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CLINICAL TRIAL

Safety and feasibility of a functional electrical stimulation cycling-based muscular dysfunction diagnostic method in mechanically ventilated patients

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Abstract

Background: A nonvolitional diagnostic method based on FES-Cycling technology has recently been demonstrated for mechanically ventilated patients. This method presents good sensitivity and specificity for detecting muscle dysfunction and survival prognosis, even in unconscious patients. As the clinical relevance of this method has already been reported, we aimed to evaluate its safety and feasibility.

Methods: An observational prospective study was carried out with 20 critically ill, mechanically ventilated patients. The FES-cycling equipment was set in a specific diagnostic mode. For safety determination, hemodynamic parameters and peripheral oxygen saturation were measured before and immediately after the diagnostic protocol, as well as venous oxygen saturation and blood lactate. The creatine phosphokinase level (CPK) was measured before and 24, 48, and 72 h after the test. The time taken to carry out the entire diagnostic protocol and the number of patients with visible muscle contraction (capacity of perceptive muscular recruitment) were recorded to assess feasibility.

Results: Heart rate [91 ± 23 vs. 94 ± 23 bpm ($p = 0.0837$)], systolic [122 ± 19 vs. 124 ± 19 mm Hg ($p = 0.4261$)] and diastolic blood pressure [68 ± 13 vs. 70 ± 15 mm Hg ($p = 0.3462$)], and peripheral [98 (96–99) vs. 98 (95–99) % ($p = 0.6353$)] and venous oxygen saturation [71 ± 14 vs. 69 ± 14% ($p = 0.1317$)] did not change after the diagnostic protocol. Moreover, blood lactate [1.48 ± 0.65 vs. 1.53 ± 0.71 mmol/L ($p = 0.2320$)] did not change. CPK did not change up to 72 h after the test [99 (59–422) vs. 125 (66–674) ($p = 0.2799$) vs. 161 (66–352) ($p > 0.999$) vs. 100 (33–409) ($p = 0.5901$)]. The time taken to perform the diagnostic assessment was 11.3 ± 1.1 min. In addition, 75% of the patients presented very visible muscle contractions, and 25% of them presented barely visible muscle contractions.

Registered in the Brazilian Clinical Trial Registration Platform: Number: RBR-10gyv7wn.

Conclusions: The FES cycling-based muscular dysfunction diagnostic method is safe and feasible. Hemodynamic parameters, peripheral oxygen saturation, venous oxygen saturation, and blood lactate did not change after the diagnostic protocol. The muscle damage marker (CPK) did not increase up to 72 h after the diagnostic protocol.

KEYWORDS

feasibility, FES-cycling, intensive care unit, muscle, safety, weakness

1 | INTRODUCTION

Critical illness survival rates have significantly increased in intensive care units (ICU). At the same time, several sequelae have emerged in ICU survivors, such as intensive care unit-acquired weakness (ICU-AW).[1] The most common muscular disorders found in ICU patients are myopathy, polyneuropathy, and polyneuromyopathy, which are a combination of these disorders, with rates ranging from 25% to 45% of patients admitted to the ICU.[2]

Several diagnostic methods have been used to detect ICU-AW, ranging from simple strength measurement (Medical Research Council score and dynamometry)[3] to nonvolitional muscular evaluations (evocated peak torque, chronaxie determination, and electroneuromyography).[4–6] However, it is difficult to perform a functional muscular performance evaluation in the intensive care unit, due to disease severity and the level of consciousness of the patient (a large number of patients are in a coma or sedated). For the last two decades, many types of diagnostic equipment have been developed to perform nonvolitional measurements. These generally consist of a force transducer and an adjustable platform, combined with supramaximal stimuli applied over either a motor nerve or a muscle belly.[7] Several different muscle groups were tested, including the adductor pollicis,[8] ankle dorsiflexors,[9] and quadriceps.[5]

Functional electrical stimulation associated with cycle ergometry (FES-cycling) technology can be used to assess muscular performance. The aim is to promote cycle ergometry exercise induced by functional electrical stimulation. FES-cycling uses computer-driven electrical pulses delivered by transcutaneous electrodes to induce muscle contractions. The stimulating electrode creates a localized electric field that depolarizes the cell membranes of nearby neurons. If the depolarization reaches a critical threshold, an influx of sodium ions from the extracellular space to the intracellular space produces an action potential that propagates in both directions away from the site of stimulation. Action potentials that propagate proximally in the peripheral nerve axons will ultimately be annihilated at the cell body, and action potentials that propagate distally will be transmitted across the neuromuscular junction and cause muscle fibers to contract.[10] This muscle contraction must be synchronized, harmonic, and sufficient to promote a cycling movement.[11]

The equipment objectively provides a stimulator and measures the mechanical response of the muscles to power output and torque.[12] In addition, the technology can also measure the stimulation cost (electrical charge delivery rate per watt of power output).[13] Recently, this nonvolitional diagnostic method, based on FES-Cycling technology, was demonstrated in mechanically ventilated patients (69% of whom were sedated), and cut-off points were established for muscle dysfunction. This diagnostic method presents good sensitivity and specificity for detecting muscle dysfunction, even in unconscious patients. Critically ill patients present low values of torque and power output and a high stimulation cost when compared to healthy individuals. Additionally, a low stimulation cost was related to a 3-fold higher chance of survival and may be a useful tool to stratify patient severity.[14]

Apparently, the FES setup for diagnostic uses is completely different from the setup for therapeutic uses. In one therapeutic cohort,[15] patients performed FES-cycling with a 250 μs pulse width and 0–60 mA intensity. In another therapeutic cohort,[16] the patients performed FES-cycling with a 250 μs (average size legs) or 300 μs (legs with edema) pulse width and 20–30 mA intensity. For diagnostic uses,[14] a 600-μs pulse width and 117 mA average intensity were necessary to detect optimal muscle performance.

The electrical charge (pulse width and intensity) used for diagnosis is much greater than the charge for therapeutic uses. However, is this high charge safe? This is the main question of the present study. Thus, the primary aim of the current study was to evaluate the safety, while the secondary aim was to evaluate the feasibility of an FES cycling-based muscular dysfunction diagnostic method in critically ill patients undergoing mechanical ventilation. Our hypothesis was that this diagnostic method is feasible and safe (even when using high electrical charges),

without promoting any disturbance in vital signs or in the relationship between the supply and consumption of oxygen, and without causing muscular harm in critically ill patients.

2 | METHODS

2.1 | Study design

An observational prospective study was carried out from December 2021 to February 2022 in the ICU of a cardiology reference hospital in Brazil. The protocol was approved by the local ethics committee in compliance with the Declaration of Helsinki (opinion number 5.069.827/21, CAAE: 50202821.1.0000.9030) and was registered in the Brazilian Clinical Trial Registration Platform (Number: RBR-10gyv7wn). Those legally responsible for the patients signed a free and informed consent form prior to the study. Patients were consecutively admitted, and the diagnostic protocol was applied to each patient. The study included individuals over 18 years of age, of both sexes, critically ill, and mechanically ventilated (with no time limit regarding the initiation of mechanical ventilation). Patients with hemodynamic instability (mean arterial pressure < 65 or > 110 mm Hg), skin or musculoskeletal lesions that prevented FES-cycling, or lower motor neuron impairment were excluded.

2.2 | Diagnostic protocol

The patients were attached to the FES-cycling equipment (Figure 1) (MOBITRONICS®, INBRAMED, Porto Alegre, Brazil). Equipment height and distance and leg support positions were individually adjusted to prevent knee hyperextension and promote proper range of motion. The skin was cleaned, and a trichotomy was performed when necessary (in two patients) before the electrode placement. Self-adhesive electrodes, made of adhesive hydrogel and rubber (Arktus, Santa Tereza do Oeste, Brazil), were placed bilaterally on the belly of the quadriceps (vastus lateralis and vastus medialis) (5 × 9 cm electrode size), hamstrings (5 × 9 cm electrode size), and tibialis anterior muscles (5 × 5 cm electrode size), and then plugged into the electrical stimulation device cables.

Eight electrical stimulation channels were used, with the stimulation device being part of the cycling system. The FES (biphasic, interval, and rectangular shaped pulse) was set with the same pulse width and intensity in all eight channels. As a large pulse width is usually needed in critically ill patients, the pulse width range was started at a minimum of 500 μs. For the same reason, pulse amplitude intensity was started at a minimum of 50 mA. The following parameter sets were used: 500 μs pulse width for 50–100 mA intensity; 600 μs pulse width for 101–130 mA intensity; 700 μs pulse width for 131–160 mA intensity; 800 μs pulse width for 161–190 mA intensity; 900 μs pulse width for 191–220 mA intensity; and 1000 μs pulse width for 221–250 mA intensity.

The FES parameters of pulse width and intensity were set to promote the highest visible muscle contractions without pain. Prior to the assessment, the right vastus lateralis channel was activated for one second to detect the quality of muscle contraction and the pain threshold. The stimulation level started at 500 μs and 50 mA and was then ramped up in increments of 10 mA (with the corresponding pulse width, described previously) until it caused pain. When the pain threshold was reached, the stimulation increase was stopped, after which the stimulation was reduced to the previous level (10 mA before the pain threshold). Pain was evaluated in conscious patients by self-report. The patients were asked to indicate "yes or no" by nodding or shaking their heads in response to the question: "Does it hurt?" If the patient answered "yes," the parameters were reduced until a "no" answer was given. Pain in unconscious patients was evaluated by the Critical-Care Pain Observation Tool,[17] with a cut-off point ≥ 2 for pain.

To maintain muscle synergy during the cycling movement, the FES was triggered (ON) and stopped (OFF) by the crank position. The equipment has a sensor to detect the 360° crank position, and the FES trigger/stop was set according to physiological joint positions during the cycling movement. In one leg, quadriceps channels (vastus lateralis and vastus medialis) were triggered at around 90° of hip and knee flexion and stopped at around 10° of hip flexion and 160° of knee extension. In the opposite leg, hamstrings and tibialis anterior channels were triggered at around 30° of hip and knee flexion and stopped at around 75° of hip and knee flexion.

The equipment was set in the diagnostic mode to perform an automatic preset combination (unchangeable) of different passive isokinetic cycle ergometry cadences (rotations per minute—RPM) and electrical stimulation frequencies (1st = 10 RPM and 50 Hz, 2nd = 10 RPM and 75 Hz, 3rd = 10 RPM and 100 Hz, 4th = 15 RPM and 50 Hz, 5th = 15 RPM and 75 Hz, 6th = 15 RPM and 100 Hz, 7th = 20 RPM and 50 Hz, 8th = 20 RPM and 75 Hz, and 9th = 20 RPM and 100 Hz), maintaining the previously selected pulse width and intensity throughout the diagnostic protocol (in all combinations). As the equipment was in a passive isokinetic operation mode, the cycling cadence was unchanged, regardless of the pushing/pulling leg movements produced by electrical stimulation. The patients performed 7 cycling movements in each

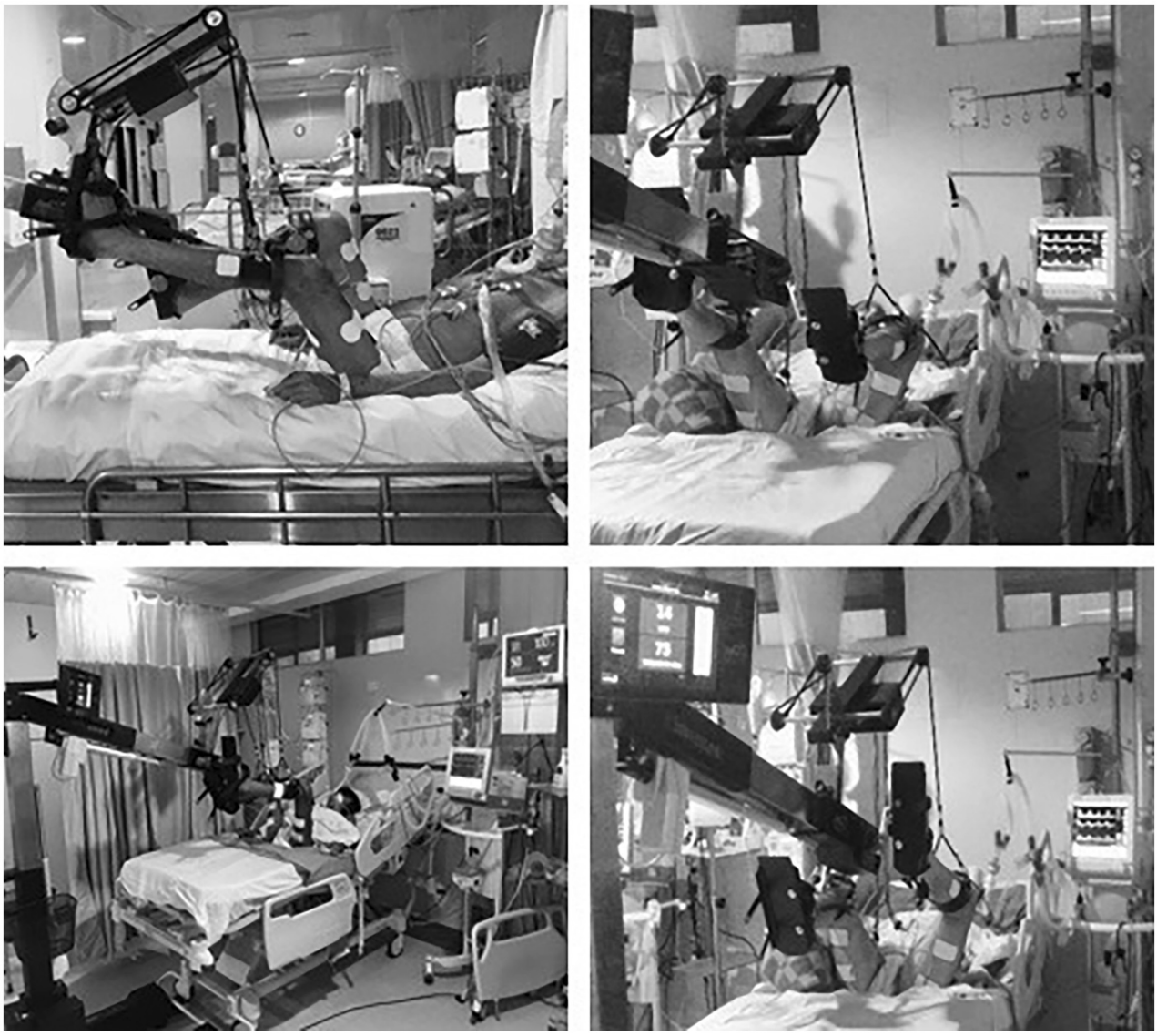

FIGURE 1 Patients attached to the FES-cycling equipment.

combination (63 cycling movements in total). The patients did not undertake any voluntary effort (most of the patients were sedated, and all conscious patients were asked not to make any voluntary effort). All the work was performed by the FES-cycling equipment.

The equipment recorded torque (newton meter—Nm) and power output (watts—W), in addition to the stimulation cost (microcoulomb/watt—μC/W), during the entire cycle ergometry cadences and electrical stimulation frequency combinations. The equipment software reported the maximal torque and power output, as well as the minimal stimulation cost achieved. Torque information is generated by the servo motor drive, which has an auto tuning that provides a specific electrical charge to maintain the programmed rotation. The torque calculation is based on the variance of the electrical charge applied to maintain the rotation. The servo motor drive also provides the angular velocity. The power output values are achieved using a mathematical calculation (torque times angular velocity). The stimulation cost represents the electrical charge (intensity times pulse width) delivered by the electrical stimulator, divided by the power output.

2.3 | Safety and feasibility assessment

The safety and feasibility assessment protocol was based on that previously described by Silva et al.[18] Patients were evaluated for 3 consecutive days after testing to determine their safety. For this purpose, 4-mL blood samples were collected daily by central venous access (all patients already had central venous access). Hemodynamic parameters (blood pressure and heart

rate) and peripheral oxygen saturation were measured before and immediately after the diagnostic protocol (IPM-9800, Mindray, China), as well as venous oxygen saturation and blood lactate (ABL 800 flex, Radiometer, Denmark). The creatine phosphokinase level in the blood (CPK) was measured before (baseline) and 24, 48, and 72 h after the test (Vitros XT 7600, Ortho Clinical Diagnostics, USA). Baseline high and low CPK levels were established using the reference values for healthy subjects provided by the manufacturer (130 IU/L for females and 170 IU/L for males).

The time taken to carry out the entire diagnostic protocol was recorded to its assess feasibility. The total time was divided into: (1) time to prepare the patient (attach the patient to the equipment, place the electrodes, plug in the cables, and set pulse width and intensity parameters); (2) time to set the angles for trigger/stop; (3) time to test the execution (execution of the 90 cycling movements); and (4) time for equipment release (unplugging the cables and removing the electrodes and the equipment). The number of patients with very visible, barely visible, or nonvisible muscle contractions was also recorded.

2.4 | Statistical analysis

Data normality was verified using the Shapiro-Wilk test. Data are presented as means ± standard deviations (when data are normally distributed) or as medians and interquartile ranges (when data are non-normally distributed) and percentages. Differences in hemodynamic parameters, peripheral oxygen saturation, venous oxygen saturation, and blood lactate were evaluated by the paired *t*-test or Wilcoxon test (according to data normality). Differences in the CPK level were evaluated by the Friedman test and Dunn's multiple comparisons test. The post hoc power of effect size achieved was also computed.[19] The effect size conventions for hemodynamic parameters, peripheral oxygen saturation, venous oxygen saturation, and blood lactate were: trivial < 0.2, small > 0.2, medium > 0.5, and large > 0.8 (*t*-test family). The effect size conventions for CPK levels were: small > 0.1, medium > 0.25, and large > 0.40 (*F*-test family). A statistically significant value of $p < 0.05$ was set for all analyses. GraphPad Prism 9.0 and GPower 3.0.10 software programs were used.

3 | RESULTS

A total of 26 patients were initially enrolled in the study, but 6 were excluded due to hemodynamic instability. The characteristics of the patients are presented in Table 1. Pulse width, intensity, total charge, power output, torque, and stimulation cost are presented in Table 2. Pulse width, intensity, and total charge parameters set for each patient are presented in Table 3.

TABLE 1 Patients characteristics.

Variable	Value
Age (years)	61 ± 20
Sex (male/female)	13/7
ICU stay (days)	6.0 (2.0–8.0)
Mechanical ventilation (days)	4.5 (2.0–8.0)
SAPS III	71 ± 10
24 h water balance (mL)	1275 ± 1562
Glucose (mg/dL)	178 ± 79
Vasoactive drugs use (*n*, %)	14 (70)
Sedation use (*n*, %)	17 (85)
Corticoids use (*n*, %)	6 (30)
Lower limbs edema (*n*, %)	10 (50)
Sepsis (*n*, %)	19 (95)
Main diagnosis	
Acute myocardial infarction (*n*, %)	4 (20)
Heart failure (*n*, %)	2 (10)
Shock (*n*, %)	4 (20)
Arrhythmia (*n*, %)	7 (35)
Myocarditis (*n*, %)	1 (5)
Acute pulmonary edema (*n*, %)	1 (5)
Acute respiratory failure (*n*, %)	1 (5)

Note: Mean ± standard deviation. Median (interquartile range).
Abbreviations: ICU, intensive care unit; SAPS III, Simplified Acute Physiology Score III.

3.1 | Safety

Heart rate = 91 ± 23 versus 94 ± 23 bpm ($p = 0.0837$), systolic = 122 ± 19 versus 124 ± 19 mm Hg ($p = 0.4261$), and diastolic blood pressure = 68 ± 13 versus 70 ± 15 mm Hg ($p = 0.3462$) did not change after the diagnostic protocol (trivial effect size) (Figure 2). Peripheral oxygen saturation = 98 (96–99) versus 98 (95–99) % ($p = 0.6353$) and venous oxygen saturation = 71 ± 14 versus 69 ± 14% ($p = 0.1317$) likewise not change after the diagnostic protocol (effect size and power) (Figure 3). Moreover, blood lactate = 1.48 ± 0.65 versus 1.53 ± 0.71 mmol/L ($p = 0.2320$) did not change after the diagnostic protocol (trivial effect size). The CPK level in all patients = 99 (59–422) versus 125 (66–674) ($p = 0.2799$) versus 161 (66–352) ($p > 0.999$) versus 100 (33–409) ($p = 0.5901$) did not change up to 72 h after the test (with a small

TABLE 2 Pulse width, intensity, total electrical charge, power output, torque, and stimulation cost.

Variable	Value
Pulse width (μs)	600 (500–800)
Intensity (mA)	124 ± 46
Total electrical charge (μC)	86 790 ± 48 048
Power output (watts)	2.9 (1.8–4.1)
Torque (Nm)	1.9 (1.1–4.3)
Stimulation cost (μC/w)	27 666 (15 371–57 975)

Note: Median (interquartile range). Mean ± standard deviation.

Abbreviations: mA, milliampere; Nm, newton meter; μC/w, microcoulombs per watt; μC, microcoulombs; μs, microseconds.

TABLE 3 Pulse width, intensity, and total electrical charge parameters set in each patient.

Patient	Pulse width (μs)	Intensity (mA)	Total electrical charge (μC)
1	500	100	50 000
2	500	50	25 000
3	800	170	136 000
4	700	140	98 000
5	900	210	189 000
6	700	130	91 000
7	600	105	63 000
8	800	161	128 800
9	600	120	72 000
10	500	80	40 000
11	800	165	132 000
12	800	170	136 000
13	600	130	78 000
14	500	100	50 000
15	500	70	35 000
16	800	170	136 000
17	500	60	30 000
18	600	120	72 000
19	800	180	144 000
20	500	60	30 000

Abbreviations: mA, miliamper; μC, microcoulombs; μs, microseconds.

effect size) (Figure 5). High CPK level patients = 637 (315–1403) versus 707 (375–1324) ($p = 0.4419$) versus 463 (286–1049) ($p = 0.9019$) versus 452 (315–1199) ($p > 0.999$) did not present changes up to 72 h after the diagnostic protocol (small effect size) (Figure 5). Finally, low CPK level patients = 66 (46–99) versus 73 (50–93) ($p = 0.9653$) versus 79 (39–181) ($p > 0.999$) versus 45 (29–107) ($p = 0.9653$) did not present changes up to 72 h after the test (small effect size) (Figure 4).

3.2 | Feasibility

The total time taken for the diagnostic protocol was 11.3 ± 1.1 min. The time to prepare the patient was 5.0 ± 1.1 min (43% of total time); to set the pulse width and intensity parameters and angles for trigger/stop, it was 0.6 (0.5–0.7) min (7% of total time); for test execution, it was 4.4 ± 0.1 (39% of total time) min; and for equipment release, it was 1.3 ± 0.3 min (11% of total time) (Figure 5). Furthermore, 15 patients (75%) presented very visible muscle contractions, and 5 patients (25%) presented barely visible muscle contractions (Figure 6).

4 | DISCUSSION

In summary, our results showed that: (1) The FES cycling-based muscular dysfunction diagnostic method is safe and feasible; (2) Hemodynamic parameters, peripheral oxygen saturation, venous oxygen saturation, and blood lactate did not change after the diagnostic protocol; (3) CPK did not increase up to 72 h after the diagnostic protocol; (4) Only a few minutes were required to perform the FES cycling-based muscular dysfunction diagnostic method; and (5) the majority of the patients presented very visible muscle contractions.

Contrary to previously reported therapeutic uses of FES-cycling,[15,16] for diagnostic uses, the FES was set with a very high intensity (124 mA) and large pulse width (600 μs), and a huge electrical charge (86 790 μC). The highest pulse width reported (300–400 μs) during FES-cycling using the therapeutic approach in mechanically ventilated patients,[20] was much lower than that used for the diagnostic approach. These stimulation levels are necessary for the best analysis, with high levels of sensitivity and specificity for detecting muscle dysfunction and survival prognosis.[14] Critically ill, mechanically ventilated patients commonly present neuromyopathy, and neuromuscular electrophysiological disorders may appear. Some patients can present chronaxie higher than 1000 μs.[21] In these patients, a high intensity and large pulse width are needed to generate the best muscular performance during diagnostic uses. In the present study, the patients presented strong muscle dysfunction. The torque and power output were below, and the stimulation cost was above the cut-off points (4.04 Nm, 4.53 W, and 7461 μC/W, respectively).[14]

As the clinical utility of the diagnostic method was reported in a previous study (including muscle dysfunction detection, cut-off points to analyze variables, and six-month survival prognosis), in the present study we primarily focused on establishing the safety of this method. The use of a high-intensity stimulation level was safe, with no reported side effects, muscle injuries, or clinical instability.

ICU patients commonly present with clinical instability and severe disease situations. Safety is therefore mandatory for any diagnostic method in this population. As previously reported,[22] an acute cardiac event during FES-cycling is an objective safety parameter to stop the procedure. Besides the huge electrical charge, the high intensity and large pulse width were safely used during the diagnostic method. No adverse events were recorded.

Medrinal et al.[23] demonstrated heart rate and blood pressure increases during FES-cycling in mechanically ventilated patients. As physiological changes were previously reported, we measured hemodynamic parameters before and immediately after the diagnostic protocol, looking for persistent clinical instability (persistent tachycardia or hypertension) caused by the diagnostic protocol. Even when hemodynamic parameters changed during FES-cycling for diagnostic use (physiological pattern), they returned to baseline values immediately after exercise cessation. No clinical instability was detected.

Godja et al.[24] demonstrated mild elevation of arterial lactate and reduced venous oxygen saturation during FES-cycling in healthy subjects. Arterial lactate increases during exercise as a result of isocapnic buffering of lactic acid produced during glycolytic muscle fiber contraction.[25] During FES-cycling, the arterial-mixed venous oxygen content difference[11] and oxygen extraction[23] are increased, reducing venous oxygen saturation. Again, as physiological changes were previously reported, we measured these variables before and immediately after the

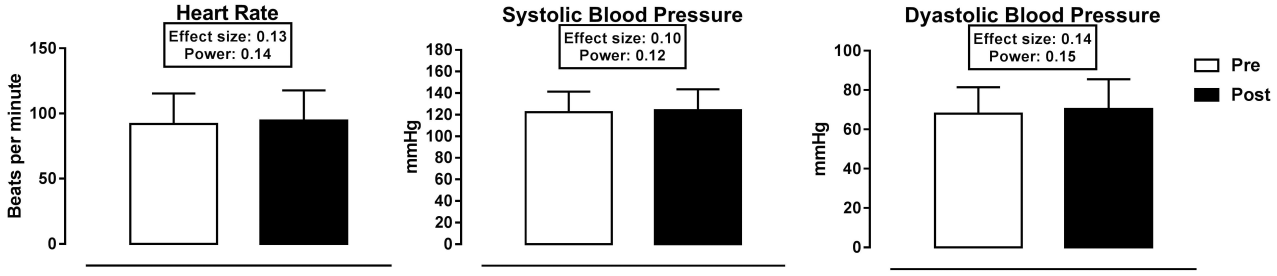

FIGURE 2 Hemodynamic parameters.

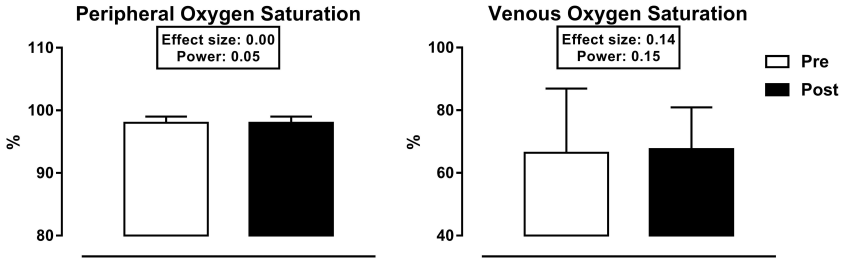

FIGURE 3 Peripheral and venous oxygen saturation.

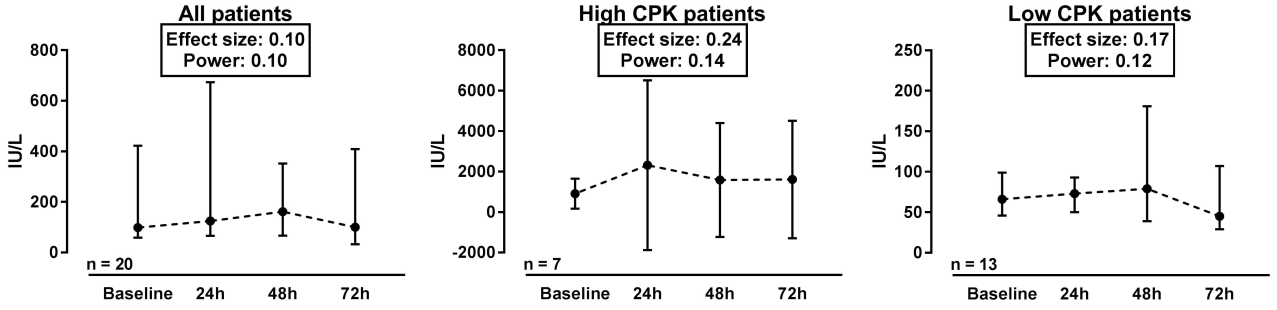

FIGURE 4 Creatine phosphokinase level.

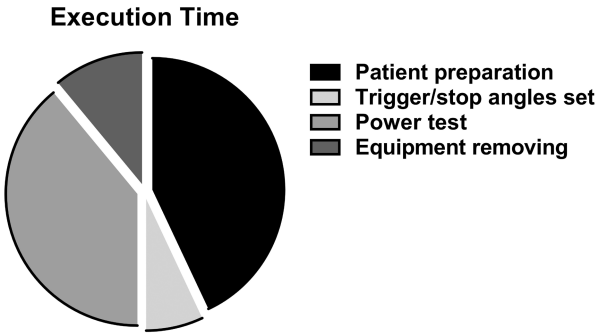

FIGURE 5 Execution time.

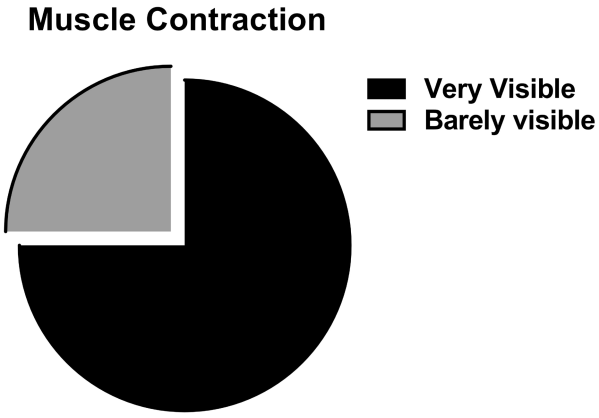

FIGURE 6 Muscle contraction visibility.

diagnostic protocol, looking for persistent clinical instability (persistent high lactate levels, peripheral or venous oxygen saturation alterations) caused by the diagnostic protocol. No clinical instability was detected, and after the protocol, all parameters presented the same values as before its execution.

Skeletal muscle damage can be indirectly assessed by blood CPK levels. The FES cycling-based muscular dysfunction diagnostic method promoted no muscle damage. Muscle tissue may be damaged following intense, prolonged activation as a result of both metabolic and mechanical factors. Indeed, rhabdomyolysis may result from direct and indirect damage to the muscle membrane, and may lead to leakage of intracellular muscle components into the extracellular fluid.[26] In cases of electrical stimulation-induced muscle damage, creatine kinase levels increase on the first day after stimulation.[27] In the present study, no significant variations in CPK levels were observed up to 72 h after the diagnostic protocol, even in high baseline CPK level patients. The three patients with the highest baseline CPK level (>1200 IU/L) had cardiac arrest up to 96 h prior to the assessment. Creatine kinase elevation is a common finding following successful cardiopulmonary resuscitation after cardiac arrest, and this elevation is related to both physical as well as electrical injury (defibrillation) sustained during cardiopulmonary resuscitation.[28,29]

The short time taken to perform the entire examination is important for adherence to the diagnostic method. As the FES cycling-based muscular dysfunction diagnostic method only requires a few minutes to perform, it can easily be carried out on a large number of patients on the same day. It can also be included as a routine assessment before each therapeutic FES-cycling session, providing muscular monitoring during the ICU stay.

All patients presented visible muscle contractions during the assessment. The majority of the patients presented very visible muscle contractions. Segers et al.[30] demonstrated that critically ill patients with sepsis, edema, or receiving vasopressors are less likely to respond to electrical stimulation with adequate muscle contraction. We achieved adequate muscle contraction in the present study, even in these situations. A possible reason for this was the FES parameters used. Another possible reason was the cycling movement, which changed muscle length and position, improving motor unit recruitment.[31]

This study has a number of limitations. (1) The sample size was not calculated, and a convenience sample of consecutive patients was used; (2) cardiac patients composed almost the entire sample, due to the profile of the hospital; and (3) limited muscle groups were used. The tibialis anterior muscle produces limited power output. This muscle was used because the equipment was developed mainly to be applied to critically ill patients, and in critical illness situations, tibialis anterior atrophy and ankle alterations are very common. When we used the diagnostic mode, the same muscle groups as the therapeutic mode were required. Thus, the equipment was developed for quadriceps (vastus lateralis and vastus medialis), hamstrings, and tibialis anterior muscle stimulation. According to the manufacturer, vasti and not rectus femoris were selected because they are more superficial and easily stimulated in legs with edema (a common situation in critically ill patients), and the hamstring is a large posterior muscle.

5 | CONCLUSION

The FES cycling-based muscular dysfunction diagnostic method is safe and feasible. No adverse events were reported, and no clinical instability was detected. Hemodynamic parameters, peripheral oxygen saturation, venous oxygen saturation, and blood lactate did not change after the diagnostic protocol. Muscle damage markers did not increase up to 72 h after the diagnostic protocol. The entire assessment took only a few minutes, and the majority of the patients presented very visible muscle contractions.

AUTHOR CONTRIBUTIONS

Thainá Figueiredo: Conceptualization, Methodology, Data curation, Formal analysis, Investigation, Writing—original draft, and Writing—review and editing. Murillo Frazão: Conceptualization, Methodology, Formal analysis, Investigation, Writing—original draft, and Writing—review and editing. Luís A. Werlang, Adelar Kunz and Maikel Peltz: Investigation, Writing—original draft, and Writing—review and editing. Veridiana C. Furtado, Edgar B. Júnior, Júlio M. Júnior and Rosane M. Silva: Methodology, Data curation, Investigation, and Writing—review and editing. Dário C. Sobral Filho: Conceptualization, Methodology, Formal analysis, Investigation, Writing—original draft, and Writing—review and editing.

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CONFLICT OF INTEREST STATEMENT

Luís A. Werlang, Adelar Kunz and Maikel Peltz are employees of INBRAMED, and they provided technical support for this research. INBRAMED did not provide any financial support for this research. Murillo Frazão is a technical consultant at INBRAMED.

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