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Impact of EEG neurofeedback training on EEG and sEMG signals after high-intensity exercise

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Abstract: Purpose: From the perspectives of electroencephalography (EEG) and surface electromyography (sEMG), this study explores the effects of EEG neurofeedback training (EEG-NF) on acute fatigue induced by blood flow restriction (BFR) intervention, providing new ideas and methods for the elimination of sports fatigue. We hypothesized that EEG-NF could effectively promote the recovery of EEG and sEMG signals after BFR intervention and improve sports performance. Methods: Thirty-four college students majoring in sports were selected as subjects and randomly divided into an experimental group ($n = 17$) and a control group ($n = 17$). BFR intervention was used to induce acute sports fatigue. The control group underwent 33 min of natural recovery after fatigue onset, whereas the experimental group underwent 33 min of EEG-NF. EEG signals, sEMG signals, heart rate (HR), blood oxygen saturation (SpO_2), blood lactate (LA), Borg Rating of Perceived Exertion (RPE), maximum thigh strength, maximum lower-leg strength, vertical jump height, and standing long jump distance were measured at three time points: before the experiment, immediately after exercise, and after the intervention. Results: After the intervention, the levels of LA and RPE, as well as β waves in the central and occipital regions and θ waves in the central and parietal regions, were significantly lower in the experimental group than in the control group ($P < 0.05$). The α waves in the central, parietal, and occipital regions and the MF and MPF of the vastus medialis, rectus femoris, and gastrocnemius externus muscles were significantly higher in the experimental group than in the control group ($P < 0.05$). The maximum thigh strength, vertical jump height, and standing long jump distance were significantly higher in the experimental group than in the control group ($P < 0.05$). Conclusion: For acute

fatigue induced by BFR intervention, EEG-NF can enhance the regulatory function of the central nervous system (CNS) and promote the elimination of central fatigue. At the same time, EEG-NF can also promote the elimination of peripheral muscle fatigue. Based on the synchronous changes in EEG and sEMG signal parameters, it is speculated that EEG-NF may improve the elimination of peripheral muscle fatigue by promoting the regulatory function of the CNS, thereby improving sports performance.

Keywords: Blood flow restriction training; Acute exercise-induced fatigue; EEG neurofeedback; Electroencephalogram signals; Surface electromyography signal

1. Preface

Exercise-induced fatigue is a phenomenon characterized by a temporary decline in exercise capacity and physical function caused by exercise [1]. Exercise-induced fatigue can be divided into two forms: central fatigue and peripheral fatigue. Compared with peripheral fatigue, central fatigue occurs more commonly during exercise training. Even when the exercise load does not cause athletes to experience obvious physiological fatigue, they may still experience increased energy consumption due to prolonged excitation or high concentration of cerebral cortical neurons, leading to a central protective inhibition process, that is, central fatigue [2]. When athletes experience obvious physiological fatigue, changes in the internal environment of the body may further impair central nervous system (CNS) function through pathways such as an imbalance in the ratio of 5-hydroxytryptamine (5-HT) to dopamine (DA) in the brain, accumulation of lactic acid (LA) in the brain, and increased brain blood ammonia concentration [3,4], thereby exacerbating central fatigue. In addition, central fatigue not only reduces CNS function but also aggravates peripheral muscle fatigue through the coupling relationship between neuromuscular functions. Therefore, compared with peripheral muscle fatigue, central fatigue has a greater impact on and poses greater harm to athletes. Nutritional and energy supplementation, hyperbaric oxygen therapy, and transcranial electrical stimulation are currently common intervention methods for central fatigue. Although these methods can promote the elimination of central fatigue to a certain extent, they also have drawbacks, such as potential doping risks, long duration, poor specificity, and

low athlete compliance, making them difficult to widely apply in sports training.

Electroencephalography-neurofeedback training (EEG-NF) is a method that collects cerebral cortical EEG signals through electrodes, analyzes and processes them using a computer, and then converts the EEG signals into intuitive visual and auditory information [5]. Its principle is to enable self-regulation and training based on feedback information, thereby changing EEG activity patterns and regulating brain function to achieve targeted improvements in CNS regulatory ability. Studies have indicated that EEG-NF can significantly improve central fatigue in athletes, reduce inhibitory neurotransmitters in the brain, enhance reaction speed, and improve positive emotional states [6]. Additionally, compared with traditional fatigue elimination methods, EEG-NF has advantages such as high safety, no risk of doping, short duration, and non-invasiveness, with high athlete compliance, making it suitable for promotion and application in sports training.

However, due to limitations related to technical equipment, coaches, and athletes' cognitive aspects, relatively few studies have investigated the application of EEG-NF in sports training, and its intervention effects on central fatigue require further research and confirmation. In addition, due to the coupling relationship between neural and muscular functions, there is still a lack of research evidence regarding whether EEG-NF can promote the recovery of skeletal muscle function by regulating CNS function. Surface electromyography (sEMG) signals are an important means of monitoring skeletal muscle fatigue characteristics in athletes during sports training and are widely used. Some studies have indicated that, by constructing a fusion model using the time-domain signals of EEG and sEMG as node features, the classification accuracy of the fusion model after exercise-induced fatigue is higher than that of models based on a single signal, suggesting that EEG and sEMG signals provide complementary information and can accurately reflect the body's exercise state [7]. In view of this, this study uses EEG and sEMG as indicators for monitoring fatigue in athletes and investigates the intervention effects of EEG-NF on exercise-induced fatigue from the perspectives of central fatigue (EEG) and peripheral fatigue (sEMG), thereby providing new ideas and methods for promoting fatigue elimination in

athletes during sports training.

2. Research Subjects and Methods

2.1 Research Subject

Subjects were recruited for this study through the official WeChat account of the Center for Sports Science at Hubei Minzu University, campus posters, and on-site registration. The inclusion criteria were as follows: (1) male college students majoring in sports who had participated in systematic sports training during the previous six months (to avoid the influence of hormones, menstrual cycles, etc., on the test results); (2) no physical or mental illness; (3) not currently taking any medications or receiving treatment for any disease; and (4) willingness to participate after understanding the research procedures, purpose, test indicators, and application of the research results. After screening, participants underwent electrocardiography, cardiovascular function, sports injury, and other examinations at the Affiliated Hospital of Hubei Minzu University (Grade A, Class III). Participants were excluded based on the results of these examinations. The exclusion criteria were as follows: (1) persistent unstable angina pectoris or other cardiovascular diseases, skin diseases, neurological sensory disorders, etc. [8]; (2) severe sports injuries or a history of surgery within the previous 3 years; and (3) any other condition that did not meet the eligibility criteria or was considered unsuitable for participation in high-intensity sports training.

Prior sample size estimation was conducted using G*Power 3.1 with a single-factor repeated-measures design and between-within-group interaction. The effect size (f) was set to 0.25, the significance level (α) to 0.05, and the statistical power ($1-\beta$) to 0.80, resulting in a minimum required sample size of 28 participants. To account for potential sample loss due to exercise-related injuries, acute exercise-induced fatigue, and other factors, the sample size was increased. A total of 36 eligible participants were recruited using a paired random grouping method. First, the included participants or subjects were paired based on age, skeletal muscle content, and other characteristics. The paired subjects were then randomly assigned to either the natural recovery group (control group) or the biofeedback intervention group (experimental group), with 18 subjects in each group. Two subjects did not complete

the entire experimental procedure; therefore, 34 subjects ultimately completed the trial. Detailed participant information is presented in Table 1. All subjects were informed of the trial procedures and provided written informed consent. The study was reviewed and approved by the Ethics Committee of Hubei Minzu University [(2024)072] and the Chinese Clinical Trial Registry [ChiCTR2600132290].

Table 1 Subjects' Basic Information

Group	Number (Name)	Age (years)	Height (cm)	Body Weight (kg)	Training Years (years)	Skeletal Muscle (kg)
Experimental group	17	21.82±1.63	177.02±4.44	72.10±4.69	6.06±0.83	39.82±3.64
Control group	17	21.00±1.23	175.53±3.72	74.05±7.60	6.29±0.77	37.64±3.53
<i>t</i>		1.67	1.063	-0.902	-0.858	1.770
<i>P</i>		0.105	0.296	0.375	0.397	0.086

2.2 Test Plan

Blood flow restriction (BFR) intervention was used to induce acute exercise-induced fatigue. To avoid the influence of sequence effects on the test results, subjects were tested using the ABBA balanced method [9], following the sequence “control group–experimental group–experimental group–control group.” Baseline data, including height, weight, lower-limb one-repetition maximum (1RM) values, lower-limb dimensions, and blood pressure (BP), were measured before the test. The arterial occlusion pressure (AOP) of each subject was calculated and combined with the lower-limb 1RM value to induce acute exercise-induced fatigue. For the 1RM test, subjects first completed a specific warm-up and then performed 10, 6, 3, and 1 repetitions in a non-fatigued state. The 1RM squat test was conducted by an unblinded tester. During the test, an appropriate initial load was selected based on the last submaximal load, and the load for each subsequent squat was increased by 5–15 kg until the participant could no longer maintain proper form. The maximum load completed was recorded as the 1RM [10]. After exercise, the experimental group underwent a 33-min EEG-NF, and the control group underwent a 33-min natural recovery. The specific experimental procedure is depicted in Figure 1.

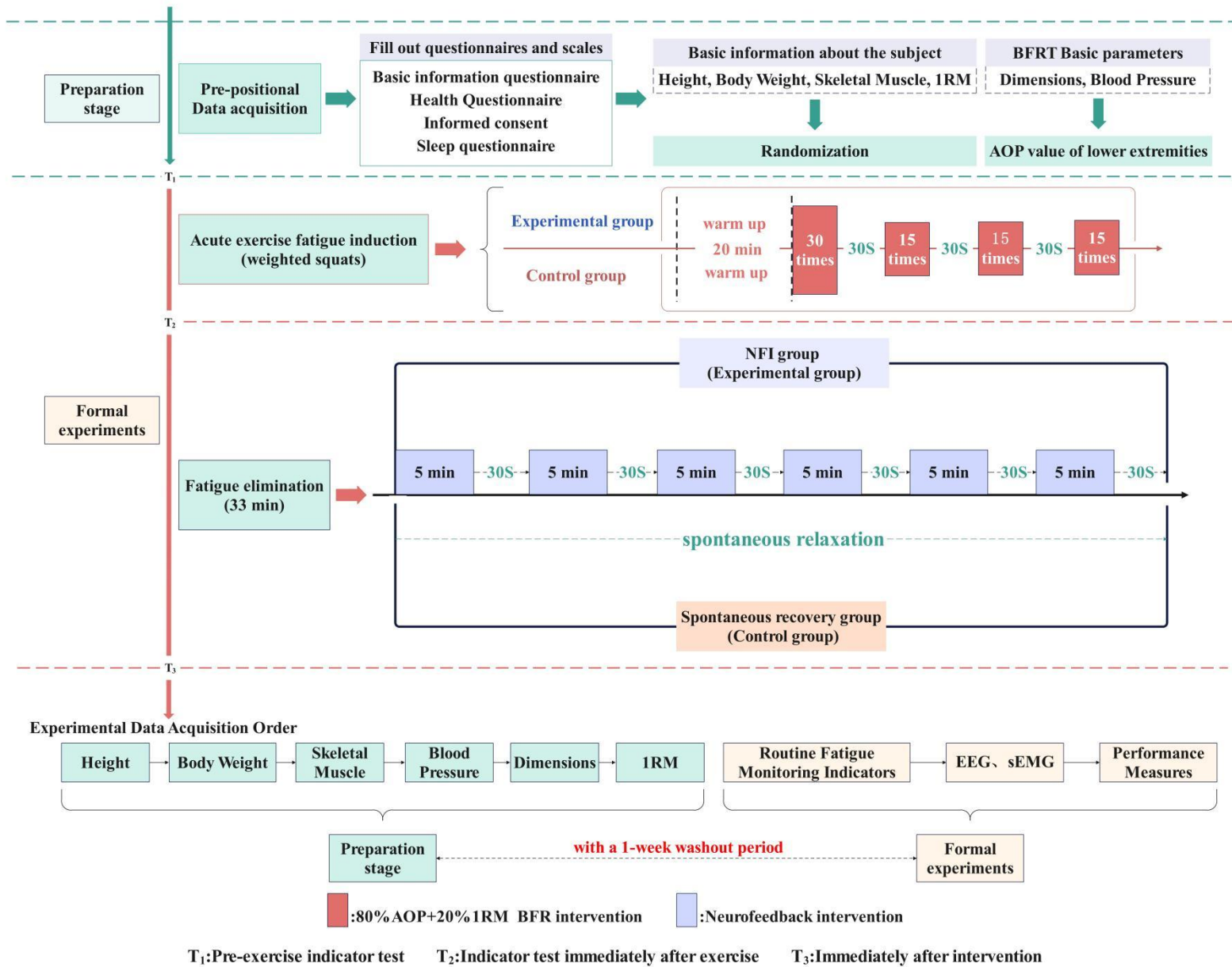


Figure 1 Experimental Process Diagram

2.2.1 Acute Exercise-Induced Fatigue Induction Plan

Subjects were instructed to avoid vigorous exercise within 12 hours before the test, refrain from consuming stimulants such as coffee, and ensure adequate sleep. Testing was conducted between 9:00 and 11:00 AM at the Sports Physiology Laboratory of Hubei Minzu University (room temperature, 20–23°C; humidity, 45%–55%). Before the experiment, subjects entered the laboratory wearing a Team Pro heart rate (HR) monitor (Polar, Finland), aktos sEMG sensors (Cometa, Italy), and the Neuracle NeuSen W EEG cap (Neuracle, China), and immediate pre-exercise data

were recorded. Immediately before exercise, a finger pulse oximeter (Wolman, Germany) was placed on the tip of the ring finger to measure blood oxygen saturation (SpO_2), and an electronic blood pressure monitor (Wolman, Germany) was used to measure blood pressure in the seated position. Subjects first completed a 20-min warm-up. The warm-up consisted of 15 min of low-intensity aerobic exercise to increase muscle temperature and promote blood circulation, without inducing fatigue. Subsequently, subjects completed two sets of drop jumps, with three repetitions per set and a 1-min interval between sets. This type of rapid eccentric–concentric contraction was used to activate muscle tissue, induce a post-activation potentiation effect, and improve subsequent force output. Dynamic stretching and joint mobility exercises, such as dynamic lunges and hip rotations, were also performed. Static stretching was strictly prohibited to avoid its inhibitory effect on explosive power. Finally, subjects completed four 30-m sprints to recruit fast-twitch muscle fibers and optimize neuromuscular conduction function, thereby bringing subjects to an optimal physiological state to adapt to the subsequent low-load experimental protocol [11].

Subsequently, BFR intervention was used to induce acute exercise-induced fatigue. BFR intervention was performed using a cuff inflation device (KAATSU Master, Japan) with a cuff width of 5 cm. The distance from the inguinal crease to the superior border of the patella was measured using a measuring tape. The cuff was wrapped around the lower one-third of the thigh, ensuring that it was perpendicular to the longitudinal axis of the thigh. The initial pressure was set at 50 mmHg [12], and the AOP of each subject was determined based on the relationship between blood flow occlusion pressure, thigh circumference, systolic blood pressure, and diastolic blood pressure. The specific formula was as follows:

$$P_{AOP} = 5.893l + 0.734P_{DBP} + 0.912P_{SBP} - 220.046$$

Note: In the formula, P_{AOP} represents the AOP; l represents thigh circumference; P_{DBP} represents diastolic blood pressure; and P_{SBP} represents systolic blood pressure.

Based on the training program reported in previous studies [13], subjects performed weighted deep squats under BFR, with a BFR training intensity of 80% AOP + 20% 1RM. The exercise protocol consisted of 30+15+15+15 repetitions per

set for a total of four sets, with a 30-s rest interval between sets. At the beginning of the exercise, the barbell was positioned stably above the deltoid muscles. Subjects stood with their feet shoulder-width apart or slightly wider, with the toes slightly outward, the chest held up, the abdomen drawn in, the core engaged, and the spine maintained in a neutral position. During the squat, the hips and knees were flexed, the buttocks were pushed backward, and the body was controlled during the vertical descent until the thighs were parallel to the ground. The knees were kept aligned with the toes to avoid inward rotation, while the heels remained firmly planted and the center of gravity remained centered. During the upward movement, the heels pushed against the ground, and the buttocks and legs exerted force to drive the body vertically upward. The trunk was maintained in a stable position without forward bending throughout the movement. The entire fatigue-induction process was assessed for movement standardization by two experimenters, one observing from the sagittal plane and the other from the coronal plane.

Table 2 Baseline Parameters of Blood-Flow Restriction Training for Subjects

Group	Lower limb dimension (cm)		SBP	DBP	1RM	80% AOP	
	L	R	(mmHg)	(mmHg)	(kg)	L	R
Experimental group	53.94±6.79	54.73±7.10	130.00±12.03	76.06±5.75	108.82±13.41	217.77±42.77	221.49±44.14
Control group	54.51±4.97	56.08±5.50	128.76±10.03	76.76±9.22	106.71±13.26	219.98±35.07	227.35±28.42

2.2.2 Exercise Fatigue Elimination Plan

The experimental group used the SPIRIT-8 multichannel biofeedback system and its supporting BioTrace software to implement EEG-NF. Electrodes were placed according to the international 10–20 electrode system, with A1 and A2 serving as reference electrodes and F3, F4, and FPz serving as signal-collection electrodes. Brain electrical activity at each lead was synchronously recorded and provided to the subjects in the form of real-time visual and auditory feedback.

Before training, individual baseline EEG signals were collected from the subjects, and the average amplitude of each frequency band was calculated. Feedback thresholds were then established accordingly. The suppression thresholds for theta waves (4–8 Hz) and beta2 waves (20–30 Hz) were set at 40% above their respective

baseline average amplitudes (corresponding to 2.6 and 2.0 μV , respectively), whereas the enhancement thresholds for sensory motor rhythm (SMR; 12–15 Hz) and beta1 waves (13–20 Hz) were set at 35% below their respective baseline average amplitudes (corresponding to 1.4 and 1.6 μV , respectively). During training, subjects controlled the movement of an arrow on the screen by autonomously regulating their EEG activity. The arrow moved toward the center of the target only when the amplitudes of theta and beta2 waves were simultaneously maintained below their preset thresholds and the amplitudes of SMR and beta1 waves were simultaneously increased above their preset thresholds. An auditory reinforcement tone with a duration of 0.5 s was triggered synchronously and repeated once per second. The detailed procedure is shown in Figure 2. Each training session consisted of six training blocks, each lasting 5 min. After each training block, subjects rested with their eyes closed for 30 s, resulting in a total session duration of 33 min. To reduce inter-operator differences, all electrodes were placed by the same trained researcher according to the international 10–20 system.

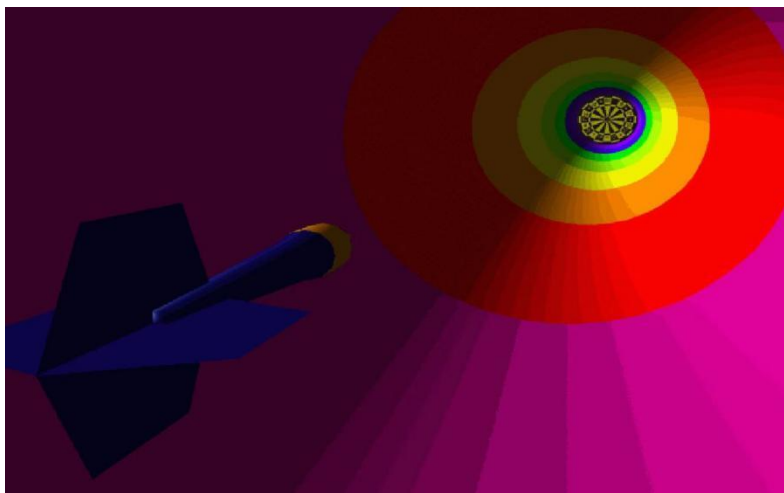


Figure 2 One Interface of the EEG-NF Module

The control group underwent 33 min of natural rest and recovery without any additional recovery interventions. During this period, subjects were prohibited from using electronic devices to avoid the effects of electronic devices on brain waves.

2.3 Test Indicators

The following indicators were collected from subjects in both groups at three time points: before exercise, immediately after the exercise intervention, and

immediately after the recovery intervention.

(1) Conventional fatigue monitoring indicators

In this study, the Borg Rating of Perceived Exertion (RPE), HR, SpO₂, and blood lactate (LA) were used as indicators to assess the fatigue status of the subjects. The RPE was assessed using a standardized questionnaire based on the Borg RPE Scale (6–20 points). HR was measured using a Team Pro HR monitor (Polar, Finland, while SpO₂ at the distal phalanx of the right ring finger was simultaneously measured using a finger pulse oximeter (Wolman, Germany). LA concentration was measured at the distal phalanx of the right index finger using a Nova portable LA analyzer (SensLab GmbH, Germany). Before the RPE assessment, each participant was given a detailed explanation by a professionally trained individual regarding the meaning of the scale and the subjective fatigue sensations corresponding to scores of 6–20. Subjects were clearly informed that the assessment represented a comprehensive subjective experience, including muscle fatigue, shortness of breath, and physical exertion. The researchers remained blinded to the subjects' assessments to avoid bias. Data were recorded based on the subjects' self-reported scores, and all data collection was performed simultaneously with the measurement of other routine indicators [14].

(2) EEG Signal Indicators

Raw EEG signals were obtained using the NeuSen W (Neuracle, China) wireless EEG acquisition system and recorded at a sampling frequency of 1000 Hz. Subjects washed and dried their hair 2 h before the test. Thirty minutes before the test, subjects entered the Sports Physiology Laboratory of Hubei Minzu University and wore a 16-channel EEG cap. The electrode layout followed the international 10–20 system, with REF serving as the reference electrode and GND serving as the ground electrode on the forehead. GT-10 medical conductive paste (GreenTech, China) was applied through the electrode holes, and the amount was adjusted to maintain electrode impedance below 5 k Ω . EEG signals were collected in the open-eye state before and after exercise and during the corresponding post-exercise intervals. Signals recorded during the simulated test and rest periods were excluded.

After acquisition of the raw EEG signals, the data were processed using

MATLAB R2024a and the EEGLAB toolbox. A band-pass filter of 0.5–60.0 Hz was applied, followed by a 50-Hz notch filter. The filtered EEG signals were divided into 10-s segments. Independent component analysis (ICA) was then performed to decompose the EEG data into independent components, and artifacts caused by blinks, electrocardiographic (ECG) activity, power-line interference, and head movements were removed.

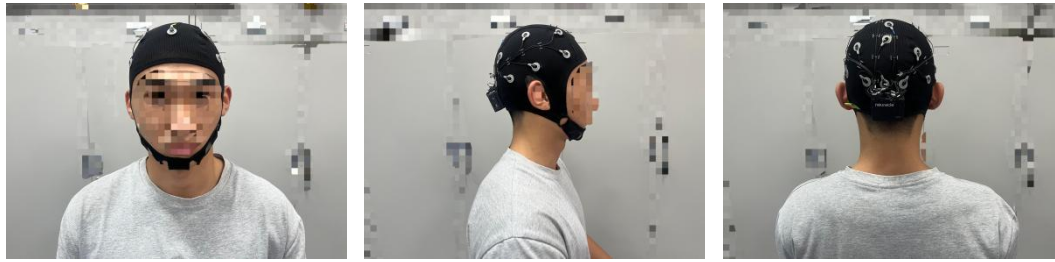


Figure 3 Specific Wearing Method of the EEG Cap

To improve the stability of data evaluation, power spectral analysis of the target signals was performed using the Welch method. After calculating the power spectral density (PSD), band power was calculated according to the corresponding formula, representing the power of the signal within a specific frequency range. The calculation was performed using the EEGLAB program, and the data were expressed in $\mu\text{V}^2/\text{Hz}$. Alpha waves (8–13 Hz), beta waves (13–30 Hz), and theta waves (4–8 Hz) were selected as EEG signal evaluation indicators. The central regions (C3 and C4), parietal regions (P3 and P4), temporal regions (T3 and T4), and occipital regions (O1 and O2) were selected as the main observation areas, and the regional mean value was used as the statistical measure. The specific electrode placement is shown in Figure 3.

(3) sEMG Index

sEMG time-domain indicators are widely used to quantify the dynamic characteristics of skeletal muscle activation levels, contraction intensity, and fatigue progression, whereas frequency-domain indicators are commonly used to monitor exercise fatigue progression, analyze metabolic characteristics, and determine fatigue thresholds [15]. In this study, the Aktos sEMG system was used to collect electromyographic data, with the vastus medialis, rectus femoris, erector spinae, and gastrocnemius lateralis selected as the main measurement sites. The specific locations

are shown in Figure 4. MateBook X Pro laptop (Huawei, China) and its MX Brio camera (Logitech, China) were used to record the electromyographic signals during the unloaded deep-squat procedure. Surface electrodes were fixed at the measurement sites with an interelectrode distance of 20 mm, and the reference electrode was placed adjacent to the measurement site. Hair was removed from the measurement sites, and the skin was cleaned with alcohol. During the preparation stage, subjects stood naturally with their feet shoulder-width apart, arms hanging naturally at both sides of the body, elbows slightly bent, palms relaxed, fingers slightly curled, and fingertips pointing downward. During the test, subjects performed the squat movement uniformly and slowly backward and downward around the hip joint, paused at the lowest position for 0.5–1 s, and then rapidly pushed the body upward to return to the starting position. After acquisition of the raw electromyographic signals, the EMG&Motion Tools program was used to process the data. First, an 8-Hz high-pass filter and a 500-Hz low-pass filter were applied [16], and the complete electromyographic data recorded during the unloaded deep-squat procedure were extracted [17]. The root mean square (RMS) amplitude, median frequency (MF), and mean power frequency (MPF) were selected as the evaluation indicators.



Figure 4 sEMG Potential Location Map

(4) Performance Indicators

The maximum strength of the lower-limb quadriceps and calf muscles was measured using the MicroFET3 portable digital muscle strength and spinal joint mobility measurement instrument (Hoggan, USA). During the quadriceps maximum

strength test, the participant was placed in a supine position, with the hands naturally resting at both sides of the body and the palms facing upward. The non-test leg was flexed at the knee, with the heel pressed against the knee of the test leg to maintain body stability. The tester stood near the proximal part of the participant's limb, with one arm passing through the popliteal fossa of the test-side lower limb and the palm supporting and stabilizing the knee joint in a flexed position. The other hand held the measuring instrument, which was placed against the distal end of the tibia. During the calf muscle maximum strength test, the participant was placed in a prone position, with the hands naturally positioned in front of the body and the palms facing downward. The non-test leg was flexed at the knee, with the dorsum of the foot pressed against the upper part of the ankle joint of the test leg to maintain body stability. The tester stood near the distal part of the participant's limb, with one arm stabilizing the posterior aspect of the test-side thigh to restrict leg movement and the other hand holding the measuring instrument, which was placed against the heel. During the test, the participant exerted force continuously for 4 s, while the tester simultaneously applied resistance in the opposite direction to maintain the stability of the measuring instrument. The test data were then recorded in kilograms (kg).

The vertical jump height during the squat jump was measured using the EZE Jump wireless vertical jump testing system (Australia). During the preparation stage, subjects assumed a natural standing position with their feet shoulder-width apart and parallel, the entire soles of the feet flat on the ground, and the upper body naturally upright. The head was held upright with the eyes facing forward, the shoulders naturally relaxed, and the arms hanging at the sides. The hip, knee, and ankle joints were maintained in a naturally extended position, with the center of gravity positioned over the center of the feet. Subjects maintained body stability throughout the test without swaying or tilting. Upon hearing the command, subjects quickly squatted until the thighs were parallel to the ground and then immediately jumped upward. To eliminate the influence of differences in knee angle on the measurement of jump height, two experimenters simultaneously assessed the standardization of the subjects' movements from the sagittal and coronal planes, maintaining the knee angle at

approximately 90. The test results were recorded in centimeters (cm).

Standing long jump performance was measured using a LaserLynx PRO laser electronic distance meter (FinishLynx, USA). During the test, subjects stood with their feet parallel behind the starting line and naturally positioned approximately shoulder-width apart. They slightly flexed their knees, swung their arms naturally backward, and maintained a relaxed and ready position. Both feet remained firmly on the ground, and the toes did not cross the starting line. After the preparatory swing, subjects swung their arms forward, pushed off the ground with both feet, jumped forward, and landed on both feet with the body leaning forward. After landing, subjects maintained body stability without moving. To eliminate the influence of the starting posture and irregular landing movements on standing long jump performance, two experimenters simultaneously assessed the standardization of the subjects' movements from the sagittal and coronal planes, strictly controlling the starting position and landing posture. The test results were recorded in centimeters (cm).

2.4 Data Processing

SPSS 27.0 and Excel were used for statistical analysis and data processing. The Shapiro–Wilk (S–W) test was used to assess the normality of the data, and Levene's test was used to assess the homogeneity of variances. An independent-samples t-test was used to compare the baseline characteristics of the two groups. A 3 (time: before exercise, after exercise, and after intervention) $\times$ 2 (group: experimental group and control group) repeated-measures analysis of variance (ANOVA) was used to assess the interaction between group and time and to perform simple-effects analyses. Partial eta squared (η^2_p) was used to assess effect size, according to Cohen's criteria: $\eta^2_p < 0.01$ indicates a minimal effect; $0.01 \leq \eta^2_p < 0.06$ indicates a small effect; $0.06 \leq \eta^2_p < 0.14$ indicates a moderate effect; and $\eta^2_p \geq 0.14$ indicates a large effect. Data are presented as the mean $\pm$ standard deviation (M $\pm$ SD), and $P < 0.05$ was considered statistically significant.

3 Research Results

3.1 Comparison of routine fatigue-monitoring indicators between the two subject groups

A three-way repeated measures ANOVA was conducted on fatigue indicators with 3 (time: pre-exercise, post-exercise, post-intervention) $\times$ 2 (groups: experimental group, control group) factors. LA ($F = 3.713$, $P = 0.032$, $\eta^2_p = 0.341$), RPE ($F = 6.202$, $P = 0.004$, $\eta^2_p = 0.205$; Table 3; Figure 5) indicated an interaction between the group and time variables. The LA ($P < 0.05$, $\eta^2_p = 0.091$) and RPE ($P < 0.05$, $\eta^2_p = 0.285$) of the control group after intervention were higher than those of the experimental group. The simple effect of LA within the experimental group ($P < 0.05$, $\eta^2_p = 0.961$) and the control group ($P < 0.05$, $\eta^2_p = 0.953$) was significant; the simple effect of RPE within the experimental group ($P < 0.05$, $\eta^2_p = 0.950$) and the control group ($P < 0.05$, $\eta^2_p = 0.953$) was significant. LA and RPE were higher after exercise than before exercise ($P < 0.05$) and after intervention ($P < 0.05$), and the values after intervention were higher than those before exercise ($P < 0.05$).

Table 3 Comparison of Changes in Routine Fatigue-monitoring Indicators Between the Two Subject Groups

Fatigue Index	Experimental group			Control group			<i>F</i>	<i>P</i>
	Pre-exercise	Post-exercise	Post-intervention	Pre-exercise	Post-exercise	Post-intervention		
HR (bpm)	71.76 $\pm$ 7.42	169.12 $\pm$ 6.00	73.65 $\pm$ 8.34	70.53 $\pm$ 5.46	167.59 $\pm$ 7.39	74.76 $\pm$ 6.14	0.411	0.666
SpO ₂ (%)	98.35 $\pm$ 0.79	94.47 $\pm$ 1.13	98.24 $\pm$ 0.83	98.29 $\pm$ 0.77	94.59 $\pm$ 1.33	98.47 $\pm$ 0.63	0.249	0.780
LA (mmol/L)	1.77 $\pm$ 0.14	11.18 $\pm$ 1.21 [*]	5.01 $\pm$ 0.72 ^{*▲}	1.75 $\pm$ 0.10	10.69 $\pm$ 1.28 [*]	5.68 $\pm$ 0.65 ^{*▲#}	3.713	0.032
RPE (Points)	7.35 $\pm$ 0.79	17.65 $\pm$ 1.17 [*]	11.41 $\pm$ 1.00 ^{*▲}	7.47 $\pm$ 0.80	17.59 $\pm$ 0.94 [*]	12.82 $\pm$ 1.07 ^{*▲#}	6.202	0.004

Note: ^{*}Compared with the same group before exercise, $P < 0.05$; [▲]Compared with the same group after exercise, $P < 0.05$; [#]difference between groups at the same time point is $P < 0.05$, [&]interaction between time and group, $P < 0.05$, as mentioned above.

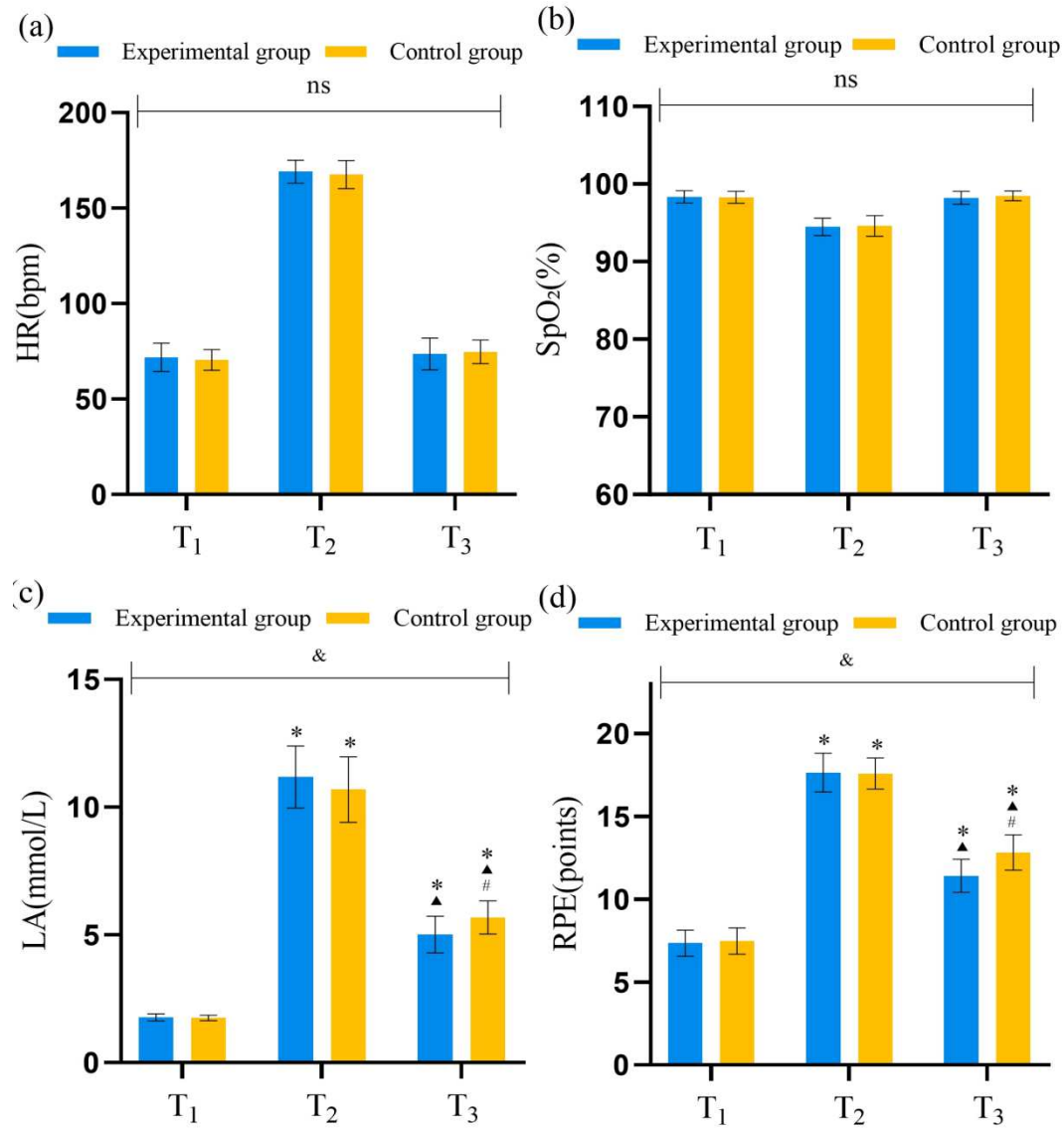


Figure 5 Comparison of Changes in Routine Fatigue-Monitoring Indicators Between the Two Subject Groups

Note: (a) Comparison of the HR of subjects in the two study groups; (b) comparison of SpO₂ of the subjects in the two groups; (c) comparison of LA of the subjects in the two groups; (d) comparison of RPE of the subjects in the two groups.

3.2 Comparison of Changes in EEG Signal between the Two Subject Groups

3.2.1 Comparison of changes in α -wave indicators between the two subject groups

Central area ($F = 3.443$, $P = 0.040$, $\eta^2_p = 0.125$), parietal lobe ($F = 3.251$, $P = 0.047$, $\eta^2_p = 0.119$), and occipital lobe ($F = 3.298$, $P = 0.045$, $\eta^2_p = 0.121$; Table 4; Figure 6) display an interaction between group and time. After the between-group

intervention, the control group in the central area ($P < 0.05$, $\eta^2_p = 0.291$), parietal lobe ($P < 0.05$, $\eta^2_p = 0.201$), and occipital lobe ($P < 0.05$, $\eta^2_p = 0.178$) were lower than the corresponding factors of the experimental group. The simple effect within the central area group was significant in the experimental group ($P < 0.05$, $\eta^2_p = 0.704$) and the control group ($P < 0.05$, $\eta^2_p = 0.595$); the simple effect within the parietal lobe group was significant in the experimental group ($P < 0.05$, $\eta^2_p = 0.426$) and the control group ($P < 0.05$, $\eta^2_p = 0.639$); the simple effect within the occipital lobe group was significant in the experimental group ($P < 0.05$, $\eta^2_p = 0.721$) and the control group ($P < 0.05$, $\eta^2_p = 0.707$). The central area and occipital lobe groups were lower after exercise than before exercise ($P < 0.05$) and after intervention than before intervention ($P < 0.05$). The parietal lobe experimental group was lower after exercise than before exercise ($P < 0.05$) and after intervention than before intervention ($P < 0.05$); the parietal lobe control group was lower after exercise than before exercise ($P < 0.05$) and after intervention than before intervention ($P < 0.05$).

Table 4 Comparison of α -Wave Changes between the Two Subject Groups ($\mu V^2/Hz$)

Part	Experimental group			Control group			<i>F</i>	<i>P</i>
	Pre-exercise	Post-exercise	Post-intervention	Pre-exercise	Post-exercise	Post-intervention		
Central area	3.88±0.19	3.29 ± 0.11 *	3.72±0.19*▲	3.79±0.26	3.24 ± 0.06*	3.47±0.20*▲#	3.443	0.040
Parietal lobe	4.63±0.31	3.72 ± 0.60*	4.26±0.37*▲	4.58±0.36	3.68±0.27*	3.81 ± 0.29*#	3.251	0.047
Temporal lobe	2.92±0.31	2.38±0.26	2.61±0.35	3.01±0.21	2.34±0.26	2.57±0.28	0.695	0.504
occipital lobe	5.16±0.49	3.77 ± 0.21 *	4.79±0.37*▲	5.09±0.50	3.80±0.21*	4.41±0.27*▲#	3.298	0.045

Note: * Compared with the same group before exercise, $P < 0.05$; ▲ Compared with the same group after exercise, $P < 0.05$; # difference between groups at the same time point, $P < 0.05$, & interaction between time and group, $P < 0.05$, as mentioned above.

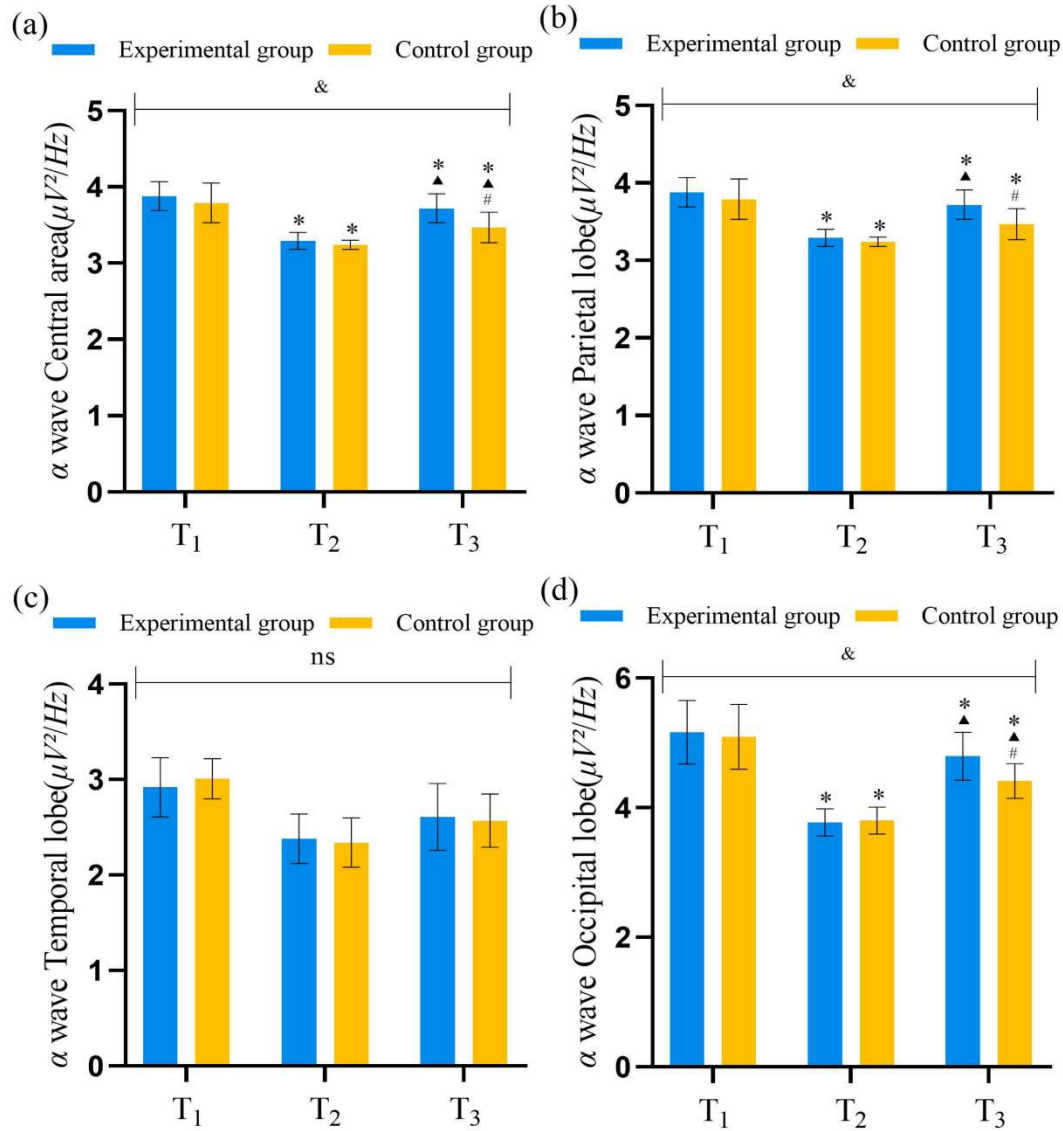


Figure 6 Comparison of Changes in EEG Signal α -Wave Indicators Between the Two Subject Groups

Note: (a) Comparison of α -waves in the central area of the subjects in the two groups; (b) comparison of α -waves in the parietal lobe of subjects in the two groups; (c) comparison of α -waves in the temporal lobe of subjects in the two groups; (d) comparison of α -waves in the occipital lobe of subjects in the two groups.

3.2.2 Comparison of β -wave index changes in the two subject groups

The central area ($F = 3.482$, $P = 0.039$, $\eta^2_p = 0.127$), occipital lobe ($F = 4.795$, $P = 0.013$, $\eta^2_p = 0.167$; Table 5; Figure 7) displayed an interaction between group and time. After the between-group intervention, the control group in the central area ($P < 0.05$, $\eta^2_p = 0.270$) and the occipital lobe ($P < 0.05$, $\eta^2_p = 0.277$) were higher than in the

experimental group. The simple effect within the central area group in the experimental group ($P < 0.05$, $\eta^2_p = 0.666$) and the control group ($P < 0.05$, $\eta^2_p = 0.814$) was significant; the simple effect within the occipital lobe group in the experimental group ($P < 0.05$, $\eta^2_p = 0.766$) and the control group ($P < 0.05$, $\eta^2_p = 0.797$) was significant. The central area and occipital lobe within the same group were higher after exercise than before exercise ($P < 0.05$), after intervention ($P < 0.05$), and after intervention than before exercise ($P < 0.05$).

Table 5 Comparison of β -wave Changes Between the Two Subject Groups ($\mu V^2/Hz$)

Part	Experimental group			Control group			<i>F</i>	<i>P</i>
	Pre-exercise	Post-exercise	Post-intervention	Pre-exercise	Post-exercise	Post-intervention		
Central area	0.55±0.12	1.06 ± 0.23 [*]	0.75±0.08 ^{*▲}	0.57±0.07	1.10 ± 0.13 [*]	0.87±0.11 ^{*▲#}	3.482	0.039
Parietal lobe	0.93±0.15	1.38±0.17	1.04±0.05	0.89±0.12	1.35±0.20	1.13±0.10	2.661	0.080
Temporal lobe	0.93±0.07	1.20±0.17	1.09±0.10	0.97±0.08	1.18±0.14	1.08±0.12	0.902	0.412
occipital lobe	0.73±0.57	1.19 ± 0.14 [*]	0.95±0.10 ^{*▲}	0.75±0.08	1.22 ± 0.10 [*]	1.10±0.13 ^{*▲#}	4.795	0.013

Note: ^{*}Compared with the same group before exercise, $P < 0.05$; [▲]Compared with the same group after exercise, $P < 0.05$; [#]difference between groups at the same time point, $P < 0.05$, [&]interaction between time and group, $P < 0.05$, as mentioned above.

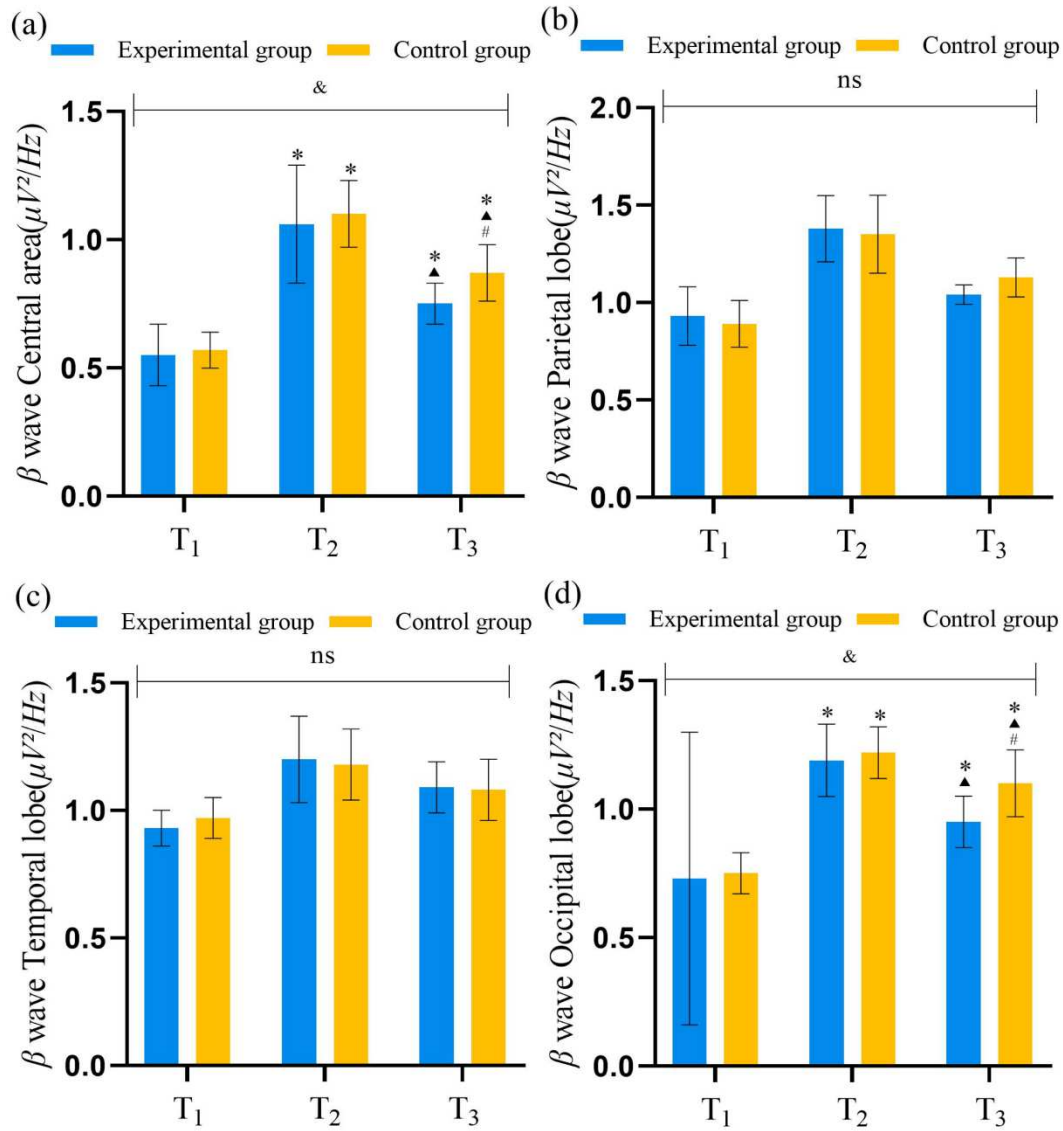


Figure 7 Comparison of Changes in EEG signal β -wave Indicators Between the Two Subject Groups

Note: (a) Comparison of β -waves in the central area of subjects in the two groups; (b) comparison of β -waves in the parietal lobe of subjects in the two groups; (c) comparison of β -waves in the temporal lobe of subjects in the two groups; (d) comparison of β -waves in the occipital lobe of subjects in the two groups.

3.2.3 Comparison of changes in θ -wave indicators between the two subject groups

The central area ($F = 7.135$, $P = 0.002$, $\eta^2_p = 0.229$), parietal lobe ($F = 3.389$, $P = 0.042$, $\eta^2_p = 0.124$; Table 6; Figure 8) showed an interaction between group and time. After the between-group intervention, the central area of the control group ($P < 0.05$,

$\eta^2_p = 0.303$) and the parietal lobe ($P < 0.05$, $\eta^2_p = 0.204$) were higher than those of the experimental group. The simple effect within the central area of the experimental group ($P < 0.05$, $\eta^2_p = 0.285$) and the control group ($P < 0.05$, $\eta^2_p = 0.206$) was significant; the simple effect within the parietal lobe of the experimental group ($P < 0.05$, $\eta^2_p = 0.447$) and the control group ($P < 0.05$, $\eta^2_p = 0.461$) was significant. The central area of the experimental group was higher after exercise than before exercise ($P < 0.05$) and after intervention ($P < 0.05$). The central area of the control group was higher after exercise than before exercise ($P < 0.05$), and that after intervention was higher than that before exercise ($P < 0.05$). The parietal lobe of the experimental group was higher after exercise than before exercise ($P < 0.05$) and after intervention ($P < 0.05$); the parietal lobe of the control group was higher after exercise than before exercise ($P < 0.05$) and that after intervention was higher than that before exercise ($P < 0.05$).

Table 6 Comparison of θ -wave Changes Between the Two Subject Groups ($\mu V^2/Hz$)

Part	Experimental group			Control group			<i>F</i>	<i>P</i>
	Pre-exercise	Post-exercise	Post-intervention	Pre-exercise	Post-exercise	Post-intervention		
Central area	1.76±0.33	2.14±0.21*	1.83±0.24 [▲]	1.77±0.37	2.13±0.22*	1.98±0.29* [#]	7.135	0.002
Parietal lobe	1.52±0.42	2.22±0.25*	1.68±0.35 [▲]	1.56±0.33	2.25 ± 0.27*	2.09±0.38* [#]	3.389	0.042
Temporal lobe	2.23±0.37	3.09±0.51	2.57±0.44	2.26±0.41	3.02±0.46	2.76±0.45	2.387	0.103
Occipital lobe	1.43±0.47	2.07±0.52	1.78±0.49	1.42±0.51	2.08±0.42	1.86±0.43	0.094	0.910

Note: *Compared with the same group before exercise, $P < 0.05$; [▲]Compared with the same group after exercise, $P < 0.05$; [#]difference between groups at the same time point is $P < 0.05$, [&]interaction between time and group, $P < 0.05$, as mentioned above.

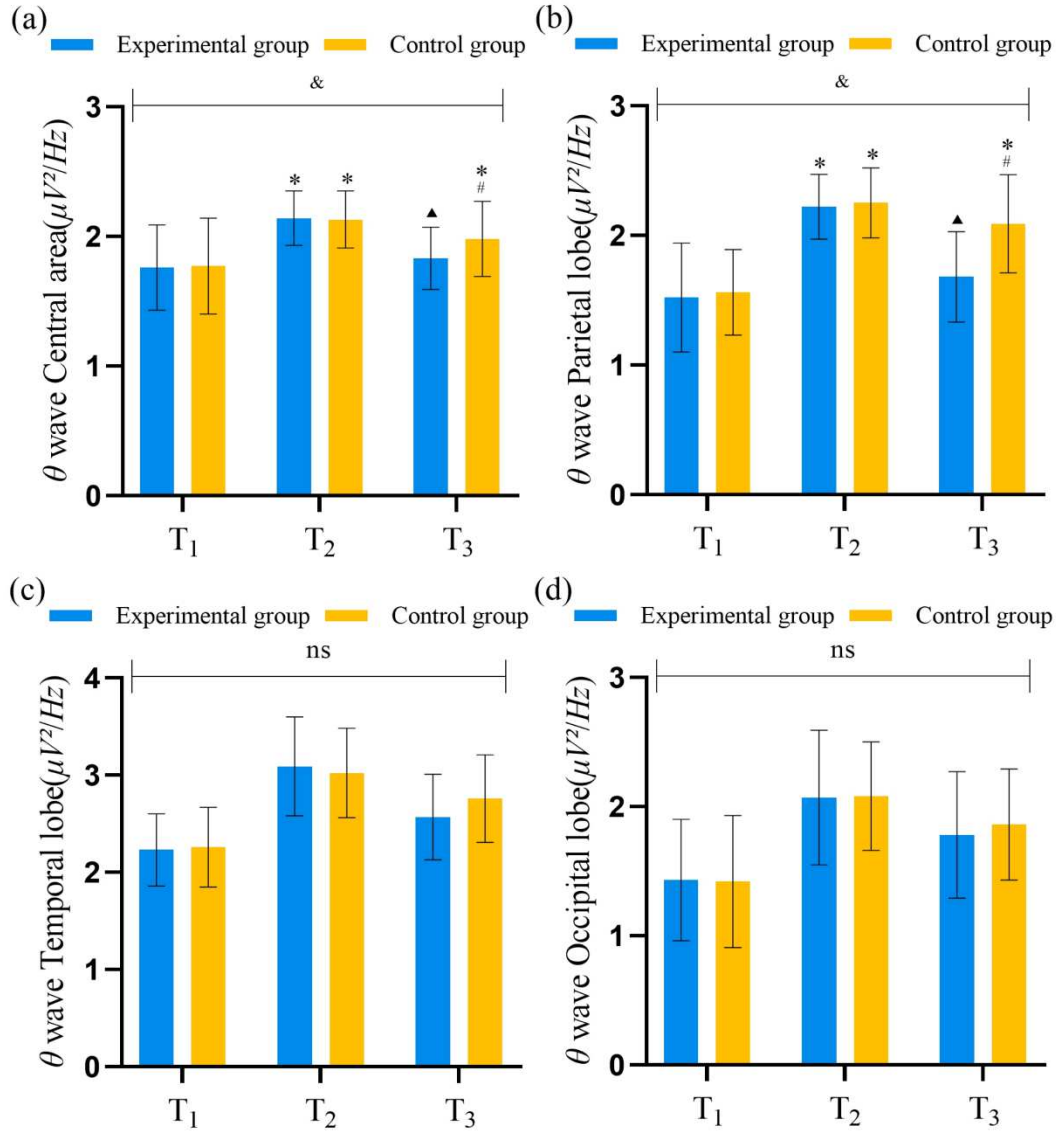


Figure 8 Comparison of Changes in EEG signal θ -wave Indicators Between the Two Subject Groups

Note: (a) Comparison of θ -waves in the central area of subjects in the two groups; (b) comparison of θ -waves in the parietal lobe of subjects in the two groups; (c) comparison of θ -waves in the temporal lobe of subjects in the two groups; (d) comparison of θ -waves in the occipital lobe of subjects in the two groups.

3.3 Comparison of Changes in sEMG Signals Between the Two Subject Groups

3.3.1 Comparison of changes in RMS indicators for the two subject groups

The vastus medialis ($F = 3.931$, $P = 0.026$, $\eta^2_p = 0.141$), vastus lateralis ($F = 3.590$, $P = 0.035$, $\eta^2_p = 0.130$), and gastrocnemius externus ($F = 3.484$, $P = 0.039$, $\eta^2_p = 0.127$; Table 7; Figure 9) have an interaction between group and time. After the

between-group intervention, the vastus medialis ($P < 0.05$, $\eta^2_p = 0.170$), vastus lateralis ($P < 0.05$, $\eta^2_p = 0.252$), and gastrocnemius externus ($P < 0.05$, $\eta^2_p = 0.115$) of the control group were higher than those in the experimental group. The simple effect within the vastus medialis group of the experimental group ($P < 0.05$, $\eta^2_p = 0.736$) and the control group ($P < 0.05$, $\eta^2_p = 0.763$) was significant; the simple effect within the vastus lateralis group of the experimental group ($P < 0.05$, $\eta^2_p = 0.679$) and the control group ($P < 0.05$, $\eta^2_p = 0.737$) was significant; the simple effect within the gastrocnemius externus group of the experimental group ($P < 0.05$, $\eta^2_p = 0.721$) and the control group ($P < 0.05$, $\eta^2_p = 0.675$) was significant. The vastus medialis, vastus lateralis, and gastrocnemius externus were higher after exercise within the same group than before exercise ($P < 0.05$) and after intervention ($P < 0.05$), and that after intervention was higher than that before exercise ($P < 0.05$).

Table 7 Comparison of RMS Indexes Between the Two Subject Groups (*uV*)

Part	Experimental group			Control group			<i>F</i>	<i>P</i>
	Pre-exercise	Post-exercise	Post-intervention	Pre-exercise	Post-exercise	Post-intervention		
Musculus femoris medialis	163.92±9.12	203.57±13.67*	177.98±6.18*▲	161.33±7.78	205.09±14.82*	189.06±6.56*▲#	3.931	0.026
Musculus rectus femoris	161.28±5.80	192.16±13.29*	173.42±5.71*▲	162.13±5.20	195.94±11.55*	183.85±7.89*▲#	3.590	0.035
Erector spinae	155.16±7.66	171.43±9.54	161.56±4.72	152.26±7.35	168.82±9.40	164.45±6.92	2.069	0.137
External sural muscle	157.81±5.44	188.29±10.69*	170.19±7.06*▲	158.16±8.34	185.38±8.79*	176.32±7.08*▲#	3.484	0.039

Note: * Compared with the same group before exercise, $P < 0.05$; ▲ Compared with the same group after exercise, $P < 0.05$; # difference between groups at the same time point, $P < 0.05$, & interaction between time and group, $P < 0.05$, as mentioned above.

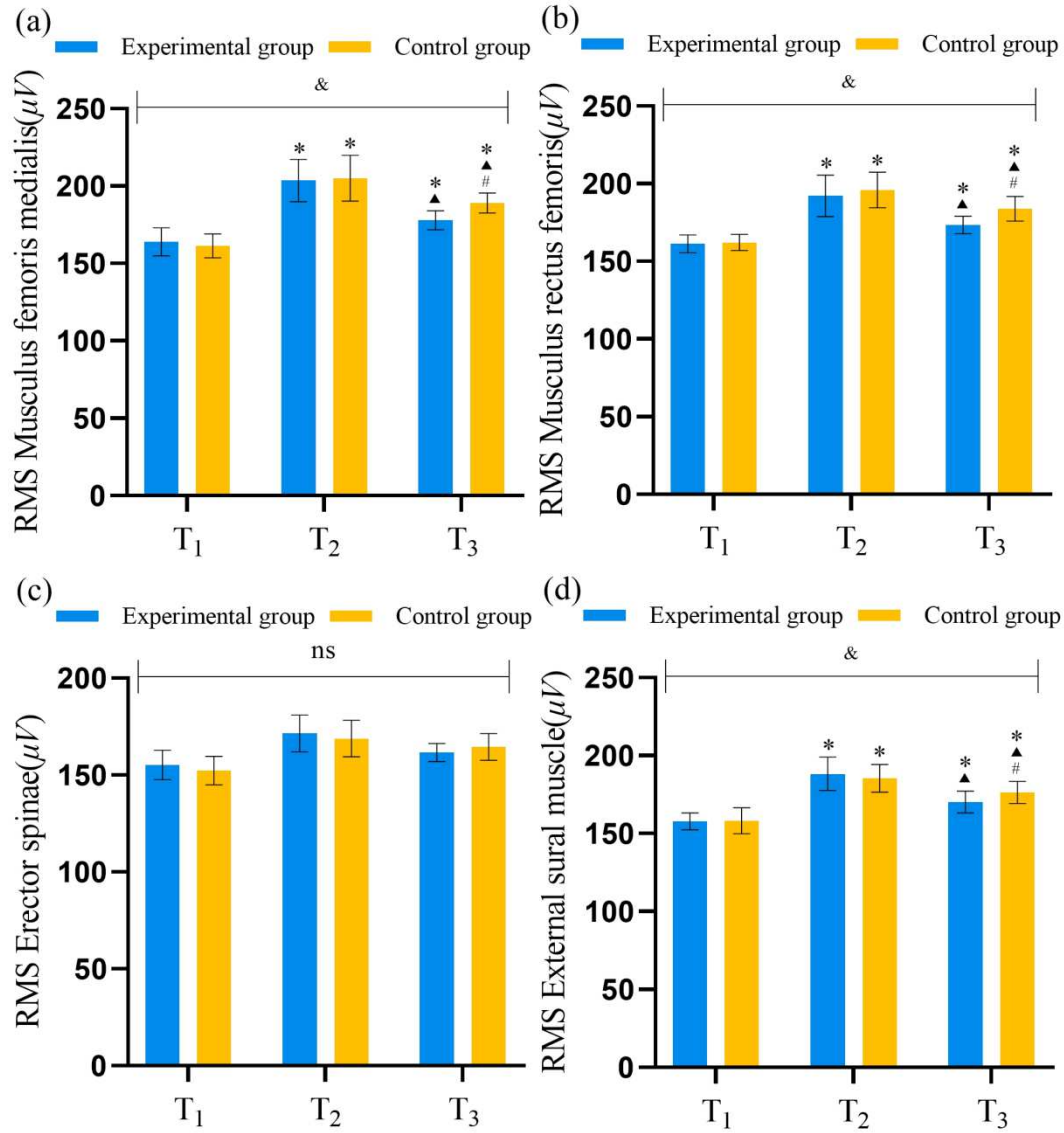


Figure 9 Comparison of Changes in sEMG Signal RMS Indexes Between the Two Subject Groups

Note: (a) Comparison of RMS in the musculus femoris medialis of subjects in the two groups; (b) comparison of RMS in the musculus rectus femoris of subjects in the two groups; (c) comparison of RMS in the erector spinae of subjects in the two groups; (d) comparison of RMS in the external sural muscle of subjects in the two groups.

3.3.2 Comparison of changes in the MF index between the two subject groups

The vastus medialis ($F = 3.216$, $P = 0.049$, $\eta^2_p = 0.118$), vastus lateralis ($F = 5.525$, $P = 0.007$, $\eta^2_p = 0.187$), and gastrocnemius externus ($F = 3.656$, $P = 0.033$, $\eta^2_p = 0.132$; Table 8; Figure 10) showed an interaction between group and time. After the between-group intervention, the vastus medialis in the control group ($P < 0.05$, $\eta^2_p =$

0.122), vastus lateralis ($P < 0.05$, $\eta^2_p = 0.110$), and gastrocnemius externus ($P < 0.05$, $\eta^2_p = 0.348$) were lower than in the experimental group. The simple effect within the vastus medialis group in the experimental group ($P < 0.05$, $\eta^2_p = 0.652$) and control group ($P < 0.05$, $\eta^2_p = 0.690$) was significant; the simple effect within the vastus lateralis group in the experimental group ($P < 0.05$, $\eta^2_p = 0.624$) and control group ($P < 0.05$, $\eta^2_p = 0.605$) was significant; the simple effect within the gastrocnemius externus group in the experimental group ($P < 0.05$, $\eta^2_p = 0.394$) and control group ($P < 0.05$, $\eta^2_p = 0.423$) was significant. The vastus medialis and vastus lateralis were lower within the same group after exercise than before exercise ($P < 0.05$) and after intervention than before exercise ($P < 0.05$). The gastrocnemius externus in the experimental group was lower after exercise than before exercise ($P < 0.05$) and after intervention than that before exercise ($P < 0.05$); the gastrocnemius externus in the control group was lower after exercise than before exercise ($P < 0.05$) and after intervention than before exercise ($P < 0.05$).

Table 8 Comparison of MF Indexes between the Two Subject Groups (Hz)

Part	Experimental group			Control group			<i>F</i>	<i>P</i>
	Pre-exercise	Post-exercise	Post-intervention	Pre-exercise	Post-exercise	Post-intervention		
Musculus femoris medialis	75.11±7.25	53.89±6.53*	66.89±5.91*▲	76.39±5.51	55.14±7.26*	60.68±5.76*▲#	3.216	0.049
Musculus rectus femoris	73.51±9.90	52.61±4.26*	65.76±5.20*▲	74.07±9.19	54.47±4.91*	63.67±5.00*▲#	5.525	0.007
Erector spinae	60.87±5.16	51.35±4.39	56.42±5.43	59.97±3.92	50.42±4.27	54.64±5.44	0.281	0.756
External sural muscle	64.97±5.97	53.73±5.29*	59.88±6.30*▲	63.31±6.45	52.34±4.66*	55.50±5.40*#	3.656	0.033

Note: * Compared with the same group before exercise, $P < 0.05$; ▲ Compared with the same group after exercise, $P < 0.05$; # difference between groups at the same time point is $P < 0.05$, & interaction between time and group, $P < 0.05$, as mentioned above.

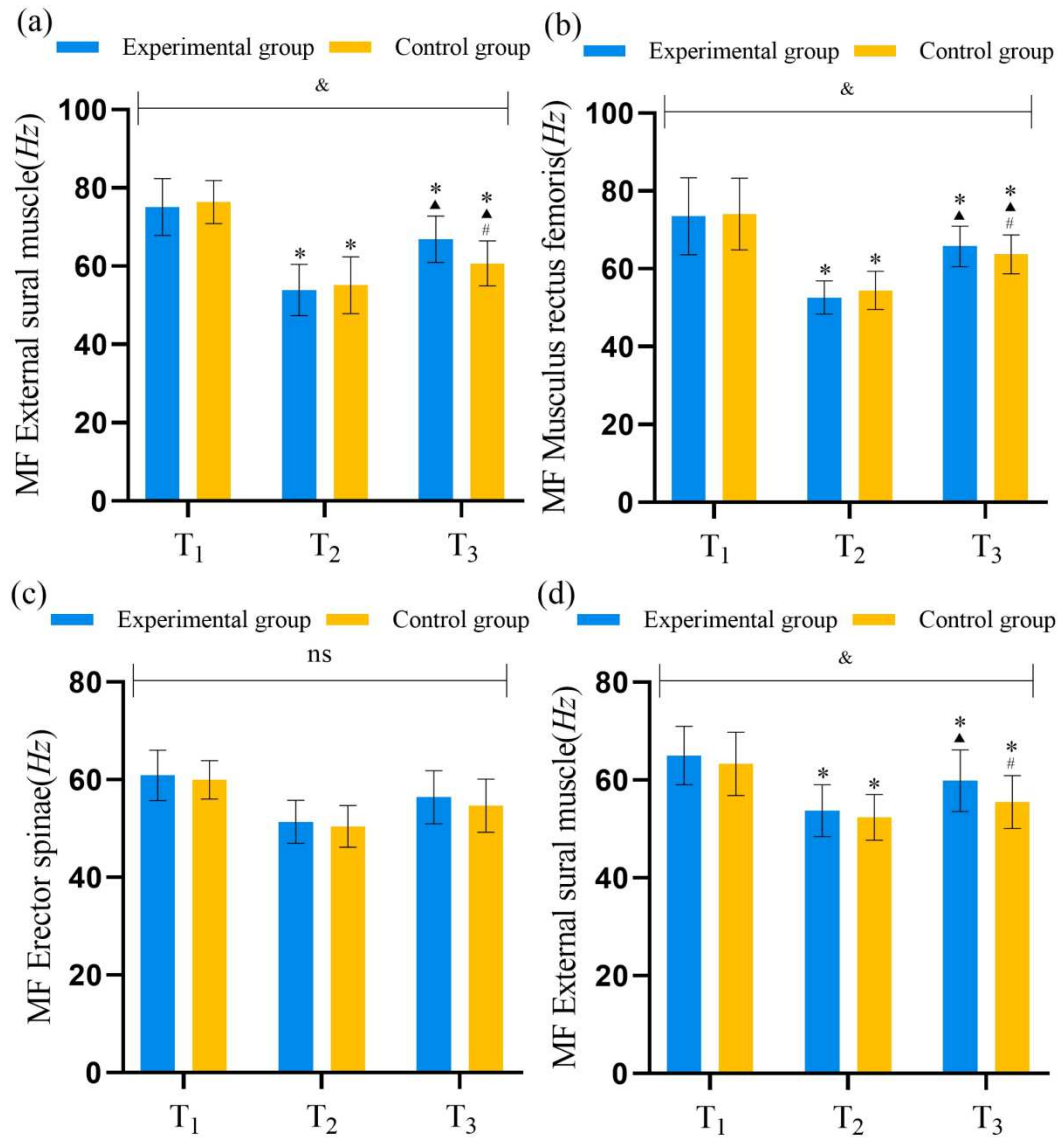


Figure 10 Comparison of Changes in sEMG Signal MF Indexes Between the Two Subject Groups

Note: (a) Comparison of MF in the musculus femoris medialis of subjects in the two groups; (b) comparison of MF in the musculus rectus femoris of subjects in the two groups; (c) comparison of MF in the erector spinae of subjects in the two groups; (d) comparison of MF in the external sural muscle of subjects in the two groups.

3.3.3 Comparison of changes in MPF indicators between the two subject groups

The vastus medialis ($F = 3.961$, $P = 0.026$, $\eta^2_p = 0.142$), vastus lateralis ($F = 3.247$, $P = 0.048$, $\eta^2_p = 0.119$), and gastrocnemius ($F = 3.273$, $P = 0.047$, $\eta^2_p = 0.120$; Table 9; Figure 11) showed an interaction between group and time. After the between-group intervention, the vastus medialis in the control group ($P < 0.05$, $\eta^2_p =$

0.148), vastus lateralis ($P < 0.05$, $\eta^2_p = 0.205$), and gastrocnemius ($P < 0.05$, $\eta^2_p = 0.105$) were lower than in the experimental group. The simple effect within the vastus medialis group in the experimental group ($P < 0.05$, $\eta^2_p = 0.684$) and control group ($P < 0.05$, $\eta^2_p = 0.597$) was significant; the simple effect within the vastus lateralis group in the experimental group ($P < 0.05$, $\eta^2_p = 0.520$) and control group ($P < 0.05$, $\eta^2_p = 0.524$) was significant; the simple effect within the gastrocnemius group in the experimental group ($P < 0.05$, $\eta^2_p = 0.555$) and control group ($P < 0.05$, $\eta^2_p = 0.599$) was significant. The vastus medialis and gastrocnemius were lower after exercise within the same group than before exercise ($P < 0.05$) and after intervention ($P < 0.05$), which was lower than before exercise ($P < 0.05$). The vastus lateralis in the experimental group was lower after exercise than that before exercise ($P < 0.05$), and, after intervention ($P < 0.05$), it was lower than that before exercise ($P < 0.05$); the vastus lateralis in the control group was lower after exercise than that before exercise ($P < 0.05$), and, after intervention, it was lower than that before exercise ($P < 0.05$).

Table 9 Comparison of MPF Indexes between the Two Subject Groups (Hz)

Part	Experimental group			Control group			<i>F</i>	<i>P</i>
	Pre-exercise	Post-exercise	Post-intervention	Pre-exercise	Post-exercise	Post-intervention		
Musculus femoris medialis	73.76±5.25	54.20±5.76*	66.97±6.00*▲	72.53±5.43	55.85±6.06*	62.64±5.89*▲#	3.961	0.026
Musculus rectus femoris	68.23±7.55	52.32±6.86*	63.29±5.09*▲	67.01±6.87	51.85±6.71*	56.06±5.08*#	3.247	0.048
Erector spinae	52.38±4.94	45.35±3.84	49.58±4.86	53.77±5.09	46.42±3.71	48.28±3.23	1.403	0.256
External sural muscle	61.42±5.02	48.60±4.67*	57.59±5.17*▲	63.19±5.32	49.23±4.10*	53.60±5.23*▲#	3.273	0.047

Note: * Compared with the same group before exercise, $P < 0.05$; ▲ Compared with the same group after exercise, $P < 0.05$; # difference between groups at the same time point is $P < 0.05$, & interaction between time and group, $P < 0.05$, as mentioned above.

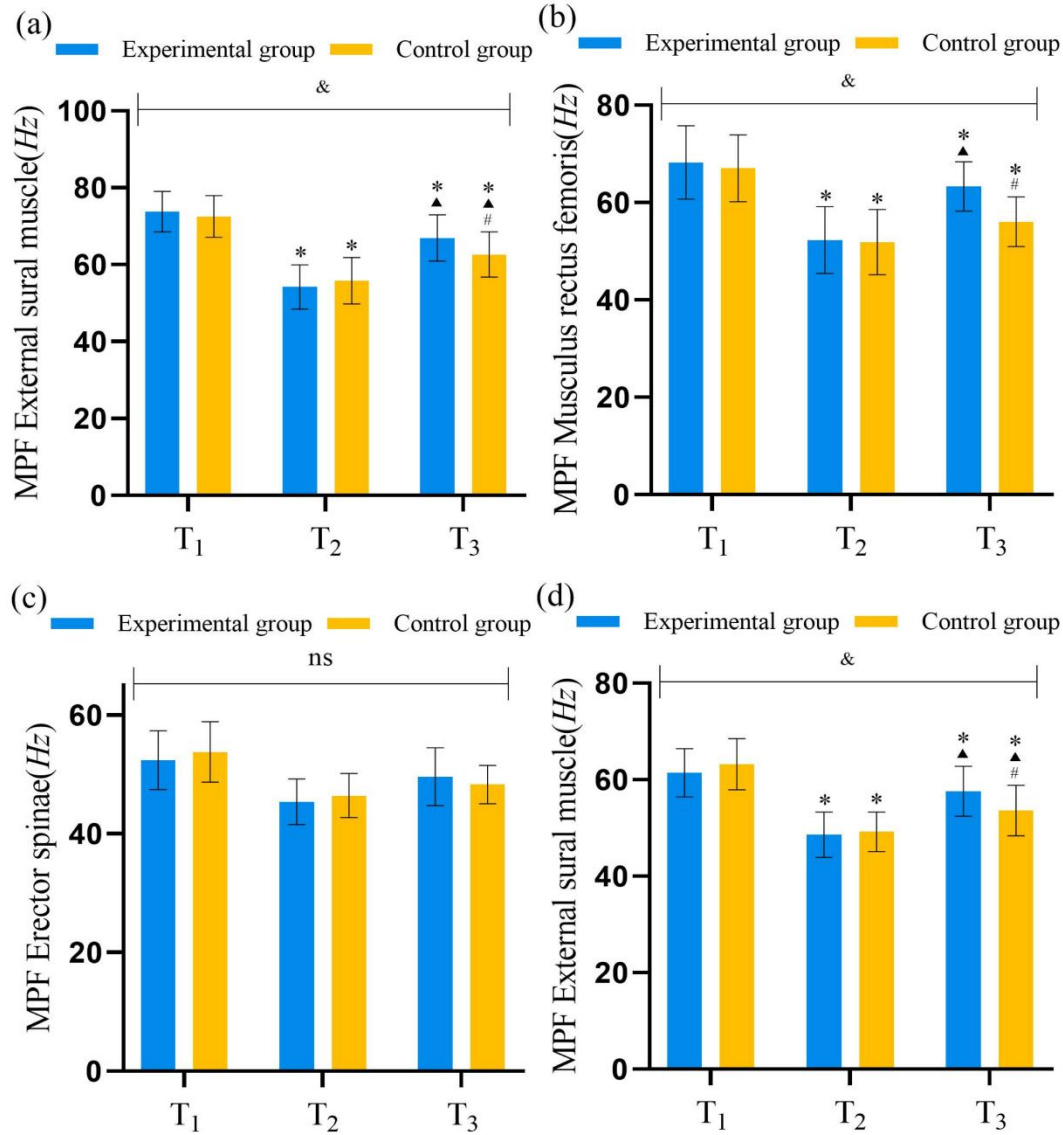


Figure 11 Comparison of changes in sEMG signal MPF Indexes Between the Two Subject Groups

Note: (a) Comparison of MPF in the musculus femoris medialis of subjects in the two groups; (b) comparison of MPF in the musculus rectus femoris of subjects in the two groups; (c) comparison of MPF in the erector spinae of subjects in the two groups; (d) comparison of MPF in the external sural muscle of subjects in the two groups.

3.4 Comparison of changes in exercise performance indicators between the two subject groups

Thigh maximum strength ($F = 3.440$, $P = 0.040$, $\eta^2_p = 0.125$), vertical jump height ($F = 4.199$, $P = 0.021$, $\eta^2_p = 0.149$), standing long jump ($F = 3.520$, $P = 0.037$, $\eta^2_p = 0.128$; Table 10; Figure 12) showed an interaction between the group and time

factors. After the between-group intervention, the control group's thigh maximum strength ($P < 0.05$, $\eta^2_p = 0.096$), vertical jump height ($P < 0.05$, $\eta^2_p = 0.092$), and standing long jump ($P < 0.05$, $\eta^2_p = 0.093$) were lower than those of the experimental group. The simple effect within the thigh maximum strength group of the experimental group ($P < 0.05$, $\eta^2_p = 0.568$) and the control group ($P < 0.05$, $\eta^2_p = 0.574$) was significant; the simple effect within the vertical jump height group of the experimental group ($P < 0.05$, $\eta^2_p = 0.674$) and the control group ($P < 0.05$, $\eta^2_p = 0.698$) was significant; the simple effect within the standing long jump group of the experimental group ($P < 0.05$, $\eta^2_p = 0.652$) and the control group ($P < 0.05$, $\eta^2_p = 0.665$) was significant. Thigh maximum strength, vertical jump height, and standing long jump were lower within the same group after exercise than before exercise ($P < 0.05$) and after intervention ($P < 0.05$), and it was lower after intervention than that before exercise ($P < 0.05$).

Table 10 Comparison of Exercise Performance Indexes between the Two Subject Groups

Part	Experimental group			Control group			<i>F</i>	<i>P</i>
	Pre-exercise	Post-exercise	Post-intervention	Pre-exercise	Post-exercise	Post-intervention		
Thigh muscle maximal strength (kg)	52.70±5.55	38.95±4.98*	45.81±4.55*▲	53.06±5.23	40.22±4.59*	43.64±4.63*▲#	3.440	0.040
Calf muscle maximal strength (kg)	32.79±4.28	28.71±3.94	32.54±4.84	32.68±4.22	29.03±4.08	30.92±4.64	2.847	0.068
Vertical Jump Height (cm)	49.31±4.97	34.74±3.25*	42.95±4.43*▲	50.92±5.34	35.59±3.18*	40.81±4.14*▲#	4.199	0.021
Standing long jump (cm)	242.44±14.52	194.06±14.72*	221.98±15.55*▲	245.73±15.48	197.41±14.48*	214.85±13.86*▲#	3.520	0.037

Note: *Compared with the same group before exercise, $P < 0.05$; ▲Compared with the same group after exercise, $P < 0.05$; #difference between groups at the same time point is $P < 0.05$, &interaction between time and group, $P < 0.05$, as mentioned above.

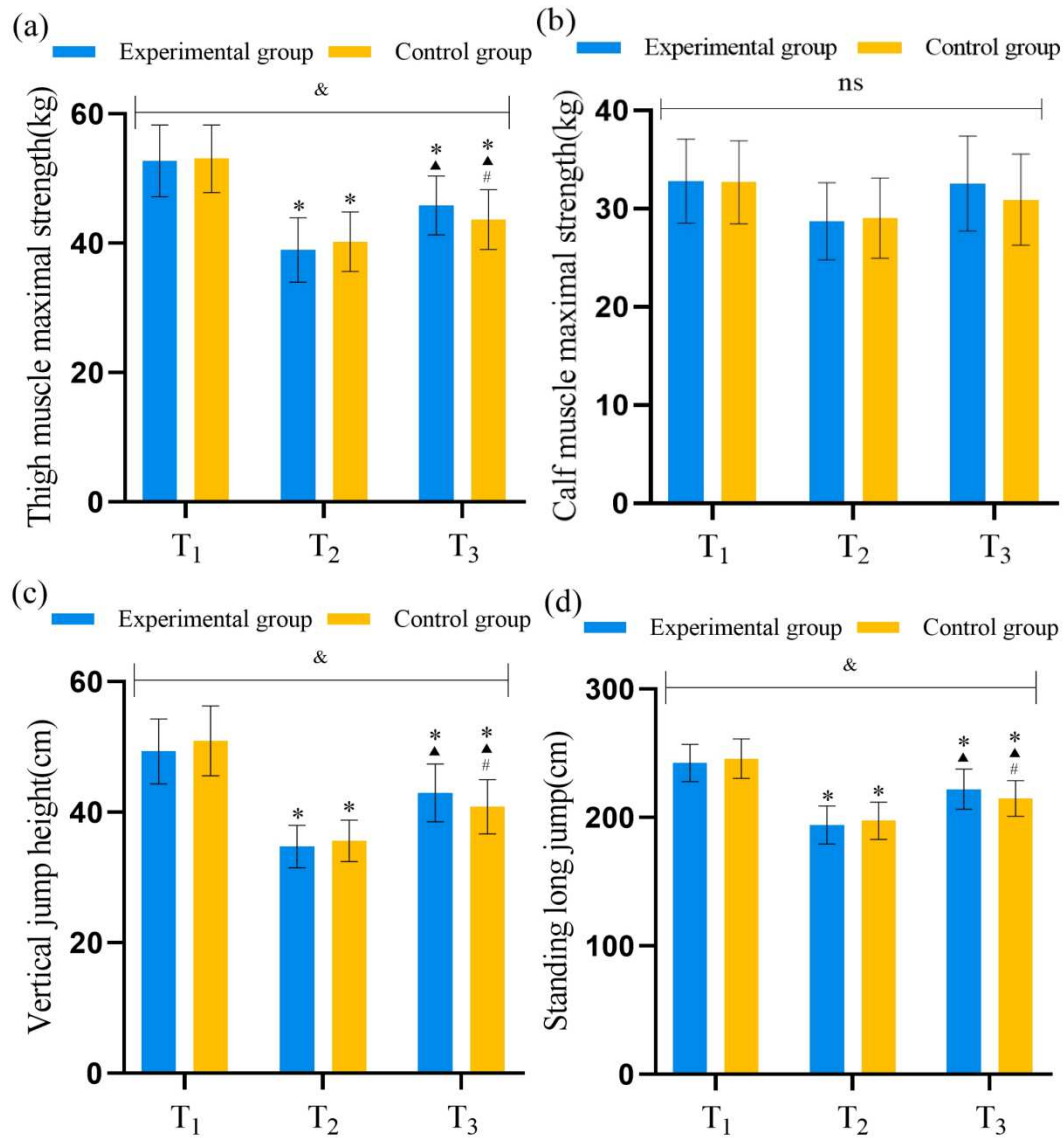


Figure 12 Comparison of Exercise Performance Indexes Between the Two Subject Groups

Note: (a) Comparison of thigh muscle maximal strength of subjects in the two groups; (b) comparison of calf muscle maximal strength of subjects in the two groups; (c) comparison of vertical jump height of subjects in the two groups; (d) comparison of standing long jump of subjects in the two groups.

4 Discussion and Analysis

4.1 Analysis of changes in EEG and sEMG signals after BFR intervention

The results of combining conventional fatigue monitoring indicators with performance indicators show that the BFR intervention program (80% AOP + 20% 1RM) adopted in this study can effectively induce subjects to produce

exercise-induced fatigue. Under fatigue conditions, the alpha waves of the subjects' EEG significantly increase, the theta waves significantly decrease, the EEG beta waves display an increasing trend, whereas the RMS of the vastus medialis and rectus femoris muscles increases significantly, and the MF and MPF significantly decrease. This change pattern is consistent with previous findings [18,19]. The present results confirm that acute BFR intervention not only causes peripheral muscle fatigue of the skeletal muscles but also inhibits CNS function, resulting in central fatigue.

BFR intervention is widely used as a novel training method in competitive sports, primarily through the precise regulation of limb arterial blood flow using a pressure device while partially restricting venous return. This promotes the accumulation of metabolic products and activates the mechanistic target of rapamycin (mTOR) pathway under low-intensity loads, thereby simulating the physiological adaptations associated with high-intensity resistance training. In an ischemic environment, BFR induces muscle cell swelling and activates mechanosensitive channels and growth factor receptors, promoting the secretion of insulin-like growth factor-1 and growth hormone and thereby enhancing anabolic hormone secretion. Meanwhile, local accumulation of LA and a decrease in pH activate the mTOR signaling pathway, promoting ribosomal biogenesis and the rate of muscle protein synthesis and thereby increasing muscle volume [20]. In addition, BFR intervention can optimize neuromuscular coordination by promoting the recruitment of fast-twitch muscle fibers and enhancing the recruitment and mobilization of motor units, thereby improving muscle strength [21].

Although BFR intervention can effectively enhance skeletal muscle function, the ischemic and hypoxic environment can also induce fatigue. During BFR intervention training, restricting muscle blood flow leads to the accumulation of metabolic products such as LA and NH_3 , which inhibit calcium release from the sarcoplasmic reticulum and cross-bridge cycling, thereby inducing peripheral muscle fatigue [22]. BFR intervention training can also induce central fatigue. High-intensity exercise can lead to an imbalance in the ratio of 5-HT to DA in the brain, with 5-HT inhibiting the excitability of motor neurons by activating the raphe nuclei, while decreased activity

of the DA pathway further weakens motivation and cortical drive [3]. Additionally, the accumulation of LA in the brain and glycogen depletion can inhibit mitochondrial ATP synthesis, reducing the efficiency of synaptic transmission. Increased blood ammonia concentrations can also cross the blood–brain barrier and directly inhibit the catalytic activity of astrocyte glutamine synthetase, thereby hindering the conversion of glutamate to glutamine. This results in delayed clearance and excessive accumulation of glutamate in the synaptic cleft, disrupts the balance between glutamate and γ -aminobutyric acid (GABA), and exacerbates excitotoxicity, thereby impairing CNS regulatory function [4] and contributing to central fatigue. Moreover, the accumulation of metabolic products induced by BFR intervention can activate group III/IV muscle afferent nerves, exacerbating central fatigue by inhibiting corticospinal excitability [23].

Due to the coupling relationship between neuromuscular functions, central fatigue can further intensify peripheral skeletal muscle fatigue. During central fatigue, weakened inhibitory feedback from type III and IV afferent nerves may reduce the activity of inhibitory interneurons in the motor cortex, thereby maintaining the output of descending excitatory commands, accelerating the depletion of phosphocreatine and ATP, and increasing the concentrations of metabolic waste products such as LA and H^+ ions, thereby exacerbating muscle fatigue [24]. In addition, neurotransmitter imbalances can directly reduce the frequency and synchrony of impulses transmitted from the cortex to spinal α -motor neurons, leading to a decrease in the number of recruited motor units and discharge frequency and preventing full muscle activation [25,26]. Moreover, central fatigue enhances the subjective perception of fatigue by increasing the sensitivity of the prefrontal cortex and insula to signals of bodily discomfort, prompting individuals to terminate exercise prematurely and indirectly limiting the capacity for sustained muscle work [27]. Therefore, central fatigue not only directly weakens neural drive signals but also exacerbates muscle fatigue at both the output and control levels by enhancing inhibitory pathways and increasing psychological tolerance thresholds.

4.2 Analysis of the intervention effect of EEG-NF on the EEG and sEMG signals

of the subjects

In this study, after natural recovery, the amplitude of theta waves in the EEG signals of the control group showed a decreasing trend, whereas the amplitude of alpha waves showed an increasing trend, suggesting that the natural recovery process can promote the recovery of CNS function and the elimination of central fatigue. After EEG-NF intervention, the amplitude of theta waves in the experimental group was significantly lower than that in the control group, whereas the amplitude of alpha waves was significantly higher, indicating that EEG-NF can optimize the synchronization of EEG rhythms and exert effective intervention effects, thereby improving CNS function. This conclusion is consistent with the findings of other scholars [28,29]. Previous studies have indicated that EEG-NF can significantly increase alpha-wave amplitude and suppress beta- and theta-wave amplitudes in athletes, showing positive effects in alleviating sports-related central fatigue, enhancing concentration and movement accuracy, reducing irrelevant interference, and improving task focus [28,29]. EEG-NF can also improve CNS and basal ganglia excitability by modulating EEG activity in archery athletes [30].

The mechanism by which EEG-NF improves CNS function is mainly related to the regulation of neurotransmitter release. EEG-NF can significantly reduce the relative level of the inhibitory neurotransmitter 5-HT and the ratio of 5-HT to DA (5-HT/DA) [6]. Reducing the 5-HT/DA ratio may inhibit 5-HT_{2A} receptor-mediated GABA release and relieve inhibition of DA neurons, thereby enhancing the efficiency of neural transmission through the neocortical DA pathway, promoting DA release, improving motor motivation and cortical drive, and reducing CNS inhibition. Additionally, EEG-NF promotes the release of DA by enhancing the functional activity of dopaminergic neural pathways [31]. This regulation, together with training, induces significant changes in EEG signals, enhances the synchronization of neural oscillations in the prefrontal cortex and anterior cingulate gyrus, promotes neural plasticity, accelerates the recovery of brain function, and ultimately alleviates symptoms of central fatigue by reducing the concentration of inhibitory neurotransmitters and improving neurotransmitter balance. Moreover, EEG-NF can

reduce excessive excitation of the motor cortex by enhancing short-interval intracortical inhibition, optimizing the balance between neural excitation and inhibition, thereby improving the accuracy of motor execution and inhibiting irrelevant muscle activity [32]. This may reduce energy consumption and alleviate central fatigue.

In this study, after natural recovery, the RMS of the vastus medialis and rectus femoris in the control group showed a decreasing trend, whereas the MF and MPF showed an increasing trend. Natural recovery is believed to promote the alleviation of skeletal muscle fatigue through mechanisms related to the synthesis of energy substrates in muscle cells, the elimination of metabolic products such as LA, and the restoration of oxidative-antioxidant function. After the EEG-NF intervention, the RMS of the vastus medialis and rectus femoris in the experimental group was significantly lower than that in the control group, while the MF and MPF were significantly higher than those in the control group. These findings indicate that EEG-NF can further improve the electrical signals of skeletal muscles after fatigue, beyond the effects of natural recovery, thereby promoting the recovery of skeletal muscle function, which may be related to the neural-muscle functional coupling. Studies have shown that the cerebral cortex controls muscle contraction through neural conduction pathways, and EEG and sEMG signals exhibit synchronous correlations, reflecting functional coupling between the brain and muscles. Due to neuromuscular functional coupling, improvements in CNS function can affect skeletal muscle function [33]. Some studies have indicated that EEG-NF regulates neural activity in specific brain regions, activates neural activity patterns, affects the nervous system's control of muscles, and thereby alters sEMG results [34]. However, other studies have shown that EEG-NF intervention in swimmers did not result in significant changes in electromyographic signal parameters [35]. This may be because swimming involves bilateral coordination and visuomotor integration, resulting in substantially lower absolute strength demands on individual muscle groups during energy metabolism than in other terrestrial sports; consequently, the exercise may not have a significant effect on electromyographic signals. In addition, changes in

electromyographic signals are influenced by various factors, such as muscle fiber conduction velocity and action potential duration, which may mask the potential effects of training on fatigue rate.

This study shows that EEG and sEMG signal parameters exhibit synchronous changes following the induction and resolution of acute exercise-induced fatigue. The physiological mechanism may involve the CNS improving muscle fatigue through neuromuscular coupling, mainly involving central drive regulation and motor unit recruitment strategies. The motor cortex and spinal motor neurons dynamically compensate for the attenuation of peripheral muscle fiber contractile function through a hierarchical neural adaptation mechanism [36]. The motor cortex maintains the output of motor commands by enhancing descending drive and activating multi-regional neural networks, thereby compensating for the decreased contractile ability of peripheral muscle fibers and promoting the alleviation of muscle fatigue. In addition, the CNS adjusts the recruitment threshold and discharge frequency of motor units and enhances the synchronization of α -motor neurons through the corticospinal pathway, thereby improving the efficiency of neural signal transmission and accelerating motor unit recruitment. These processes may also reduce the stimulation of nerve endings by LA accumulation, enhance the regulation of fatigue perception by GABAergic neurons, and promote the alleviation of muscle fatigue. In addition, the CNS adjusts the recruitment threshold and discharge frequency of motor units and enhances the synchronization regulation of α -motor neurons through the corticospinal pathway, improving the efficiency of neural signal transmission, accelerating the recruitment speed of motor units, and reducing the stimulation of LA accumulation on the nerve endings, which, in turn, enhances the GABAergic neurons' regulation of fatigue perception and eliminates muscle fatigue [37]. Moreover, γ -motor neurons may contribute to the alleviation of skeletal muscle fatigue. γ -Motor neurons actively regulate the stretch sensitivity of muscle spindles through motor innervation of the two poles of the muscle spindles, thereby maintaining basic muscle tension [38]. γ -Motor neurons optimize motor unit recruitment by enhancing the sensitivity of the muscle spindles [39]. Meanwhile, increased muscle spindle sensitivity enables muscle

spindles to rapidly and accurately perceive and transmit signals to the CNS, prompting α -motor neurons to more precisely regulate the degree and timing of muscle contraction. This may reduce energy consumption and thereby alleviate muscle fatigue [40].

Limitations: This study has several limitations. (1) The research results are only applicable to BFR training programs using a cuff width of 5 cm. Different cuff specifications may lead to differences in the results. Currently, related studies commonly use a 5-cm cuff for lower-limb BFR. A cuff of this width can produce significant ischemic and hypoxic stimulation of the lower-limb muscles while remaining within the tolerable range for the human body. Under the same pressure application conditions, a wider cuff produces greater restriction of limb blood flow, leading to a greater degree of fatigue. Therefore, when the cuff width is greater than 5 cm, it remains uncertain whether our intervention program can effectively promote the recovery of EEG and EMG signals. Thus, the conclusions of this study apply only to fatigue-relief scenarios induced using a 5-cm cuff. (2) To avoid the interference of confounding factors such as hormone levels and the menstrual cycle on the test results, this study included only male subjects. Therefore, the findings apply only to male subjects, and it remains uncertain whether the same intervention method is equally effective in females. The effectiveness of this intervention program in women therefore requires further verification.

5 Conclusion

For acute fatigue induced by BFR intervention, EEG-NF can enhance the regulatory function of the CNS and promote the alleviation of central fatigue. At the same time, EEG-NF can also promote the alleviation of peripheral muscle fatigue. Based on the synchronous changes observed in EEG and sEMG signal parameters, it is speculated that EEG-NF may promote the alleviation of peripheral muscle fatigue by improving the regulatory function of the CNS.

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