz (time constant [s]: 0.1591549, Order 4) and a high cutoff of 60 Hz (Order 4), with a notch filter (50 Hz). Resting EEG was analyzed by a board-certified epileptologist (CR). Seizures or interictal epileptiform discharges (IED) are described for the 10 min before exercise and the 30 min after exercise. The EEG findings were analyzed in three 10-minute blocks after exercise to assess changes over time. For each of the four presented time points (pre-exercise: 0–10 min, post-exercise: block 1: 0–10 min, block 2: 11–20 min, block 3: 21–30 min), sleep stages were determined according to the manual of the American Academy of Sleep Medicine (wakefulness (W), Non-REM1 (N1), Non-REM2 (N2), Non-REM3 (N3), Rem (R)) [16]. Scoring was based on EEG parameters only, and the most frequently scored sleep stage was assigned to the respective 10 min block of recoding.

The incremental exercise test on the bicycle ergometer was conducted until subjective exhaustion (maximum rate of perceived exhaustion scale). Objective exhaustion was assessed retrospectively based on ventilatory, metabolic, cardiovascular, and performance criteria. The exercise protocol started with a warm-up of 24 Watt (W) for 2 min, followed by an increment of 12 W per minute until exhaustion. The protocol ended with a cool down at 24 W for 2 min. Participants were monitored using ECG, blood pressure, and spirometry. Cardiorespiratory fitness was assessed by relative VO_2peak (ml/kg*min) and classified as very low, low, fair, good, or excellent, considering age and sex [19]. Three participants with higher baseline fitness levels (athletes) performed another exercise protocol (#26: warm-up 50 W, increment 50 W/ 3 min, cool down 50 W; #28a: ramp protocol starting at 50 W, increments of 20 W/ 1 min, cool down 50 W; #29: warm-up 100 W, increments of 50 W/ 3 min, cool down 100 W).

3. Results

Twenty-nine PWE (female n = 14, male n = 15) were included; 30 examinations are reported. One patient (#28a, #28b) performed two tests ten months apart because he was not fully cleared for bike training after the first examination. All characteristics are presented in Table 1. Participants were 37.16 ± 12.95 years old with a disease duration of 17.60 ± 18.56 years (mean/standard deviation). Cardiorespiratory fitness, assessed by the relative VO_2peak, was 34.00 ± 12.40 ml/kg*min (mean ± standard deviation). Fitness levels were classified as low in 15 patients, fair in four, good in four, and excellent in seven patients.

Nineteen PWE were seizure-free for more than six months at the time of measurement. One patient had three to four seizures in the last six months, five patients had one to two seizures in the previous six months, one patient had one seizure per month, one patient had one seizure per week, another patient had one to two seizures per week, and one patient had more than one seizure per day. Except for two patients (#28, #29), none of the patients had experienced EAS before the exercise test.

For four patients, the seizure type was unknown. Nineteen patients suffered from generalized seizures (generalized n = 1, generalized motor n = 15, generalized non-motor n = 3). Five patients had focal seizures with impaired awareness, and three patients with non-impaired awareness. Another patient suffered from focal to bilateral tonic-clonic seizures. One patient reported visual auras.

One patient was not taking anti-seizure medications (ASM). Twenty-one patients were on monotherapy, six patients were on two ASDs, and one patient was on three ASDs.

In one patient, a clinical seizure occurred immediately after the exercise test (#28b) while he was still on the bike. In this patient, an EEG after exercise could not be recorded. This was one of two patients (#28, #29) who had reported EAS prior to the investigation. Another patient had a subclinical seizure during block 1 of the post-exercise EEG (#27). This patient had a seizure frequency of 2 seizures per week at baseline and did not reach subjective (but objective) exhaustion.

No patient experienced a clinical seizure during the exhaustive bicycle exercise. Two patients (#14, #25) with generalized epilepsies revealed IEDs only after exercise but not before. For patient #14, one generalized 3–4/sec spike-and-wave paroxysm was detected in block 1 after exercise in sleep stage N1, two in block 2 in sleep stage N2, and one in block 3, also in sleep stage N2. Patient #25 showed no IEDs in block 1 (sleep stage N1), but two bursts of generalized spikes with left frontal maximum in block 2 (sleep stage N1) and eleven bursts of generalized spikes with left frontal maximum and ten left frontal spikes in block 3 (sleep stage N2) after exercise. Abnormal results in eleven other patients did not differ between the EEGs pre- and post-exercise (Table 1). No significant EEG changes after exercise were observed in any other patient.

N2 sleep appeared in six patients (#2, #4, #5, #6, #13, #26, #28b) before exercise. In ten patients, N2 sleep appeared during varying post-exercise blocks (patient number (block number)): #2 (1), #10 (1, 2, 3), #13 (2, 3), #14 (2, 3), #17 (2), #25 (3), #26 (3), #27 (1), #28a (2, 3), #29 (2, 3)).

4. Discussion

In a cohort of community-acquired PWE with mixed epilepsy syndromes, two patients experienced seizures (one clinical and one subclinical) after an exhaustive exercise test. Two other patients exhibited post-exercise changes in interictal epileptiform discharges and deeper sleep stages compared to pre-exercise. No seizures or EEG changes were observed after exercise in the remaining 26 patients.

This study demonstrates the feasibility of an exhaustive exercise test in a cohort of PWE who suffer from different seizure types of varying frequencies. Two previous studies on patients with temporal lobe epilepsy and juvenile myoclonic epilepsy reported no seizures during or after an exercise test. The cohorts examined included patients with both

Table 1
Patient characteristics and EEG findings.

Patient age [yrs] / sex	Disease duration [yrs]	Seizure type	Seizure frequency	ASM (dose [mg])	Clinical EEG pre-exercise (sleep stage)	Clinical EEG post-exercise block 1 (sleep stage)	Clinical EEG post-exercise block 2 (sleep stage)	Clinical EEG post-exercise block 3 (sleep stage)	Exercise-induced seizures before	Rel. VO2peak [ml/kg*min], classification [19]
#1 33/ m	5	unknown	seizure free	lamotrigine (200)	normal (N1)	normal (N1)	n.r.	n.r.	no	32.55 low
#2 23/ f	6	gen. motor	seizure free	lamotrigine (300)	normal (N2)	normal (N2)	normal (N1)	normal (N1)	no	20.17 low
#3 45/ m	40	gen. non-motor	seizure free	lamotrigine (175) valproic acid (1500)	normal (N1)	normal (N1)	normal (N1)	normal (N1)	no	32.24 low
#4 35/ m	3	gen. motor	seizure free	lamotrigine (150)	normal (N2)	normal (N1)	normal (N1)	normal (N1)	no	40.85 fair
#5 24 /f	8	gen. motor	seizure free	levetiracetam (1500)	2x gen. 3/sec spike-wave complexes (N2)	1x gen. 3/sec spike-wave complexes (N2)	n.r.	n.r.	no	45.42 excellent
#6 33/ m	4	gen. motor	seizure free	lamotrigine (350)	normal (N2)	normal (N1)	normal (N1)	normal (N1)	no	43.25 fair
#7 48/ f	35	gen. motor	1 seizure per week	lamotrigine (400)	normal (N1)	normal (N1)	normal (N1)	normal (N1)	no	25.99 fair
#8 55/ f	55	gen. motor gen. non-motor	seizure free	valproic acid (2000) ethosuximide (250)	normal (N1)	normal (N1)	normal (N1)	normal (N1)	no	17.05 low
#9 41/ m	40	unknown	seizure free	valproic acid (1000)	normal (N1)	normal (N1)	normal (N1)	normal (N1)	no	33.57 low
#10 37/ f	8	unknown	seizure free	lamotrigine (100)	normal (N1)	normal (N2)	normal (N2)	normal (N2)	no	25.84 low
#11 48/ f	2	FIA	seizure free	levetiracetam (1500)	normal (N1)	normal (N1)	normal (N1)	normal (N1)	no	20.93 low
#12 32/ m	6	gen. motor	seizure free	levetiracetam (1500)	abundant spikes and sharp waves centro-parieto-occipital right (N1)	abundant spikes and sharp waves centro-parieto-occipital right (N1)	abundant spikes and sharp waves centro-parieto-occipital right (N1)	abundant spikes and sharp waves centro-parieto-occipital right (N1)	no	29.81 low
#13 35/ m	2	gen. motor	1–2 seizures in the last 6 months	no medication	2x gen. 4–5/ sec spike-and-waves gen. (N2)	normal (N1)	2 x generalised polyspike complexes (N2)	2 x generalised polyspike complexes (N2)	no	40.25 fair
#14 24/ m	3	gen. motor	1 seizure per month	valproic acid (600)	normal (N1)	1x gen. 3–4/sec spike-and-waves gen. (N1)	2x gen. 3–4/ sec spike-and-waves gen. (N2)	1x gen. 3–4/ sec spike-and-waves gen. (N2)	no	49.48 good
#15 59/ m	7	gen. motor	seizure free	valproic acid (1500)	normal (N1)	normal (N1)	normal (N1)	normal (N1)	no	22.11 low
#16 20/ f	1	gen.	seizure free	levetiracetam (3000)	4x generalized 4–5/sec spike-and-waves (N1)	7x gen. 4–5/sec gen. spike-and-waves (N1)	6x gen. 4–5/ sec gen. spike-and-waves (N1)	4x gen. 4–5/ sec gen. spike-and-waves (N1)	no	39.70 good
#17 49/ m	48	gen. motor	seizure free	valproic acid (1000) carbamazepine (400)	normal (N1)	normal (N1)	normal (N2)	normal (N1)	no	39.54 good
#18 49/ f	40	FIA	seizure free	carbamazepine (600)	mild (l > r) bifrontal slowing and mild left temporal slowing (N1)	mild (l > r) bifrontal slowing and mild left temporal slowing (N1)	mild (l > r) bifrontal slowing and mild left temporal slowing (N1)	mild (l > r) bifrontal slowing and mild left temporal slowing (N1)	no	34.01 excellent

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Table 1 (*continued*)

Patient age [yrs] / sex	Disease duration [yrs]	Seizure type	Seizure frequency	ASM (dose [mg])	Clinical EEG pre-exercise (sleep stage)	Clinical EEG post-exercise block 1 (sleep stage)	Clinical EEG post-exercise block 2 (sleep stage)	Clinical EEG post-exercise block 3 (sleep stage)	Exercise-induced seizures before	Rel. VO2peak [ml/kg*min] , classification [19]
#19 26/ m	5	FIA focal aware	>1 seizure per day	lacosamide (600)	mild focal slowing and breach rhythm left temporal (N1)	mild focal slowing and breach rhythm left temporal (N1)	mild focal slowing and breach rhythm left temporal (N1)	mild focal slowing and breach rhythm left temporal (N1)	no	34.14 low
#20 27/ f	3	focal aware visual aura	1–2 seizures in the last 6 months	levetiracetam (3000) lamotrigine (150)	mild to moderate left parieto-occipital focal slowing (N1)	mild to moderate left parieto-occipital focal slowing (N1)	mild to moderate left parieto-occipital focal slowing (N1)	mild to moderate left parieto-occipital focal slowing (N1)	no	43.22 excellent
#21 55/ f	1.5	FIA	seizure free	lamotrigine (300)	normal (N1)	normal (N1)	normal (N1)	normal (N1)	no	31.81 excellent
#22 33/ f	11	un-known	seizure free	lamotrigine (200)	normal (N1)	normal (N1)	normal (N1)	normal (N1)	no	27.41 low
#23 24/ f	9	gen. non-motor	seizure free	lamotrigine (350)	normal (N1)	normal (N1)	normal (N1)	normal (N1)	no	24.59 low
#24 56/ f	52	gen. motor focal aware	seizure free	lamotrigine (400) carbamazepine (800)	normal (N1)	normal (N1)	normal (N1)	normal (N1)	no	17.50 low
#25 50/ m	25	gen. motor	1–2 seizures in the last 6 months	valproic acid (2750) lamotrigine (300) lacosamide (200)	normal (N1)	normal (N1)	2 bursts of generalized spike and spike complexes with left frontal maximum (N1)	11 bursts of generalized spike and spike complexes with left frontal maximum and 10 left frontal spikes (N2)	no	25.31 low
#26 14/ m	0.75	FIA	1–2 seizures in the last 6 months	levetiracetam (1500) lamotrigine (150)	moderate, at times rhythmic left temporal slowing, TIRDA, left anterior to mid-temporal spikes (N2)	mild left temporal slowing (N1)	mild left temporal slowing, 1 left anterior to mid-temporal spike (N1)	moderate, at times rhythmic left temporal slowing, TIRDA, left anterior to mid-temporal spikes (N2)	no	49.39 good
#27 60/ f	56	gen. motor	1-2x/week (myoclonic)	phenytoin (250)	mild to moderate left temporal slowing, 1 left temporal spike (N1)	mild to moderate left temporal slowing, 2 left temporal spikes, 1x 2–3/sec gen. spike and wave for 6 sec, one 70 sec subclinical seizure with bilateral onset out of stage 2 NREM sleep (no symptoms) (N2)	mild to moderate left temporal slowing, 3x 2–3/sec gen. spike and wave paroxysms (2, 5 & 10 sec) (N2)	mild to moderate left temporal slowing, 2 left temporal spikes (N1)	no	15.43 low
#28a 30/ m	17	FIA with bilateral tonic clonic	1–2 seizures in the last 6 months	lamotrigine (400)	mild bilateral theta and delta slowing with anterior pre-dominance (N1)	normal (N1)	normal (N2)	mild bilateral theta and delta slowing with anterior pre-dominance (N2)	yes	53.56 excellent
#28b 31/ m	18			lamotrigine (200)	3 isolated spike and wave complexes (N2)	n.r.	n.r.	n.r.	yes	53.73 excellent

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Table 1 (*continued*)

Patient age [yrs] / sex	Disease duration [yrs]	Seizure type	Seizure frequency	ASM (dose [mg])	Clinical EEG pre-exercise (sleep stage)	Clinical EEG post-exercise block 1 (sleep stage)	Clinical EEG post-exercise block 2 (sleep stage)	Clinical EEG post-exercise block 3 (sleep stage)	Exercise-induced seizures before	Rel. VO2peak [ml/kg*min], classification [19]
#29 19/m	2 months	gen. motor	3–4 seizures in the last 6 months	levetiracetam (2000)	mild left fronto-temporal slowing (N1)	mild left fronto-temporal slowing (N1)	mild left fronto-temporal slowing (N2)	mild left fronto-temporal slowing (N2)	yes	71.16 excellent

yrs = years, m = male, f = female, n.r. = not recorded, N1: sleep stage NREM1, N2: sleep stage NREM2, ASM: anti-seizure medication.

controlled and uncontrolled seizures [8,9]. Fialho et al. (2017) reported a seizure in one patient 12 min after an exhaustive exercise test on a treadmill [6] (similar to the case of this study, where the seizure occurred immediately after the cool-down period of the exercise test). Notably, the patient population in the literature and our study was not restricted to patients with EAS. Although EAS have been described in the literature [20–23], they appear to be rare and specific pathomechanisms are largely unknown. Our patient who experienced a seizure immediately after the exercise test (#28) had previously experienced two seizures during a semi-professional cycling race but was one of two subjects reporting EAS. The results of this patient, who is the only patient investigated twice, revealed that exercise associated EEG changes may vary longitudinally, similar to routine EEG investigations. In the other patient, a subclinical seizure in block 1 of the post-exercise EEG (#27) was associated with stage N2 sleep that also appeared during block 1 only. It is, therefore, unclear whether exercise, sleep, or the normal course of epilepsy was the cause of this subclinical seizure. The history of subclinical seizures in this patient prior to our investigation was unknown. Further IEDs detected in the measurements before and after exercise (blocks 2, 3) in this patient appeared during sleep stage N1.

Another study described reduced IEDs during and after the exercise testing compared to a resting measurement [7]. In our study, two patients showed an increase in IED frequency after exercise as compared to the pre-exercise EEG. Although both patients exhibited deeper sleep stages post-exercise, the relatively low number of patients may not justify any systematic conclusion about the impact of post-exercise sleep on IED frequency. Single exercise sessions are known to decrease sleep latency and impact other characteristics of sleep [24], so sleep stages need to be considered when interpreting post-exercise EEGs (and whether exercise-associated fatigue may be relevant in EAS). However, exercise-associated exhaustion itself may increase the probability of detecting IEDs. The mechanisms of exercise-associated IEDs and seizures are still unknown, factors like lactic acidosis, hyperventilation, hyponatremia, hyperthermia, fatigue, and stress are discussed [2,4,5]. Seizure triggers in general have been described in some patients. Balamurugan et al. (2013) [25] report that in 405 patients with active seizures, at least one trigger factor in 89 % of patients. Although this study did not include exercise as a trigger factor, it may be hypothesized that exercise may trigger seizures in some patients but not in others. The underlying reasons determining who might be affected by EAS are still unknown.

As the only patient in our study who presented with EAS developed a seizure after one of two exercise test sessions, obtaining a detailed seizure history as suggested by the position paper of the ILAE on exercise and epilepsy [26] and implementing a safety protocol are of utmost importance. Similar to a safety protocol in epilepsy monitoring units, algorithms and adaptations of the infrastructure should be implemented to minimize possible seizure-related injuries.

This study employed an exercise test until exhaustion, because complete exhaustion may be the strongest trigger factor for EAS and allows reasonable standardization across subjects. Objective exhaustion was assessed using ECG, blood pressure, and spirometry; subjective exhaustion was measured using the perceived rate of exertion (Borg) scale. In our cohort, patients revealed low to excellent cardiorespiratory fitness at baseline. It is known that load parameters such as VO_2 peak (or VO_2 max) are higher in treadmill than bicycle exercise [27]; therefore the comparison to other studies using a treadmill to determine cardiorespiratory fitness may be limited. However, a bicycle ergometer was used in our study because PWE often have lower physical activity levels [4] and may be less experienced with running on a treadmill until exhaustion. Study participants could familiarize themselves more easily with a bicycle ergometer to decrease the risk of falling and injuries. A flat-step protocol (1-minute steps) was chosen for most subjects since patients often do not tolerate well any high and abrupt increases of the load [28]. In addition, using one-minute steps with smaller increments of the load has the advantage of bringing the participant closer to exhaustion compared to longer stages with bigger increments. According to the American Heart Association, an exercise test on a bicycle ergometer should begin at 10 to 25 W [28]. The warm-up and cool-down load doubled the increase of the load for this test. The chosen stepwise increments of 12 W per minute in PWE in general was based on the study by de Lima et al. (2011) [9], although Allendorfer et al. (2019) had also published a bicycle ergometer test and successfully employed increments of 25 W every 2 min in PWE [29]. However, three of our participants performed a different exercise protocol (#26, #28, #29) due to higher fitness baseline levels. These participants were semi-professional or professional athletes (#26: swimming, #28: cycling, #29: cycling). Using the same protocol would have taken considerably more time and adaptation to the exercise load would have been different. We therefore believe that although a standardized exercise protocol that may be feasible for most patients, some patients require adjustments to achieve complete exhaustion physiologically.

5. Conclusion

Exercise testing on a bicycle ergometer that leads to complete exhaustion is safe and feasible in PWE with mixed epilepsy syndromes, seizure types, seizure frequencies, and different cardiorespiratory fitness levels. To determine the clinical utility of this test in the context of risk assessment for physical activities and exercise in PWE, patients with and without EAS should be repeatedly investigated. Before conducting an exhaustive exercise test, it is essential to obtain the clinical history, particularly with respect to seizure triggers and EAS, and assess the fitness levels to implement appropriate safety protocols and adjust the exercise protocol.

Ethical statement

The study was conducted in accordance with the Declaration of Helsinki and approved by the ethics committee of the Westfalian Medical Board. All authors confirm that the ethical standards of the Journal 'Epilepsy & Behavior Reports' are met.

Declaration of Generative AI and AI-assisted technologies in the writing process

During the preparation of this work the authors did not use any AI or AI-assisted technologies.

CRediT authorship contribution statement

Franziska van den Bongard: Writing – review & editing, Writing – original draft, Software, Project administration, Methodology, Investigation, Formal analysis, Data curation. **Catharina Petersen:** Writing – review & editing, Writing – original draft, Software, Project administration. **Claus Reinsberger:** Writing – review & editing, Writing – original draft, Validation, Supervision, Resources, Project administration, Methodology, Investigation, Funding acquisition, Formal analysis.

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Declaration of competing interest

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