Sleep-disordered breathing does not directly affect early cardiac rehabilitation efficacy after myocardial infarction

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ABSTRACT

Background: Sleep-disordered breathing (SDB) impairs exercise capacity after myocardial infarction (MI).

Aims: This study aimed to evaluate the impact of SDB on the efficacy of post-MI cardiac rehabilitation (CR).

Methods: The study evaluated consecutive patients up to 28 days after MI who participated in outpatient CR as part of the Polish Managed Care after Acute Myocardial Infarction program. Improvements in exercise capacity during CR were assessed based on changes in metabolic equivalents (MET) on the treadmill exercise stress test and distance on the six-minute walk test (6MWT). Home sleep apnea tests were performed to assess the presence/severity of SDB.

Results: The study included 254 patients aged 58.52 (10.51) and 39 (15.4%) women. Mild SDB with respiratory event index (REI) of 5–15 events/h was found in 82 patients (32.3%), moderate (REI of 15–30 events/h) in 54 (21.3%), and severe (REI >30 events/h) in 51 (20.1%). Obstructive sleep apnea was the dominant SDB type (89.8%). The severe-SDB group was older, with a higher body mass index and lower pre-CR exercise capacity than the others. After completing a four-week CR program, the entire group improved their MET (7.52 [2.26] to 9.02 [2.55]; P <0.001) and the 6MWT (645.00 m [518.00–814.00] to 786.500 m [638.00–998.50]; P <0.001). The severity of SDB did not influence the degree of improvement in MET and 6MWT in absolute values (P = 0.59 and P = 0.21) and percentages (P = 0.86 and P = 0.28).

Conclusions: Although severe SDB is one factor that negatively affects post-MI exercise capacity, it does not worsen the efficacy of early CR.

Key words: cardiac rehabilitation, exercise capacity, myocardial infarction, obstructive sleep apnea, sleep-disordered breathing

INTRODUCTION

Sleep-disordered breathing (SDB) can have a multifactorial impact on cardiovascular (CV) risk, cardiac function, and physical activity after myocardial infarction (MI). Both types of SDB, obstructive sleep apnea (OSA) and central sleep apnea, cause repetitive breathing cessation, nocturnal desaturation, and periodic arousals from sleep [1]. Arousals and fluctuations in the partial pressure of oxygen/carbon dioxide cause metabolic dysregulation, oxidative stress, inflammation, and endothelial dysfunction [1, 4]. Thus, they increase the risk of developing atherosclerotic CV diseases [4]. Particularly in patients after MI, nocturnal hypoxia and increased afterload during apneic events may lead to a disproportion between oxygen supply and demand and promote adverse left ventricular remodeling with the development of heart failure (HF) with reduced left ventricular ejection fraction (LVEF) [5]. In patients with moderate-to-severe OSA, despite successful revascularization, global and regional left ventricular systolic function recovery is poorer [6], infarct size and scar formation are more extensive, and the systolic sphericity index increases after three months of follow-up [7, 8]. Moreover,

WHAT'S NEW?

Severe sleep-disordered breathing, especially obstructive sleep apnea, was diagnosed in 20.1% of patients participating in the Polish Managed Care after Acute Myocardial Infarction program. Although severe sleep-disordered breathing was one factor that negatively affected baseline and endpoint exercise capacity in a cohort of 254 participants after myocardial infarction, it did not directly impact the improvement achieved through physical training during early cardiac rehabilitation.

these patients have a higher coronary artery calcification score/coronary plaque volume [9] and higher plaque instability/vulnerability [10], which may be associated with multivessel coronary artery disease (CAD) and an increase in the risk of CV events or death. Nocturnal intermittent hypoxia also worsens prognosis in participants with concomitant HF, ventricular arrhythmias, atrial fibrillation, hypertension, stroke, and metabolic disorders [4, 11–14]. In addition, physical activity in SDB individuals is impaired [15]. Sleep fragmentation leads to excessive daytime sleepiness, tiredness, and weariness [16]. Among cardiac rehabilitation (CR) participants, poor sleep quality is also associated with increased symptoms of depression, which may exacerbate perceived pain or fatigue [17]. Physical activity may also be reduced due to impaired inspiratory and peripheral muscle strength caused by hypoxia, impaired glycolytic/oxidative metabolism, and structural changes in muscle fibers [18]. A sedentary lifestyle, old age, obesity, and comorbidities are other causes of poorer mobility and reduced physical activity in SDB patients [19, 20].

It has been documented that CR based on physical exercises is an essential contribution to improving the health and prognosis of patients after an MI [21, 22]. All factors impeding its effective implementation should be eliminated before or immediately after the start of the CR program. The above data suggest the potential impact of SDB on exercise capacity and physical activity after MI. However, few data exist on whether SDB can directly impair CR's early efficacy. If it does, it would be crucial to include screening tests for SDB (particularly OSA) in patients referred to/starting a CR program, which is recommended by some scientific societies [23].

The purpose of this study is to evaluate the impact of SDB on CR's early efficacy in post-MI patients.

METHODS

Participants

We assessed consecutive patients referred for CR to the Department of Daily Cardiac Rehabilitation of the Upper Silesian Medical Center (Katowice, Poland). The study was conducted from May 2018 to January 2022, with a break during the COVID-19 pandemic for epidemiological reasons. The inclusion criteria were participation in the comprehensive Managed Care after Acute Myocardial Infarction (MC-AMI) program run by the Polish Cardiac Society, the National Health Fund, and the Ministry of Health. That program provides three to five weeks of in-hospital/outpatient CR starting within 14 days of discharge from the cardiology department after ST-/non-ST-segment elevation MI as well as one year of comprehensive cardiac care [22].

The study exclusion criteria were patients undergoing SDB treatment with positive airway pressure or intraoral devices. Patients with incomplete coronary revascularization, complex ventricular arrhythmia, relevant ischemic signs on treadmill exercise stress test (EST), or signs and symptoms of decompensated HF on admission were excluded from CR. Additionally, patients at high CV risk with LVEF of ≤35%, in ambulatory New York Heart Association functional class IV, or presenting physical limitations preventing them from engaging in standard exercises were redirected to inpatient CR.

The medical history of CV diseases, comorbidities (such as hypertension, atrial fibrillation, stroke history, diabetes, chronic kidney disease, hypercholesterolemia, and chronic obstructive pulmonary disease), and treatment applied were assessed. The course of acute MI was evaluated retrospectively based on available medical records. Anthropometric measurements, such as weight, height, body mass index (BMI), neck circumference, and waist size, were performed. The results of routine laboratory tests and echocardiography were included.

Evaluation of exercise capacity

On admission, we assessed exercise capacity using 1) symptom-limited submaximal EST according to the modified Bruce protocol, with a target heart rate (HR) of 85% of the age-predicted maximal HR and perceived exertion on the Borg scale of 13–15 ("somewhat hard" to "hard" on a scale from 6 to 20) [24]; and 2) a six-minute walk test (6MWT) [25]. Based on assessed exercise capacity, CV risk, and comorbidities, a cardiologist with experience in CR conducted qualification for models of physical exercise (A to C; Table 1) according to the recommendations of the Working Group of the Cardiac Rehabilitation and Exercise Physiology of the Polish Cardiac Society [21].

The CR program lasted 4 weeks. Qualified exercise physiologists supervised the exercise training and adapted it to individual patients' needs. After completing CR, symptom-limited submaximal EST and 6MWT were performed again. We evaluated CR efficacy based on the change in exercise capacity as an estimated amount of oxygen consumed in metabolic equivalents (MET, one MET ≈ 3.5 ml/kg/min) [26] and as distance in meters in the 6MWT.

Table 1. Cardiovascular risk and characteristics of the training parameters according to physical exercise models A, B and C

Model	A	B	C
Peak exercise capacity[a]	≥7 MET	5–6.9 MET	3–4.9 MET
CV risk	Low	Moderate	High
Exercise duration	60 min a day, 5 days a week	60 min a day, 5 days a week	45–60 min a day, 5 days a week
Training type and intensity		Aerobic continuous exercises	
	Aerobic continuous exercises on a cycle ergometer at the intensity of 50%–70% of the peak exercise capacity[a] or 11–14 points on the 6–20 Borg rating of perceived exertion scale	Aerobic continuous exercises on a cycle ergometer at the intensity of 50% of the peak exercise capacity[a] or 11–14 points on the 6–20 Borg rating of perceived exertion scale	Aerobic continuous exercises on a cycle ergometer at the intensity of 40%–50% of the peak exercise capacity[a] or 11–14 points on the 6–20 Borg rating of perceived exertion scale
		Resistance exercises	
	2–3 series of resistance exercises (10–15 repetitions with intensity from 30%–80% of 1-repetition maximum)	1–2 series of resistance exercises (10–15 repetitions with intensity from 30%–80% of 1-repetition maximum)	1–2 series of resistance exercises (5–10 repetitions with intensity from 30%–40% of 1-repetition maximum)
		General fitness	
	Flexibility, balance, and inspiratory muscle training	Flexibility, balance, and inspiratory muscle training	Flexibility, balance, and inspiratory muscle training

[a]Assessed on submaximal, symptom-limited exercise stress test on admission. Physical exercise was preceded by a 5-to-10-minute warm-up and followed by a 5-to-10-minute cool-down

Abbreviations: CV, cardiovascular; MET, metabolic equivalent

Polysomnography evaluation

On admission, we assessed the presence/severity of SDB using a portable polysomnography system (Alice NightOne, Philips Respironics, Murrysville, PA, US) for conducting home sleep apnea tests (HSAT) [27, 28]. The device identifies flow restrictions using a nasal cannula, hemoglobin oxygen saturation (SpO_2), HR using a pulse oximeter, and respiratory effort using a chest belt. HSAT recordings were analyzed manually according to the recommendations of the American Academy of Sleep Medicine [28]. We defined an apnea episode as a 90%–100% reduction in airflow that lasted ≥10 sec and a hypopnea episode as a 30% reduction in airflow that lasted ≥10 sec, leading to a decrease in SpO_2 by ≥4%. We classified apnea episodes with preserved respiratory muscle movements as obstructive and without respiratory effort as central. To assess SDB severity, we used the respiratory event index (REI), which is defined as the frequency of apneas and hypopneas per hour of recording. We defined REI of less than five as normal. We classified SDB with REI of 5–14 events/hour as mild, 15–30 events/hour as moderate, and >30 events/hour as severe. We assessed daytime sleepiness using the Epworth Sleepiness Scale [29].

Statistical analysis

The results were analyzed using MedCalc 20.210 software (MedCalc Software Ltd., Ostend, Belgium). The Kolmogorov–Smirnov test was used to explore normality of the distributions. Quantitative parameters were described by the arithmetic mean (standard deviation) or median (interquartile range). Qualitative data were expressed as numbers and frequency in percentages.

Univariable analysis of independent variables, such as the values of anthropometric, polysomnographic, and exercise capacity parameters per SDB severity, was carried out by the analysis of variance with ANOVA, with the Tuke–Kramer *post hoc* test, or the Kruskal–Wallis rank test with Conover's *post hoc* test, depending on the variables' distribution. Univariable analysis of dependent variables describing changes in exercise capacity over time (expressed as ΔMET and Δ6MWT in absolute values) was performed using Student's t-test for paired samples or the Wilcoxon paired order test as appropriate. Categorical comparisons were analyzed using the χ^2 test. The Pearson correlation coefficient, r, was computed to assess the relationships between SpO_2 and REI.

The significance of the influence of age, sex, BMI, LVEF, pre-CR exercise capacity, model of physical exercise, and SDB severity (i.e., REI, apnea duration, SpO_2 during sleep, and the percentage of total sleep time spent with oxygen saturation <90% [TST90]) on the post-CR submaximal exercise capacity was determined using multivariable linear regression. All candidate variables were entered in one step in the "full model" (enter method). In turn, in the stepwise method, variables were entered into the model sequentially if their associated significance level was <0.05. After entering each subsequent variable in the model and checking, all variables that became insignificant (P>0.10) were removed. In all tests performed, two-tailed P <0.05 was considered the limit of statistical significance.

Ethics approval and consent to participate

The study received a positive opinion from the Bioethical Committee of the Medical University of Silesia in Katowice, Poland (Approval No. KNW/0022/KB1/77/18 on September 25, 2018 and PCN 0022/KB1/77/I/18/20 on October 13, 2020). Informed consent was obtained from all subjects involved in the study.

Table 2. Characteristics of anthropometric parameters, coexisting diseases, and medications used per sleep-disordered breathing severity

Predictor	All patients n = 254 (100.0%)	Study subgroups according to SDB severity				*P*-value
		None (I) n = 67 (26.4%)	Mild (II) n = 82 (32.3%)	Moderate (III) n = 54 (21.3%)	Severe (IV) n = 51 (20.1%)	
Age, years, mean (SD)	58.52 (10.51)	55.93 (11.81)	58.49 (9.84)	58.81 (9.58)	61.67 (10.05)	0.03 I ≠ IV
Sex, male, n (%)	215 (84.6)	51 (76.1)	69 (84.1)	50 (92.6)	45 (88.2)	0.08
Body mass, kg, mean (SD)	88.94 (15.68)	84.50 (16.19)	89.27 (15.09)	90.34 (14.91)	92.76 (15.83)	0.03 I ≠ IV
BMI, kg/m^2, mean (SD)	29.59 (4.73)	28.17 (4.12)	29.58 (4.71)	30.10 (4.95)	30.90 (4.90)	0.01 I ≠ IV
Neck size, cm, mean (SD)	41.98 (3.50)	40.50 (3.87)	41.95 (3.05)	42.72 (2.93)	43.23 (3.60)	<0.001 I ≠ II I ≠ III I ≠ IV
Waist size, cm, mean (SD)	103.80 (11.29)	99.86 (11.48)	104,19 (10.48)	106.80 (12.28)	107.67 (9.31)	0.02 I ≠ IV
MI type (STEMI), n (%)	104 (40.9)	22 (32.8)	34 (41.5)	28 (51.9)	20 (39.2)	0.21
Number of coronary vessels with significant lesions, median (IQR)	2.0 (1.0–2.0)	2.0 (1.0–2.0)	2.0 (1.0-2.0)	1.0 (1.0–2.0)	2.0 (1.0–2.0)	0.91
LVEF, %, median (IQR)	55.00 (48.00–55.00)	55.00 (50.00–55.00)	52.00 (48.00–55.00)	55.00 (48.00–55.00)	55.00 (48.00–55.00)	0.58
Hypertension, n (%)	208 (81.9)	50 (74.6)	69 (84.1)	43 (79.6)	46 (90.2)	0.07
AF, n (%)	16 (6.3)	4 (6.0)	4 (4.9)	3 (5.6)	5 (9.8)	0.42
Stroke, n (%)	7 (2.8)	1 (1.5)	1 (1.2)	3 (5.6)	2 (3.9)	0.21
Diabetes, n (%)	56 (22.0)	10 (14.9)	17 (20.7)	14 (25.9)	15 (29.4)	0.25
CKD, n (%)	16 (6.9)	7 (10.9)	2 (2.6)	3 (6.2)	4 (9.1)	0.82
Hypercholesterolemia[a], n (%)	161 (69.1)	47 (73.4)	50 (64.9)	30 (62.5)	34 (77.3)	0.87
COPD, n (%)	12 (4.7)	3 (4.5)	4 (4.9)	5 (9.3)	0 (0.0)	0.55
Smoking status (lifetime), n (%)	132 (52.0)	35 (52.2)	42 (51.2)	31 (57.4)	24 (47.1)	0.76
Smoking status (current smokers), n (%)	83 (32.7)	23 (34.3)	25 (30.5)	17 (31.5)	18 (35.3)	0.93
Beta-blockers, n (%)	224 (88.2)	59 (88.1)	73 (89.0)	44 (81.5)	48 (94.1)	0.42
ACEi/ARB, n (%)	234 (92.1)	61 (91.0)	76 (92.7)	48 (88.9)	49 (96.1)	0.78
MRA, n (%)	69 (27.2)	13 (19.4)	24 (29.3)	16 (29.6)	16 (31.4)	0.41
Loop diuretics, n (%)	28 (11.0)	8 (11.9)	7 (8.53)	6 (11.1)	7 (13.7)	0.83

[a]Total cholesterol >5.0 mmol/l or lipid-lowering treatment

Abbreviations: ACEi, angiotensin-converting enzyme inhibitors; AF, atrial fibrillation; ARB, angiotensin receptor blockers; BMI, body mass index; CKD, chronic kidney disease with estimated glomerular filtration rate of <60 ml/min/1.73 m^2; COPD, chronic obstructive pulmonary disease; IQR, interquartile range; MI, myocardial infarction; MRA, aldosterone receptor antagonists; SD, standard deviation; SDB, sleep-disordered breathing; STEMI, ST-segment elevation myocardial infarction

RESULTS

Cohort characteristics

A total of 254 patients enrolled in the MC-AMI program constituted the study group, including 215 (84.6%) men and 39 (15.4%) women (Table 2). One hundred four patients (40.9%) were after ST-segment elevation MI, and the remaining patients (150, 59.1%) were after non-ST-segment elevation MI. The median time from acute MI to the start of CR was 13.0 days (interquartile range [IQR] 11.00–15.75).

For 245 participants hospitalized for acute MI in our center, complete MI data were retrospectively obtained. The median number of vessels with significant lesions was 2.0 (IQR 1.0–2.0). Single-vessel disease was diagnosed in 115 patients (46.9%). In 128 participants (52.2%), lesions were found in two or more vessels. All patients with multivessel disease (left main coronary artery or three or more affected vessels) had a consultation with a cardio surgery specialist, and percutaneous coronary intervention (PCI) was performed in those who were not eligible or did not consent to coronary artery bypass grafting. Primary PCI of the left anterior descending artery or diagonal branches was performed in 124 patients, the left circumflex artery or marginal branches in 77 patients, and the right coronary artery or its branches in 98 patients. Four patients had PCI of the left main coronary artery, 5 of the intermediate artery, and 1 of the left internal mammary artery-left anterior descending artery coronary bypass. A total of 169 patients (69.0%) underwent intervention on 1 coronary vessel, 63 participants (25.7%) had a two-stage intervention on 2 vessels, and 6 patients (2.4%) — PCI of 3 vessels. Conservative treatment was administered in 7 patients (3.1%) — in two cases, MI with non-obstructive coronary arteries was diagnosed; in four cases, the lesion concerned a narrow peripheral artery segment; in one case, the attempt to open chronic total occlusion failed. In one patient, angina recurred in the first 24 hours after the procedure, and PCI of the lesion located proximal to the previously placed stent was performed.

Overall, 106 patients (41.7%) qualified for physical exercise model A, 96 (37.8%) for model B, and 48 (18.9%) for model C. Four participants (1.6%) qualified for the C/D model and were characterized by an LVEF of <35% with maintained effort tolerance of ≥3 MET. They started the CR program from individual exercises with the intensity of a 10%–15% increase in the baseline HR.

SDB was found in 187 (73.6%) participants, with a median REI of 17.20 events/hour (9.38–30.88). Severe SDB

Table 3. Characteristics of home sleep apnea test parameters per sleep-disordered breathing severity

Predictor	All patients n = 254 (100.0%)	SDB severity				*P*-value
		None (I) n = 67 (26.4%)	Mild (II) n = 82 (32.3%)	Moderate (III) n = 54 (21.3%)	Severe (IV) n = 51 (20.1%)	
REI, events/h, median (IQR)	11.25 (4.90–25.40)	2.30 (1.53–3.78)	9.10 (6.90– 11.40)	20.25 (17.90–25.30)	41.90 (33.93–50.35)	<0.001
Participants with OSA/ CSA, n (%)	168 (66.1)/19 (7.5)	–	76 (92.7)/6 (7.3)	48 (88.9)/6 (11.1)	44 (86.3)/7 (13.7)	0.23
Average episode duration, sec, median (IQR)	21.90 (18.20–25.70)	18.80 (15.10–25.48)	22.90 (19.50–26.60)	21.30 (19.30–24.50)	22.70 (19.38–25.38)	0.004 I ≠ II I ≠ IV
Maximal episode duration, sec, median (IQR)	54.50 (42.00–74.00)	36.50 (22.75–55.75)	57.50 (43.38–73.63)	54.75 (46.00–76.50)	65.00 (56.00–88.38)	<0.001 I ≠ II I ≠ III I ≠ IV II ≠ IV III ≠ IV
Average SpO_2, %, median (IQR)	93.00 (92.00–94.00)	93.00 (92.00–95.00)	93.00 (92.00–94.00)	93.00 (92.00–94.00)	92.00 (92.00–93.75)	0.009 I ≠ IV II ≠ IV III ≠ IV
Minimal SpO_2, %, median (IQR)	85.00 (82.00–88.00)	88.00 (84.00–90.00)	85.00 (83.00–88.00)	84.00 (82.00–87.00)	82.00 (78.00–85.00)	<0.001 I ≠ II I ≠ III I ≠ IV II ≠ IV III ≠ IV
TST90, %, median (IQR)	1.57 (0.10–7.19)	0.35 (0.00–2.84)	0.90 (0.07–3.26)	2.92 (0.35–9.09)	7.19 (1.80–26.62)	<0.001 I ≠ III I ≠ IV II ≠ III II ≠ IV III ≠ IV
Sleepiness on ESS, points, median (IQR)	5.0 (3.0–8.0)	4.0 (2.3–7.0)	5.0 (3.0–9.0)	5.0 (4.0–9.0)	4.0 (3.0–8.0)	0.26

Abbreviations: CSA, central sleep apnea; ESS, Epworth Sleepiness Scale; OSA, obstructive sleep apnea; REI, respiratory event index; SpO_2, arterial oxygen saturation estimated by pulse oximetry; TST90, the percentage of total sleep time spent with oxyhemoglobin saturation below 90%; other — see Table 2

was diagnosed in 51 (20.1%), moderate in 54 (21.3%), and mild in 82 patients (32.3%) (Table 3). Among SDB patients, OSA was dominant in 167 (89.8%). However, in patients with LVEF <40%, central sleep apnea was more common (8 [36.4%]) than in others (12 [7.2%]); *P* <0.001. Desaturation correlated with the REI (for minimal SpO_2, correlation coefficient r –0.303; 95% confidence interval [CI] for r –0.411 to –0.186, *P* <0.001; for TST90, r = 0.242, 95% CI for r 0.123 to 0.355; *P* <0.001). None of the participants diagnosed with OSA started positive airway pressure therapy during the CR program.

The severe-SDB group was older, with a higher BMI and waist size (Table 2). However, it did not differ regarding the prevalence of ST-segment elevation MI (*P* = 0.21), median number of affected coronary vessels (*P* = 0.01), New York Heart Association class (*P* = 0.28), LVEF (*P* = 0.52), or comorbidities. Most patients with severe SDB were qualified for physical exercise model B (24, 47.1%) and C (13, 25.5%) because of their lower pre-CR exercise capacity (Table 1 and 4). In contrast, most patients with no/mild/moderate SDB exercised according to models A (93, 45.81%) and B (72, 35.5%).

Assessment of CR efficacy

Six patients did not complete CR. Four had bradycardia (sinus node dysfunction in 3 and 2-degree atrioventricular block in 1), so they were redirected to the cardiology department. One patient had recurrent MI and was hospitalized in another center (detailed data not available). One patient discontinued CR due to a recurrent urinary tract infection.

Following four weeks of CR, the entire group improved in MET from 7.52 (2.26) to 9.02 (2.55) and 6MWT distance from 645.00 m (518.00–814.00) to 786.500 m (638.00–998.50), *P* <0.001 for both. Improvements in exercise capacity were evident in the groups without SDB and with SDB, ranging from mild to severe (Table 4).

The severity of SDB did not influence the degree of improvement assessed as ΔMET and Δ6MWT in absolute values (*P* = 0.59 and *P* = 0.21) and percentages (*P* = 0.86 and *P* = 0.28) (Table 4). However, after completing CR, the severe-SDB group still had a lower exercise capacity than the no-SDB or mild-SDB groups. Table 5 summarizes all factors considered to affect post-CR exercise capacity. The parameters that had an independent impact on post-CR submaximal exercise capacity in multivariable analysis were age, female sex, BMI, and pre-CR exercise capacity, but SDB severity did not.

We graphically summarized the assumptions and results of the study in Figure 1.

DISCUSSION

We evaluated the direct impact of SDB on the efficacy of outpatient CR in the early period after MI. We measured submaximal exercise capacity in 254 participants by symptom-limited EST and 6MWT at the start and end of CR. We applied HSATs to assess SDB. In our cohort of post-MI patients, the percentage of SDB with REI ≥5 events/hour

Table 4. Characteristics of the maximal exercise capacity parameters per sleep-disordered breathing severity on admission and on discharge

Predictor		SDB severity				*P*-value[a]
		None (I) n = 67 (26.4%)	Mild (II) n = 82 (32.3%)	Moderate (III) n = 54 (21.3%)	Severe (IV) n = 51 (20.1%)	
6MWT, m, median (IQR)	On admission	666.50 (525.50–867.50)	657.00 (564.00–806.00)	668.50 (496.50–860.00)	552.00 (457.00–686.50)	0.03 I ≠ IV II ≠ IV III ≠ IV
	On discharge	789.00 (704.50–1050.75)	854.00 (680.50–990.50)	821.00 (639.25–1037.25)	667.00 (531.00–881.00)	0.007 I ≠ IV II ≠ IV III ≠ IV
	P-value[b]	<0.001	<0.001	<0.001	<0.001	
	Δ (m)	160.00 (81.75–272.00)	115.00 (60.75–202.50)	153.00 (78.25–239.25)	111.00 (46.50–180.25)	0.21
	Δ (%)	24.72 (12.37–47.15)	15.29 (7.92–31.25)	24.20 (12.64–32.93)	19.93 (7.13–32.73)	0.28
MET, mean (SD)	On admission	7.75 (2.27)	7.87 (2.12)	7.51 (2.45)	6.67 (2.08)	0.02 I ≠ IV II ≠ IV
	On discharge	9.41 (2.61)	9.38 (2.20)	8.98 (2.78)	8.04 (2.52)	0.02 I ≠ IV II ≠ IV
	P-value[b]	<0.001	<0.001	<0.001	<0.001	
	Δ (MET)	1.63 (1.28)	1.53 (1.49)	1.65 (1.15)	1.33 (0.94)	0.59
	Δ (%)	23.03 (19.23)	23.96 (32.48)	24.15 (22.93)	20.47 (14.16)	0.86
MHR (bpm), mean (SD)	On admission	124.78 (18.39)	120.59 (14.98)	117.42 (16.22)	116.12 (17.50)	0.03 I ≠ IV
	On discharge	136.72 (19.17)	130.17 (18.27)	124.27 (18.64)	124.73 (20.13)	0.002 I ≠ III I ≠ IV
	P-value[b]	<0.001	<0.001	0.001	<0.001	
	Δ (bpm)	12.08 (15.56)	10.13 (14.20)	7.60 (15.77)	8.31 (13.18)	0.38
MHR% (%), mean (SD)	On admission	76.87 (9.93)	75.99 (8.93)	74.46 (9.14)	76.04 (9.51)	0.58
	On discharge	84.98 (9.50)	83.97 (11.66)	79.10 (8.94)	80.83 (11.48)	0.01 I ≠ III
	P-value[b]	<0.001	<0.001	<0.001	0.008	
	Δ (%)	8.18 (9.40)	7.94 (10.39)	5.08 (8.15)	4.67 (11.75)	0.13
Perceived exertion on Borg's scale, median (IQR)	On admission	12.0 (9.0–14.0)	13.0 (10.0–14.0)	13.0 (10.0–14.0)	14.0 (10.0–14.0)	0.16
	On discharge	11.0 (8.0–14.0)	12.0 (9.0–14.0)	12.0 (10.0–14.0)	14.0 (10.0–15.0)	0.11
	P–value[b]	0.09	0.04	0.86	0.81	
	Δ (points)	0.0 (–2.0 to 1.0)	0.0 (–1.25 to 0.0)	0.0 (–1.0 to 1.0)	0.0 (–2.0 to 2.0)	0.36

[a]*P*-value for differences between subgroup per sleep-disordered breathing severity. [b]*P*-value for differences between exercise capacity parameters on admission vs. on discharge. ΔChange of parameter value between admission and discharge

Abbreviations: MHR, maximal heart rate; MHR%, percentage of the age-predicted maximal heart rate; 6MWT, six-minute walk test; other — see Tables 1 and 3

was 73.6% (most often OSA), including severe SDB, which was diagnosed in 20.1% of participants. We found that the estimated amount of oxygen consumed in MET obtained in the EST and distance on 6MWT in the severe-SDB patients, who were older and had a higher average BMI, were lower than in those without SDB or with mild SDB, both before and after CR. Despite this, we observed improved exercise capacity achieved through physical training in all SDB groups. Furthermore, the severity of SDB did not influence the degree of improvement assessed as ΔMET and Δ6MWT and post-CR exercise capacity after adjusting for age, sex, BMI, and pre-CR exercise capacity. We concluded that although severe SDB is one factor that negatively impacts exercise capacity after MI, it does not worsen the efficacy of early CR.

Screening questionnaires and polysomnographic examinations confirmed that up to 80% of individuals with CAD referred for CR are at high risk for SDB [29–33]. As in our cohort, OSA is the most common SDB type in post-MI patients with preserved LVEF [5, 32], with a higher prevalence in older obese/overweight men [2, 30]. SDB is closely related to a specific risk profile and multi-morbidity. Male sex, age over 35, overweight/obesity, high blood pressure, diabetes, hyperlipidemia, and smoking are the main risk factors for both OSA and CAD [2, 30, 34]. However, the incidence of moderate to severe OSA in post-MI patients ranges from 35%–65% [32], which is higher than 15%–35% counted in the general population of middle-aged and older adults [2, 35].

SDB, especially OSA, is one of the factors worsening cardio-respiratory fitness [15, 36]. An increase of one unit in log-transformed apnea-hypopnea index (AHI; apnea/hypopnea episodes per hour of sleep) translates into a decrease in peak oxygen uptake by 3.20 ml/kg/min in the cardiopulmonary exercise test [36]. The effect of moderate-to-severe OSA on peak oxygen uptake is visible regardless of age [15]. The inversely proportional relationship between the maximal sleep apnea duration or the AHI/REI and estimated oxygen consumption in MET on EST or distance on 6MWT was also documented [19, 33, 37–39]. In our study, the

Table 5. Factors affecting exercise capacity after completing the rehabilitation program expressed in metabolic equivalents and the distance in 6-minute walk tests in univariable and multivariable regression

Exercise capacity expressed in metabolic equivalents						
	Full model $P<0.001$; Nagelkerke $R^2=0.84$			Stepwise regression $P<0.001$; Nagelkerke $R^2=0.82$		
Predictor	Coeff.	Standard error	*P*-value	Coeff.	Standard error	*P*-value
Age, years	–0.05	0.01	<0.001	–0.040	0.01	<0.001
Sex, female	–0.503	0.18	0.006	–0.524	0.18	0.003
BMI, kg/m^2	–0.069	0.02	<0.001	–0.053	0.02	<0.001
MI type (STEMI)	0.093	0.14	0.53	Did not enter the model[a]		
LVEF, %	0.017	0.01	0.15	Did not enter the model[a]		
MET on admission	0.789	0.07	<0.001	0.830	0.04	<0.001
Exercise model[b]	–0.072	0.20	0.72	Did not enter the model[a]		
REI, events/hour	0.0007	0.003	0.84	Did not enter the model[a]		
Maximal episode duration, s	0.002	0.002	0.25	Did not enter the model[a]		
Minimal SpO_2, %	–0.006	0.02	0.68	Did not enter the model[a]		
TST90, %	0.001	0.01	0.85	Did not enter the model[a]		
Exercise capacity expressed as a distance in 6-minute walk test						
	Full model $P<0.001$; Nagelkerke $R^2=0.82$			Stepwise regression $P<0.001$; Nagelkerke $R^2=0.79$		
Predictor	Coeff.	Standard Error	*P*-value	Coeff.	Standard Error	*P*-value
Age, years	–5.517	0.98	<0.001	–4.934	0.94	<0.001
Sex (female)	–40.416	20.97	0.055	–38.129	20.17	0.06
BMI, kg/m^2	–8.562	1.94	<0.001	–7.152	1.63	<0.001
MI type (STEMI)	11.526	16.91	0.50	Did not enter the model[a]		
LVEF, %	1.555	1.48	0.29	Did not enter the model[a]		
6MWT distance on admission, m	0.816	0.07	<0.001	0.840	0.05	<0.001
Exercise model[b]	1.539	19.76	0.94	Did not enter the model[a]		
REI, events/hour	–0.475	0.40	0.24	Did not enter the model[a]		
Maximal episode duration, s	0.374	0.31	0.24	Did not enter the model[a]		
Minimal SpO_2, %	–1.633	1.70	0.33	Did not enter the model[a]		
TST90, %	–0.055	0.47	0.91	Did not enter the model[a]		

[a]Entry threshold $P<0.05$, exit threshold $P>0.1$. [b]Physical exercise model A = 1, model B = 2, model C = 3

Abbreviations: Coeff., coefficient; other — see Tables 1–4

severe-SDB group had 13.9% lower pre-CR performance in MET than those without SDB.

Our cohort, despite a high percentage of participants with SDB, achieved a significant increase in post-CR exercise capacity, with a mean difference in MET of 1.54 (95% CI, 1.38–1.70; $P<0.001$) and a median difference in 6MWT distance of 143.00 m (95% CI, 126.50–162.00; $P<0.001$). The direct outcomes of CR do not differ from those described by other authors for post-MI patients [40]. We noticed that the degree of improvement did not depend on SDB severity (ΔMET 1.63 [23.0%], 1.53 [24.0%], 1.65 [22.9%], and 1.33 [20.5%] for the non-SDB, mild-SDB, moderate-SDB, and sever-SDB groups). However, the severe-SDB group had lower post-CR exercise capacity, with a difference of 14.6% in MET between the severe-SDB group and the non-SDB group.

Sonners et al. [33] also compared exercise capacity between participants with OSA (AHI >5 events/h) and those without OSA before and after a three-week CR program. In that cohort, untreated OSA patients had 11.7% lower pre-CR (3.3 MET [2.9–4.5] vs. 3.9 MET [3.1–5.0]) and 11.9% lower post-CR (5.3 MET [4.0–7.0] vs. 6.0 MET [4.6–7.6]) exercise capacity compared to the control group. Despite these differences, the mean improvement assessed in both groups during CR was similar and amounted to 1.8 MET (0.6–2.6) vs. 2.0 MET (0.9–3.0). In 105 post-MI patients, Hupin et al. [31] showed a 15.4% post-CR improvement in peak oxygen uptake in patients with mild OSA (3.3 ml/min/kg [6.4]), 16.8% with moderate OSA (3.4 ml/min/kg [6.4]), and 24.9% with severe OSA (5.0 ml/min/kg [7.0]) with no difference in improvement between groups. In turn, Spielmanns et al. [41], in a large cohort after cardiac surgery, documented that even moderate-to-severe symptomatic OSA did not affect a clinically significant improvement (defined as a change of >45 m in 6MWT distance) after 3 weeks of CR. Conversely, OSA with AHI >15 events/hour did not affect pre-CR exercise capacity.

In a multivariate analysis, we confirmed that the predictors of poorer post-CR performance were, among others, lower pre-CR exercise capacity, older age, and higher BMI — all of which characterized the severe-SDB individuals. In our previous article [38], we showed that

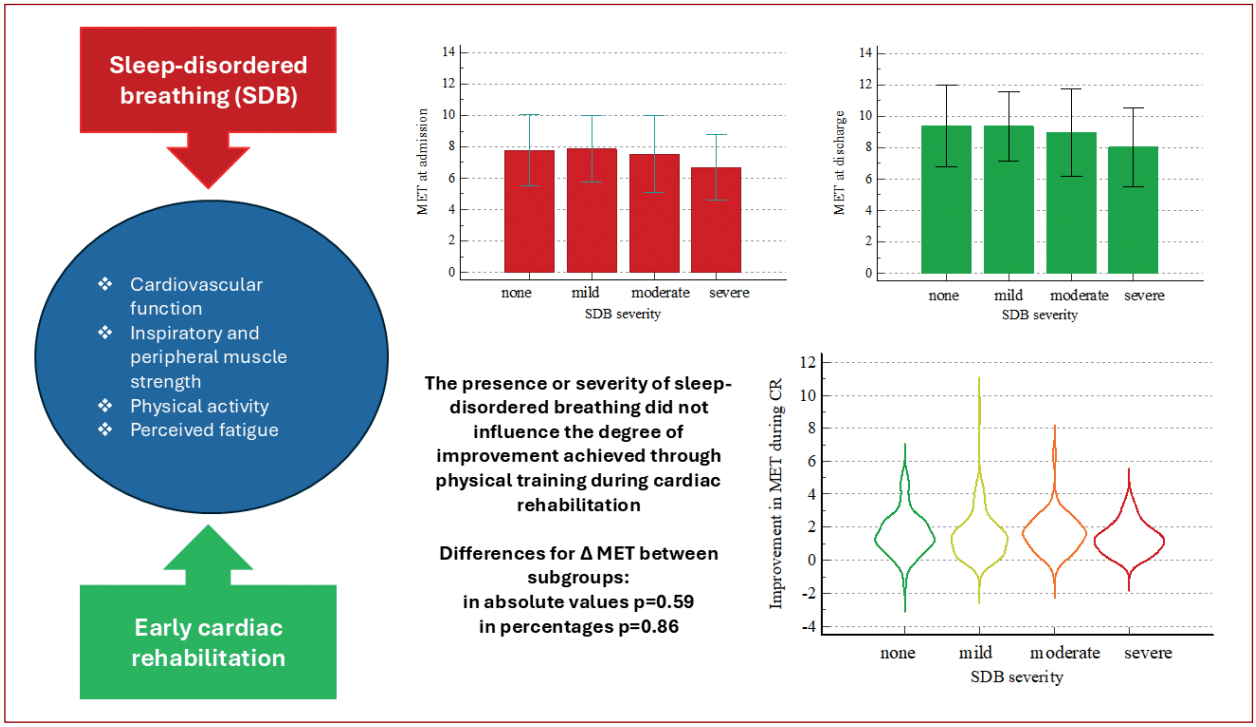

Figure 1. Central illustration. The improvement in metabolic equivalent (MET) illustrates the effiectiveness of the early cardiac rehabilitation program in patients with varying degrees of sleep-disordered breathing

the impact of risk factors that commonly accompany SDB occurrence and poor activity/impaired exercise capacity, such as older age, higher BMI, and LV systolic dysfunction, may have a more substantial impact than SDB itself on pre-CR exercise capacity. Loo et al. [39] arrived at similar conclusions regarding pre-CR exercise capacity assessed in the 6MWT based on the multiple linear regression analysis, which showed that older age, female sex, and a larger waist circumference were independently associated with poorer 6MWT results. Therefore, it is essential for a CR program to comprehensively manage risk factors, especially sedentary lifestyle and obesity, to prevent both re-MI and SDB [4, 21].

Type 1 polysomnography (PSG), performed in a sleep laboratory, is the gold standard for SDB diagnostics [28]. However, in many areas of medicine, the burden of diagnostics is shifting to outpatient departments, which allows for limiting the length of hospitalization, freeing up hospital beds, and reducing healthcare costs. Another reason is the long waiting time for hospital diagnostics, such as SDB diagnostics. According to NHF data [42], in the Silesian Voivodeship (Poland), the average waiting time for an available hospital bed in pulmonology departments performing in-laboratory PSG is about 250 days. On the other hand, the recommendations on screening for OSA in patients with CV diseases are becoming increasingly widespread [4]. Currently, they apply to patients with resistant/poorly controlled hypertension, pulmonary hypertension, or recurrent atrial fibrillation. The sleep study should also be considered when SDB signs/symptoms are present in patients with symptomatic HF, tachy-brady syndrome, ventricular tachycardia, survivors of sudden cardiac death, or patients after stroke. Moreover, patients after MI, with nocturnal angina or arrhythmias/appropriate shocks from implanted cardioverter-defibrillator, are listed as being especially likely to have comorbid SDB. This means that the problem of the lack of technical possibilities to perform in-laboratory PSG in a reasonable time will increase. The optimal solution to improve patient care seems to be to include HSAT to support the diagnosis in symptomatic patients with a high risk of OSA, including as part of CR.

In patients with a high pre-test probability, the REI has been shown to have a sensitivity of 79% and specificity of 79% for detecting moderate-to-severe OSA [43]. A current meta-analysis of 24 studies (n = 1644 participants) by Cagle et al. [44] confirms a strong correlation between AHI (r = 0.96; P <0.001), oxygen desaturation index (r = 0.75; P = 0.031), and lowest SpO_2 (r = 0.85; P = 0.03) measured by HSAT compared to in-laboratory PSG. Furthermore, studies do not indicate differences in the clinical outcomes of patients treated based on HSAT compared to PSG type I results [45]. The 2023 International Consensus Statement on Obstructive Sleep Apnea [46] reported up to 18% false negative results of HSAT compared to in-laboratory PSG in high-risk patients, with an underestimation of AHI of around 10%. The risk of underestimating the results is inversely related to disease severity (the highest in patients with mild, thus, less clinically significant OSA) [43]. This risk can be effectively managed using surveys assessing pre-test probability, scoring HSAT by trained research polysomnologists, and manual editing of total recording

time [27, 28]. Instead of total recording time, defined as the period between lights off and lights on, they should assess a time close to total sleep time, that is, without the periods when the participant is likely to awake, as evidenced by artifact, movement, and characteristic changes in heart rate and breathing [47]. In the case of negative or technically inadequate HSAT in patients with a high pre-test probability of moderate-to-severe OSA, in-laboratory PSG should be performed to exclude a false negative study [27, 28]. Furthermore, in HF patients, in-patient PSG should be preferred to diagnose the type of SDB and propose appropriate therapy [28].

Limitations of the study

The study was conducted in a defined group of patients after MI, i.e., those eligible for outpatient CR, which excluded the participation of patients with a complicated post-MI period, severe left ventricular systolic dysfunction, or after coronary artery bypass grafting. The lower incidence of CAD and MI among women compared to men in the general population made women poorly represented in the study group. The presented study used EST and 6MWT, standard qualification tests approved for Poland's CR program, instead of the cardiopulmonary exercise test to assess exercise capacity [21]. Obtaining the target HR was difficult due to most participants using beta-blockers. Therefore, decisions to complete the EST were based primarily on assessing perceived exertion on the Borg scale and coronary symptoms (symptom-limited EST). We used HSAT to assess SDB severity because it can be applied in an outpatient setting. However, all patients had the probability of OSA/sleepiness severity evaluated based on anthropometric measurements and surveys. Manual scoring HSAT was performed by trained research polysomnologists with experience in over 500 HSAT studies. We repeated or rejected inconclusive or technically inadequate tests to ensure the reliability of the research.

Article information

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