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Original Article

Obstructive sleep apnoea is associated with the development of diastolic dysfunction after myocardial infarction with preserved ejection fraction

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ABSTRACT

Background: Left ventricular diastolic dysfunction is a predictor of adverse outcome after acute myocardial infarction (AMI). We aimed to test if sleep-disordered breathing (SDB) contributes to the development of diastolic dysfunction in patients with preserved left ventricular ejection fraction after AMI.

Method: Patients with AMI, percutaneous coronary intervention and an ejection fraction ≥50% were included in this sub-analysis of a prospective observational study. Patients with AMI (n = 41) underwent cardiovascular magnetic resonance imaging (volume–time curve analysis) to define diastolic function by means of the normalised peak filling rate [nPFR; (end diastolic volume/second)]. In patients with AMI, the nPFR was assessed within <5 days and three months after AMI. Patients with AMI were stratified in patients with (apnoea-hypopnoea index, AHI ≥15/h) and without (AHI <15/h) SDB as assessed by polysomnography.

Results: At the time of AMI, the nPFR was similar between patients with and without SDB (2.90 ± 0.54 vs. 3.03 ± 1.20, p = 0.662). Within three months after AMI, diastolic function was significantly lower in patients with SDB than in patients without SDB (ΔnPFR: −0.83 ± 0.14 vs. 0.03 ± 0.14; p < 0.001; ANCOVA, adjusted for baseline nPFR). In contrast to central AHI, obstructive AHI was associated with a lower nPFR three months after AMI, after accounting for established risk factors for diastolic dysfunction [multiple linear regression analysis, B (95%CI): −0.036 (−0.063 to −0.009), p = 0.011].

Conclusion: Our data indicate that obstructive sleep apnoea impairs diastolic function early after myocardial infarction.

1. Introduction

Left ventricular (LV) diastolic dysfunction is a predictor of adverse outcome after acute myocardial infarction (AMI), even if LV systolic function is preserved [1–3]. LV filling characteristics are

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Abbreviations

AHI	apnoea-hypopnoea index
cAHI	central apnoea-hypopnoea index
CMR	cardiac magnetic resonance imaging
CSA	central sleep apnoea
EF	ejection fraction
LV	left ventricular
oAHI	obstructive apnoea-hypopnoea index
OSA	obstructive sleep apnoea
SD	standard deviation
SDB	sleep-disordered breathing

Brief summary

Patients with myocardial infarction and preserved ejection fraction underwent cardiovascular magnetic resonance imaging to define diastolic function within <5 days and three months. At baseline, diastolic function was similar between patients with and without sleep-disordered breathing (SDB). After three months, diastolic function was lower in patients with SDB than in patients without SDB, especially for patients with obstructive AHI. Our data indicate that obstructive sleep apnoea impairs diastolic function early after myocardial infarction.

altered in the subacute phase after AMI, but the mechanisms altering diastolic function after AMI are not yet fully understood.

Sleep-disordered breathing (SDB) adversely affects cardiac afterload [4], cardiac remodelling [5] and infarct size after AMI [6–8]. Patients with SDB have a lower myocardial salvage index and impaired healing after MI, resulting in larger scar areas [6]. Obstructive sleep apnoea (OSA), but not central sleep apnoea (CSA), is associated with adverse spherical remodelling after MI [8,9]. Cross-sectional analyses of a primary care cohort with cardiovascular risk factors showed that OSA is associated with the severity of LV diastolic dysfunction [10]. Proposed mechanisms of SDB and specifically OSA affecting LV diastolic function include several mechanical, neurohumoral, inflammatory, endothelial and oxidative effects as well as intermittent ischemia [11,12]. Strikingly, these mechanisms are also activated after MI, and it is likely that concomitant SDB exerts additional deleterious effects during this vulnerable phase of myocardial healing. Yet, the effects of SDB on left ventricular diastolic function after AMI are not yet known. Cardiac magnetic resonance (CMR) imaging allows the study of LV systolic and diastolic function by volumetric filling curves, which are used to assess diastolic dysfunction [13–15]. Therefore, we hypothesised that SDB and specifically OSA contribute to the development of diastolic dysfunction in patients with AMI and preserved left ventricular ejection fraction within three months after AMI.

2. Methods

2.1. Study design

To test our hypothesis, we performed a sub-analysis of a prospective observational study conducted at the University Medical Centre Regensburg, Germany (March 2009 to March 2012). The detailed design has been previously published elsewhere [6]. For this analysis, we have only included patients with a preserved left ventricular ejection fraction of ≥50% at the time of AMI. These patients were stratified for the presence of SDB (Fig. 1).

2.2. Patients

Briefly, patients aged 18–80 years with first-time AMI (ST elevation on electrocardiogram or complete occlusion of coronary artery in non-ST elevation MI) and successful percutaneous coronary intervention were eligible for this study if their symptoms had lasted less than 24 h. Known sleep-disordered breathing (both treated and untreated) was an exclusion criterion. Eligible patients had to have received an overnight in-laboratory sleep study (polysomnography) and a CMR scan within three to five days after percutaneous coronary intervention and three months after AMI. Clinical management and medication were at the discretion of the responsible physician according to current practice and guidelines. This sub-analysis only included patients with preserved ejection fraction (EF ≥ 50%) at baseline.

The study protocol was reviewed and approved by the local institutional Ethics Committee (Regensburg, 08–151) in accordance with the Helsinki Declaration of Good Clinical Practice. Written informed consent had been obtained from all patients prior to enrolment.

2.3. Polysomnography

Polysomnography was performed in all subjects using standard polysomnographic techniques (Alice System; Respironics, Pittsburgh, PA, USA) [16,17]. The sleep lab is located on the cardiology ward of the hospital to which participants were admitted with AMI. The median times to baseline and follow-up polysomnography after AMI were 3 days and 12 weeks, respectively. Respiratory efforts were measured with the use of respiratory inductance plethysmography, and airflow was measured using a nasal pressure cannula. Sleep stages, arousals, and apnoeas and hypopnoeas were determined according to the criteria of the American Academy of Sleep Medicine [18] by one experienced sleep technician blinded to the clinical data. Apnoea was defined as a cessation of inspiratory airflow for ≥10 s. Hypopnea definition with ≥30% airflow reduction and ≥4% desaturation was used [18], since hypopneas with a desaturation of at least 4% are independently associated with cardiovascular disease [19].

In addition, hypopnoeas were classified as obstructive if there was an out-of-phase motion of the ribcage and abdomen or if airflow was limited. To achieve optimal distinction between obstructive and central hypopnoea without using an oesophageal balloon, we applied additional criteria, such as flattening, snoring, paradoxical effort movements, arousal position relative to hypopnoea and associated sleep stage [rapid eye movement (REM)/non-REM] [19]. The apnoea-hypopnea index (AHI) was defined as the number of apnoeas and hypopnoeas per hour of sleep. Accordingly, the severity of OSA and CSA was expressed as the number of obstructive apnoeas and hypopnoeas (obstructive AHI) as well as the number of central apnoeas and hypopnoeas (central AHI) per hour of sleep.

2.4. Cardiac magnetic resonance acquisition protocol

CMR scans were carried out with a clinical 1·5-T scanner (Avanto, Siemens Healthcare Sector, Erlangen, Germany) using a 32-channel phased-array receiver coil. Ventricular function was analysed by means of steady-state free precession cine images in standard short axis planes and long axis planes (SSFP, trueFISP;

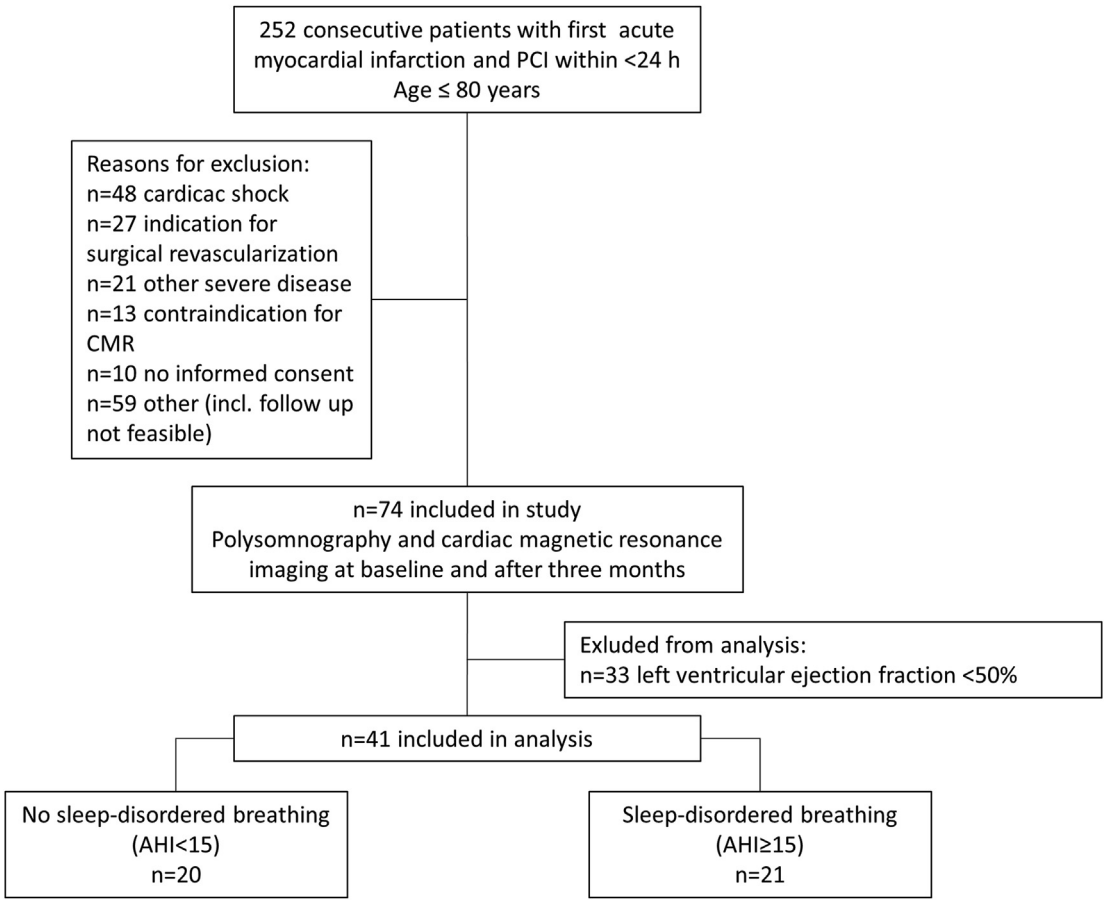

Fig. 1. Study flow-chart. PCI – percutaneous coronary intervention.

slice thickness 8 mm; inter-slice gap 2 mm; repetition time 60.06 msec; echo time 1.16 s; flip angle 60°; FOV 300 × 300 mm, matrix size 134 × 192; readout pixel bandwidth 930 Hz/pixel).

2.5. Cardiac magnetic resonance image analysis

LV volumes were calculated by assessing four-dimensional guide-point modelling ventricular function (4DVF Syngo Argus, version B15; Siemens Healthcare Sector, Erlangen, Germany) [20]. This analysis provided the time-varying course of the LV volume during the cardiac cycle (25 images per cardiac cycle). The peak filling rate and the time to peak filling rate were determined in the phase of left ventricular diastolic filling (Fig. 2 A + B). To minimize the dependency of the peak flow rate on the end-diastolic volume, we also calculated the normalised peak filling rate (nPFR) as described previously [15,21,22].

2.6. Statistical analysis

Quantitative data are expressed as mean ± standard deviation (SD). Categorical data are expressed as frequencies with percentages. Quantitative variables were compared with the independent-sample parametric (unpaired Student's) t-test. For the comparison of changes in CMR variables between the two groups, analysis of covariance (ANCOVA) was used including the group as the main factor and the baseline value of the outcome variable as a covariate to adjust for baseline differences. Categorical variables were compared using the exact unconditional Pearson Chi-Squared statistics. Associations between diastolic function and SDB were described using linear regression analysis. Multiple linear regression analyses were performed to identify predictors of diastolic function (at baseline and three months after AMI). Known potential confounders and risk factors [21], which may affect diastolic function, were entered: age, sex, body mass index and hypertension. For graphical illustrations, dot plots with 95% confidence interval (95% CI) and scatter plots with regression lines were used. All reported p-values are two-sided, and a p-value of $\leq$0.05 is considered to be statistically significant. Data entry and calculations were made with the software package SPSS 26.0 (Chicago, EUA) and Graphpad Prism (Version 6.01 for Windows, GraphPad Software, La Jolla California USA).

4. Results

4.1. Characteristics of myocardial infarction with or without sleep-disordered breathing

Patients with AMI were stratified into two groups: no SDB (AHI <15/h) and SDB (AHI ≥15/h). The two groups were similar with respect to age, sex, risk factors for coronary artery disease and haemodynamic findings (Table 1). Patients without SDB had a significantly lower body-mass index than patients with SDB (26.7 ± 2.5 vs. 29.3 ± 3.4; p = 0.008). Both groups were similar in the time from symptom onset to revascularisation, infarct-related artery, TIMI flow before or after percutaneous coronary intervention, thrombus aspiration and use of glycoprotein IIb/IIIa inhibitors during percutaneous coronary intervention. Per definition, mean AHI was significantly lower in the group without SDB than in the

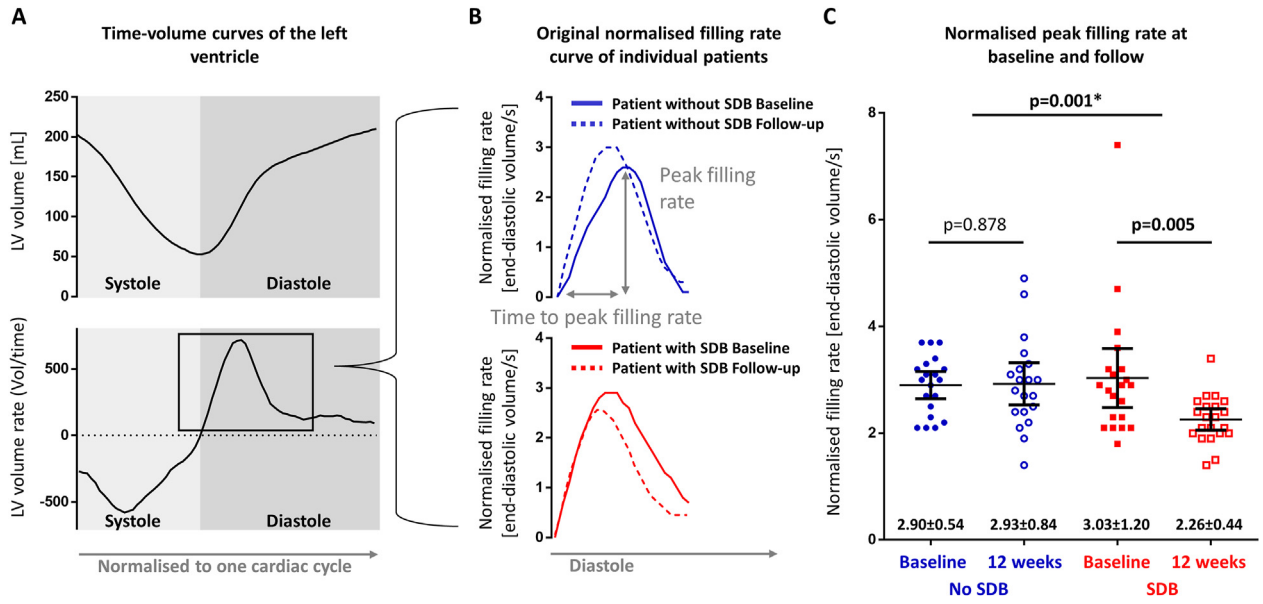

Fig. 2. Diastolic function is impaired three months after MI in patients with SDB. A) Original recording of a time–volume curve (above) and a LV volume rate curve (below) derived from CMR cine data. To allow adequate comparison of time–volume curves of different CMR recordings, the cycle length was normalised to a heart frequency of 60/min. B) Original recordings of the normalised peak filling rate (nPFR) during diastole for two individual patients (no SDB in blue; SDB in red) at baseline (solid lines) and follow-up (dotted lines). Grey arrows indicate how the peak filling rate and the time to peak filling rate were measured. The peak filling rate was normalised to the left ventricular end-diastolic volume. C) Comparison between the nPFR of patients with and without SDB from baseline and the three-month follow-up. * shows the p-value for the difference between the change in the nPFR within the three-month follow-up period in each group normalised to baseline value (ANCOVA). CMR – cardiac magnetic resonance imaging; LV – left ventricular; nPFR – normalised peak filling rate; SDB – sleep-disordered breathing. (For interpretation of the references to color/colour in this figure legend, the reader is referred to the Web version of this article.)

group with SDB (6 ± 3 vs. 31 ± 13; $p < 0.001$; Table 1). Of patients with SDB, 62% had predominantly OSA and 38% had CSA.

At the time of AMI (baseline), LV ejection fraction and LV end diastolic volume were similar in both groups. The infarct size tended to be higher in the SDB group (percentage of total LV mass; 23 ± 12% vs. 17 ± 10%; $p = 0.087$). Patients with SDB showed a significantly higher LV mass (220 ± 50 mg vs. 175 ± 43 mg; $p = 0.003$) and LV mass index (107 ± 23 g/m^2 vs. 89 ± 17 g/m^2; $p = 0.007$) than patients without SDB.

4.2. Parameters of left ventricular diastolic function

The nPFR at baseline did not differ between patients with and without SDB. Within three months after AMI, the decrease in diastolic function was significantly higher in patients with SDB than in patients without SDB (ΔnPFR: −0.83 ± 0.14 vs. 0.03 ± 0.14; $p < 0.001$; ANCOVA, adjusted for baseline nPFR; Fig. 2 B + C).

Accordingly, the time to peak filling rate and the peak filling rate were similar in both groups at baseline (Table 2). The increase in the time to peak filling and the decrease in the peak filling rate were significantly higher in the group with SDB than in the group without SDB (Table 2).

At baseline, NT-pro-BNP levels were numerically much higher in the SDB group, however, without reaching statistical significance [1077 (347; 2413) pg/mL vs 505 (250; 811) pg/mL, $p = 0.072$]. In both groups NT-pro-BNP levels were lower after three months, however, levels were higher in patients with SDB [432 (179; 793) pg/mL vs 122 (85; 259) pg/mL, $p = 0.006$]. This reflects the higher rate of diastolic dysfunction in patients with SDB.

Cardiac function is influenced by medication that is routinely prescribed after MI such as ACE-inhibitors/angiotensin-receptor blockers, beta blockers, aldosterone antagonists or loop diuretics. There was no difference in medication at discharge between patients with or without SDB. After three months, the rate of loop diuretics was higher in patients with SDB (48% vs 10%, $p = 0.015$). In addition to the higher NT-pro-BNP levels, this is another indicator of the clinical relevance of the diastolic dysfunction in patients with SDB.

4.3. Predictors of diastolic function three months after acute myocardial infarction

In univariable regression analysis, the AHI was inversely associated with the nPFR at the three-month follow-up (Fig. 3). In multiple linear regression, the AHI was significantly associated with the nPFR three months after AMI after accounting for body-mass index, age, arterial hypertension, LV mass index and infarct size ($p = 0.011$; Table 3). To evaluate whether this finding was driven by the severity of OSA or CSA, we replaced AHI with both the obstructive AHI and the central AHI in the multiple linear regression model. In contrast to the central AHI, the obstructive AHI was significantly associated with the nPFR three months after AMI. Of the established risk factors for diastolic dysfunction, history of hypertension was significantly associated with the nPFR.

In this multiple regression model, neither minimal oxygen saturation, mean oxygen saturation nor total sleep time with oxygen saturation of <90% were significantly associated with the nPFR at follow-up.

5. Discussion

This study provides the following novel findings: (1) within three months after acute MI, diastolic function was significantly lower in patients with SDB compared with patients without SDB, and (2) in contrast to the severity of CSA, the severity of OSA was associated with diastolic dysfunction three months after AMI, after accounting for established risk factors such as body-mass index, arterial hypertension and age.

Table 1
Baseline characteristics of patients with acute myocardial infarction compared to patients with and without SDB.

	No SDB (n = 20)	SDB (n = 21)	p-value
Age, years	54 ± 11	56 ± 10	0.608
Body mass index, kg/m^2	26.7 ± 2.5	29.3 ± 3.4	**0.008**
Men, n (%)	16 (80)	19 (90)	0.343
Heart rate, 1/min.	73 ± 17	76 ± 17	0.583
Systolic blood pressure, mmHg	129 ± 24	131 ± 17	0.726
Diastolic blood pressure, mmHg	79 ± 13	77 ± 10	0.526
Hypertension, n (%)	12 (59)	11 (59)	0.516
Current smoker, n (%)	18 (90)	15 (68)	0.069
Diabetes mellitus, n (%)	3 (15)	5 (23)	0.524
Hypercholesterolemia, n (%)	7 (35)	9 (41)	0.694
Symptom-to-balloon time, Min.	215 (330)	254 (853)	0.917
Non-ST-elevation, n (%)	3 (15)	4 (19)	0.731
Non-LAD-infarction, n (%)	8 (40)	8 (38)	0.901
TIMI-3 flow post PCI, n (%)	20 (100)	20 (95)	0.323
Thrombus aspiration, n (%)	8 (40)	11 (52)	0.427
Glycoprotein IIb/IIIa inhibitor, n (%)	14 (70)	14 (67)	0.819
CK at admission, U/L	242 (442)	487 (710)	0.162
LV EF, %	62 ± 6	60 ± 7	0.391
LV EDV, ml	154 ± 37	169 ± 45	0.239
LV ESV, ml	86 ± 4	83 ± 4	0.100
LV mass, g	175 ± 43	220 ± 50	**0.003**
LV mass index, mg/m^2	89 ± 17	107 ± 23	**0.007**
Infarct size, % LV mass	17 ± 10	23 ± 12	0.087
Time to peak filling rate, ms	109 ± 50	97 ± 29	0.348
Peak filling rate, mL/s	441 ± 114	499 ± 169	0.202
Normalised peak filling rate, EDV/s	2.90 ± 0.54	3.03 ± 1.21	0.655
Apnoea-hypopnea index,/h	6 ± 3	31 ± 13	**0.001**
Mean oxygen saturation, %	93 ± 2	93 ± 2	0.543
Minimum oxygen saturation, %	86 ± 4	83 ± 4	0.100
Medication at discharge			
Loop diuretics, n (%)	6 (30%)	9 (43%)	0.520
ACE inhibitors/ARB, n (%)	17 (85%)	20 (95%)	0.343
Beta blockers, n (%)	17 (85%)	19 (91%)	0.663
Aldosterone antagonist, n (%)	8 (40%)	11 (52%)	0.536
Medication at follow-up			
Loop diuretics, n (%)	2 (10%)	10 (48%)	**0.015**
ACE inhibitors/ARB, n (%)	13 (65%)	18 (86%)	0.159
Beta blockers, n (%)	14 (70%)	16 (76%)	0.734
Aldosterone antagonist, n (%)	4 (20%)	7 (33%)	0.484

Data are n (%), or mean ± standard deviation. p-values were calculated with the Student's t-test and the Fischer's exact test. Bold values mean statistical significance. ACE – angiotensin-converting enzyme; ARB – angiotensin receptor blocker; CK – creatinine kinase; EDV – end-diastolic volume; EF – ejection fraction; ESV – end-systolic volume; LAD – left anterior descending coronary artery, PCI – percutaneous coronary intervention; TIMI – Thrombolysis in Myocardial Infarction.

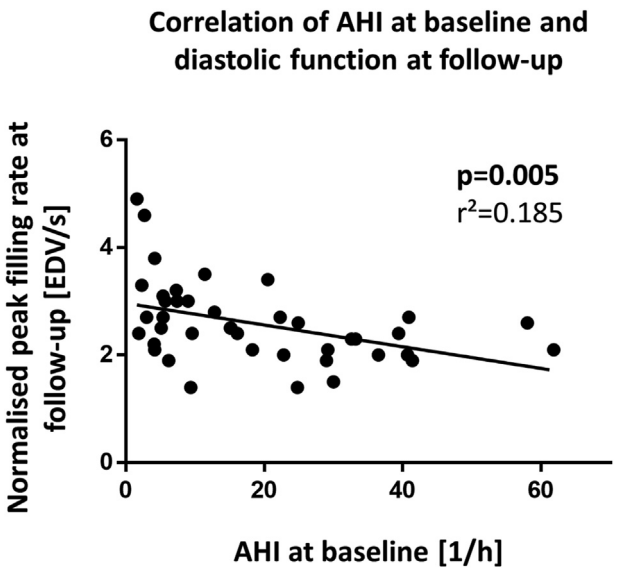

Fig. 3. Correlation between the apnoea-hypopnea index (AHI) and diastolic function at follow-up. The AHI at baseline correlated significantly with the normalised peak filling rate at follow-up. AHI – apnoea-hypopnoea index.

5.1. SDB and diastolic function after acute myocardial infarction

Several studies have suggested a high prevalence of approximately 70% for LV diastolic dysfunction in patients with AMI and preserved ejection fraction [2,23]. Although MI per se has been suggested to play an important role in the development of diastolic dysfunction [2], this development may be aggravated by other cardiovascular comorbidities or risk factors such as advanced age, hypertension, diabetes and obesity.

Our data suggest that SDB contributes to impaired diastolic function three months after AMI. The clinical relevance of this is reflected by higher levels of NT-pro-BNP and higher rates of loop diuretic intake in patients with SDB three months after MI. However, patients with SDB often have coexisting disorders, which may add to the development of diastolic dysfunction, for instance advanced age, obesity and hypertension. Multiple linear regression analyses have shown that, at the time of MI, a higher body-mass index correlates with better diastolic function. On the other hand, obesity is generally accepted to be a risk factor for diastolic dysfunction [24]. Therefore, the correlation observed

Table 2
Changes in diastolic filling parameters.

	No SDB (n = 20)	SDB (n = 21)	p-value
Time to peak filling rate (ms)			
Baseline, mean ± SD	109 ± 50	97 ± 29	0.369^T
Three months, mean ± SD	110 ± 35	129 ± 27	0.094^T
p-value (baseline vs three months)	0.881^T	**0.001^T**	
Adjusted difference between baseline and follow-up, mean ± SD	+7 ± 7	+27 ± 7	**0.044^A**
Peak filling rate (mL)			
Baseline, mean ± SD	440 ± 114	499 ± 169	0.173^T
Three months, mean ± SD	456 ± 160	394 ± 88	0.166^T
p-value (baseline vs. three months)	0.592^T	**0.006^T**	
Adjusted difference between baseline and follow-up, mean ± SD	+3 ± 26	−88 ± 26	**0.027^A**
NT-pro-BNP (pg/mL)			
Baseline, median (IQR)	505 (250; 811)	1077 (347; 2413)	0.072^U
Three months, median (IQR)	122 (85; 259)	432 (179; 793)	**0.006^U**
p-value (baseline vs three months)	**<0.001^W**	**0.001^W**	
Adjusted difference between baseline and follow-up, mean ± SD	603 ± 134	591 ± 130	0.951^A

SDB – sleep-disordered breathing. p-values were calculated with the two-sided Student's t-test (T), Mann-Whitney-U-Test (U), Wilcoxon-Test (W) or ANCOVA (A; adjusted for baseline values). P-values <0.05 are shown in bold.

Table 3
Multiple linear regression analysis of predictors of diastolic function.

Model		Normalised peak filling rate at follow-up	
		B (95% CI)	p
Model 1 with AHI	AHI (1/h)	−0.020 (−0.035 to −0.005)	**0.011**
	BMI (kg/m^2)	−0.013 (−0.086 to 0.059)	0.709
	Age (years)	0.005 (−0.016 to 0.027)	0.605
	Arterial hypertension	0.593 (0.0156 to 1.031)	**0.009**
	LV mass index (g/m^2)	0.002 (−0.011 to 0.015)	0.748
	Infarct size (%)	−1.904 (−3.929 to 0.121)	0.065
	Model summary	R^2 (adj.) = 0.299; **p = 0.006**	
Model 2 with oAHI	Obstructive AHI (1/h)	−0.036 (−0.063 to −0.009)	**0.011**
	BMI (kg/m^2)	−0.004 (−0.079 to 0.070)	0.907
	Age (years)	0.003 (−0.018 to 0.024)	0.779
	Arterial hypertension	0.666 (0.221 to 1.111)	**0.005**
	LV mass index (g/m^2)	−0.001 (−0.014 to 0.012)	0.856
	Infarct size (%)	−1.369 (−3.445 to 0.708)	0.189
	Model summary	R^2 (adj.) = 0.300; **p = 0.006**	
Model 3 with cAHI	Central AHI (1/h)	−0.013 (−035 to 0.008)	0.216
	BMI (kg/m^2)	−0.036 (−0.114 to 0.042)	0.358
	Age (years)	0.004 (−0.019 to 0.028)	0.701
	Arterial hypertension	0.535 (0.051−1.019)	**0.031**
	LV mass index (g/m^2)	0.000 (−0.015 to 0.015)	0.981
	Infarct size (%)	−2.189 (−4.437 to 0.060)	0.056
	Model summary	R^2 (adj.) = 0.179; p = 0.051	

AHI − apnoea-hypopnea-index; B − regression coefficient; cAHI − central AHI; CI − confidence interval; oAHI − obstructive AHI.

in our study is likely to be coincidental. Accordingly, our cohort showed no association between body-mass index and diastolic function at follow-up.

Interestingly, in contrast to the severity of CSA, the severity of SDB and OSA were associated with impaired diastolic function at follow-up. This finding suggests that SDB and specifically OSA have detrimental effects on diastolic function and that this effect may be easier to detect in the vulnerable period early after acute myocardial damage compared to chronic heart disease. MI and SDB share many similarities in pathogenesis, and SDB may therefore reinforce the ischemic damage of MI and prevent healing and positive remodelling. These pathomechanisms include intermittent hypoxia, stimulation of the sympathetic nervous system, elevated blood pressure and activation of pro-inflammatory and pro-fibrotic pathways [12]. Increased negative thoracic pressure swings, which—together with increased arterial blood pressure—contribute to increased ventricular transmural pressure and thus afterload, are unique for OSA. The association of obstructive but not central respiratory events and arterial hypertension with impaired diastolic function suggests that haemodynamic stress is a key mechanism for the development of diastolic dysfunction early after MI. Our group has previously demonstrated that concomitant SDB in patients with MI reduces the salvage index, which leads to a larger myocardial scar area, and that especially OSA impairs remodelling after MI [6,8]. Our current analysis strengthens these observations by showing that SDB also impairs diastolic function, which is a known and important risk factor for reduced systolic function and cardiovascular events after MI [1,2].

5.2. Strengths and limitations

The current study has strengths and limitations that warrant mention. Although assessment of diastolic function with CMR has significant advantages such as allowing reproducible imaging irrespective of body habitus [15,22], temporal resolution is significantly slower than that of echocardiographic Doppler techniques, which would allow further characterisation of diastolic function. The size of the study sample was not large. In our study, we did not examine cardiovascular outcome, but it should be noted that no patient was lost to follow-up due to death or urgent hospitalisation.

6. Summary and conclusion

Our analysis shows that patients with SDB had a greater reduction in diastolic function early after MI compared to patients without SDB. This seems to have clinical relevance as NT-pro-BNP levels and the rate of loop diuretics were higher in patients with SDB three months after MI. In contrast to the severity of CSA, the severity of OSA was an independent predictor of diastolic dysfunction. Our data indicate that OSA contributes to the development of diastolic dysfunction early after MI. Whether the development of diastolic dysfunction can be alleviated by treating SDB merits investigation in randomised controlled trials such as TEAM-ASV I (NCT02093377) [25].

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Data availability statement

The data underlying this article will be shared on reasonable request to the corresponding author.

Authors' contributions

Stefan Buchner and *Michael Arzt* were responsible for the conception, hypotheses delineation and design of the study, acquisition of funding, data acquisition, the analysis and interpretation of such information, writing the article and its revision prior to submission.

Michael Wester was responsible for the analysis and interpretation of the data, writing the article and for its revision prior to submission.

Sarah Hobelsberger, Andrea Hetzenecker, *Kurt Debl* and *Okka W Hamer* were involved in the acquisition of the data, the analysis and interpretation of such information and the critical revision of the article prior to submission.

Lars Maier was involved in the interpretation of the data and the critical revision of the article prior to submission.

Florian Zeman performed the statistical analyses.

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Conflict of interest

M.A. received lecture and consulting fees from ResMed, Philips Respironics, Boehringer-Ingelheim, NRI, Novartis, JAZZ pharmaceuticals, Bayer, Inspire and Bresotec and grant support from ResMed Foundation, ResMed, Philips Respironics and the Else-Kroehner Fresenius Foundation (2018_A159) outside the submitted work.

Stefan Buchner, Michael Wester, Christoph Fisser, Sarah Hobelsberger, Kurt Debl, Okka W Hamer and Florian Zeman have no conflicts of interest to disclose.

The ICMJE Uniform Disclosure Form for Potential Conflicts of Interest associated with this article can be viewed by clicking on the following link: https://doi.org/10.1016/j.sleep.2022.03.028.

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