Effect of early adaptive servoventilation on cardiac repolarisation in patients with myocardial infarction and sleep disordered breathing: an ancillary analysis of the randomised controlled trial TEAM-ASV I

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In patients with acute myocardial infarction and sleep disordered breathing, treatment with adaptive servoventilation may improve cardiac repolarisation after 4 weeks, eventually preventing ventricular arrhythmias in the early phase after infarction https://bit.ly/3RdKqkt

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Abstract

Background In patients with acute myocardial infarction, altered cardiac repolarisation has been linked to ventricular fibrillation and major adverse cardiac events. Treatment of sleep disordered breathing early after acute myocardial infarction may normalise the ventricular ectopic substrate. Therefore, the aim of this ancillary analysis was to assess the effect of adaptive servoventilation (ASV) on cardiac repolarisation.

Methods This analysis included 42 participants from the prospective multicentre randomised trial TEAM-ASV I. 22 were randomised to early treatment with ASV in addition to standard care, and 20 to standard care only. Cardiac repolarisation was assessed by means of T-peak-to-end- ($TpTe_c$) and QT_c- intervals and TpTe/QT-ratios derived from 12-lead ECG before and 4 and 12 weeks after percutaneous coronary intervention.

Results The population was predominantly male (79%), obese (mean±SD body mass index 31±6 $kg \cdot m^{-2}$), had severe sleep disordered breathing with an apnoea–hypopnoea index of 34±18 $events \cdot h^{-1}$ and ASV was initiated within 2.6±1 days. After 4 weeks, QT_c significantly decreased in the ASV group compared to the control group (−5.8% *versus* −1.2%, p=0.041). A similar trend was observed with respect to $TpTe_c$ (−18.0% *versus* −6.8%, p=0.119) and TpTe/QT (−17.6% *versus* −6.6%, p=0.252). After 12 weeks, the decrease in repolarisation times was similar between the groups. (QT_c −7.6% *versus* −4.5%, p=0.400; $TpTe_c$ −19.3% *versus* −18.8%, p=0.400; TpTe/QT −9.7 *versus* −13.0, p=0.652).

Conclusion In patients with first acute myocardial infarction and sleep disordered breathing, early treatment with ASV reduced cardiac repolarisation after 4 weeks, but not after 12 weeks. Thus, ASV may contribute to preventing ventricular arrhythmias in the vulnerable early phase after acute myocardial infarction.

Introduction

Malignant ventricular arrhythmias may be predicted by means of cardiac repolarisation parameters in the ECG, such as extended QT-intervals and extended intervals from the peak of the T-wave to the end of the T-wave (TpTe) [1–3]. Prolonged cardiac repolarisation has been associated with sudden cardiac death in different populations [4–6]. Previous studies reported that in patients with ST-segment elevation myocardial infarction (STEMI), cardiac repolarisation was significantly linked to ventricular fibrillation and major adverse cardiac events [2, 5].

Sleep disordered breathing (SDB) is characterised by apnoeas and hypopnoeas resulting in intrathoracic pressure swings, recurrent arousals and intermittent hypoxia [7]. Consequently, cardiac afterload, oxidative stress and sympathetic nervous system activity may be increased, leading to structural and electrical remodelling of the heart. Therefore, SDB can destabilise ventricular repolarisation and finally can cause cardiac arrhythmias [8–11]. The two main types of SDB are obstructive sleep apnoea (OSA) and central sleep apnoea (CSA) [7, 12]. OSA is caused by an obstruction of the upper airway; the inspiratory efforts against the closed pharynx result in negative intrathoracic pressure [8]. In contrast, CSA is caused by a disorder of respiratory regulation and typically shows a characteristic periodic breathing pattern with a waxing and waning tidal volume (Cheyne–Stokes respiration) [8, 13].

SDB is an independent risk factor for prolonged cardiac repolarisation in STEMI patients [14]. In addition, a prospective randomised study in OSA patients without acute myocardial infarction (AMI) revealed that withdrawal of continuous positive airway pressure (CPAP) for 2 weeks resulted in a prolongation of the QT_c, $TpTe_c$ and TpTe/QT ratio compared with continued CPAP therapy [15].

Adaptive servoventilation (ASV) may effectively treat OSA and CSA [16]. However, the effect of ASV treatment on cardiac repolarisation as a surrogate parameter for malignant ventricular arrhythmias in patients with SDB early after AMI has not yet been investigated. Therefore, we tested the hypothesis that ASV treatment leads to a reduction in cardiac repolarisation parameters, namely, QT_c, $TpTe_c$ and TpTe/QT-ratio.

Methods

Study design

Details of the study design have been published previously [17]. Briefly, the analysed patient population is a subset of the multicentre, randomised, parallel-group, open-label trial with blinded assessment of outcomes, TEAM-ASV I (ClinicalTrials.gov identifier NCT02093377), which was carried out between 2014 and 2021 to compare treatment with percutaneous coronary intervention (PCI), optimal medical therapy and ASV (ASV group) with PCI and optimal medical therapy only (control group) in patients with AMI and SDB [18]. This ancillary analysis included only patients enrolled at the University Medical Center Regensburg (Regensburg, Germany). The trial was approved by the local institutional ethics committee (Ethikkommission der Universität Regensburg, approval number 11-101-0229) and was conducted in accordance with the Declaration of Helsinki. Before undergoing the study examinations, all patients provided written informed consent [14, 17].

Patients

The full details of the study inclusion/exclusion criteria have been described previously [17]. This analysis included 42 participants from the TEAM ASV I trial (aged 18–80 years) with first AMI and successful PCI who were treated within 24 h after symptom onset at the University Medical Center Regensburg. These patients with previously undiagnosed SDB were subsequently assigned to either the group with ASV therapy (optimal drug therapy and ASV therapy) or the group without ASV therapy (only optimal drug therapy) [17]. Patients with previous AMI, previous myocardial revascularisation (PCI or surgical), or an indication for surgical revascularisation were excluded. Other exclusion criteria were cardiogenic shock, implanted cardiac assist devices, other contraindications to cardiac magnetic resonance imaging and pre-existing treated SDB [14]. After a stop of the trial 2015, in light of the findings of the SERVE-HF trial [19], recruitment was continued in January 2016 with the additional exclusion criterion of left ventricular ejection fraction (LVEF) $\leqslant$45% with predominant CSA [18]. In addition to the exclusion criteria for the main study, for this analysis, ECGs with insufficient quality at two different time points that did not allow analysis of repolarisation were excluded.

ECG measurements

In eligible patients, a routine 12-lead ECG was recorded at baseline (before PCI), after 4 weeks and after 12 weeks [17]. Cardiac repolarisation times were measured manually using the DatInf Measure 3.0 analysis software (DatInf, Tübingen, Germany). The preferred ECG lead for this purpose was V5, followed by V4, V6, II, I and III [20]. This selection was based on experiences in previous studies that faced difficulties in measuring V1 and V2 because of the augmented quantity of T-wave morphology and the possible consequences of shorter or longer TpTe times [14]. In addition, only non-infarct-related leads, in which the maximum ST-segment elevation in the pre-PCI ECG was <0.055 mV, were measured [20]. QT-, TpTe- and RR-intervals were measured according to the current literature. The QT-interval was measured from the beginning of the QRS complex to the end of the T-wave and TpTe was the time from the peak of the T-wave until the end of the T-wave. The end of the T-wave was defined as the point at which the tangent of the maximal downwards slope of the T-wave crossed the isoelectric line [15]. RR-intervals were defined

as the distance between two R-spikes. By measuring three consecutive heartbeats within one lead, the respective mean value was calculated [14]. Both QT and TpTe, were corrected for heart rate using Bazett's formula (QT_c=QT/$\sqrt{}$(RR); $TpTe_c$=TpTe/$\sqrt{}$(RR)) [21]. From the parameters TpTe and QT, the TpTe/QT-ratio was calculated [14].

Polygraphy

All patients underwent polygraphy (SOMNOscreen plus RC; SOMNOmedics, Randersacker, Germany). Polygraphy was performed within 3 days after PCI and was repeated 12 weeks after PCI [17]. The sleep laboratory is located in the cardiology ward of the University Medical Center Regensburg, to which the patients were admitted. Polygraphy evaluation includes breathing parameters (such as pressure cannula, snoring and thoracic and abdominal respiratory effort measures), oxygen saturation, ECG (including estimates of systolic and diastolic blood pressure on the basis of the pulse-transit time method using validation references), recording of body position and light detection [17]. The polygraphs were analysed by a central scoring lab according to the criteria of the American Academy of Sleep Medicine. The experienced sleep technician on duty was blinded to the clinical data [14]. Apnoea was defined as a reduction in inspiratory airflow of ⩾90% for ⩾10 s, hypopnoeas were defined as a repetitive reduction in respiratory flow of >30% and an associated decrease in oxygen saturation of 4% [13]. The apnoea–hypopnoea index (AHI) was used to classify the severity of SDB; it indicates the number of apnoeas and hypopnoeas per hour [22]. An AHI of <5 events·h^{-1} indicates no SDB; values of 5–<15 events·h^{-1} indicate mild SDB; values of 15–<30 events·h^{-1} indicate moderate SDB; and an AHI of ⩾30 events·h^{-1} indicates severe SDB [7]. The type of SDB is categorised as predominantly CSA (<50% of apnoeas are obstructive) or predominantly OSA (⩾50% of apnoeas are obstructive) [17].

Statistical analysis

Normally distributed quantitative data are expressed as mean±SD and non-normally distributed data are expressed as median and interquartile range unless otherwise indicated. The t-test was used for continuous and normally distributed variables with equal variances, categorially distributed variables were compared using the Chi-squared test. For non-normally distributed variables, the Wilcoxon–Mann–Whitney U-test was used for independent samples, and the Wilcoxon signed-rank test was used for related samples. Simple linear regression models were conducted to assess the association between the change in hypoxia and the change in QT_c. For statistical analysis, the IBM SPSS Statistics 29.0 software package (IBM, Armonk, NY, USA) was used. All reported p-values were two-sided and a p-value <0.05 was considered statistically significant. Due to the exploratory nature of the analyses, no adjustments for multiple testing were made.

Results

Patient characteristics

42 patients were included in this ancillary analysis and randomised to the ASV (n=22) or control (n=20) group (figure 1). In patients randomised to the ASV group, treatment was initiated within 2.6±1 days after AMI. Baseline characteristics such as age, body weight, heart rate and sex were similarly distributed between the ASV group and the control group (table 1). The study population was predominantly male, obese, and the majority of the patients had STEMI (table 1).

Nocturnal respiratory characteristics

The nocturnal respiratory characteristics at baseline were similar in both groups (table 2). The study population had severe SDB. After 12 weeks, the reduction in the AHI was significantly greater in the ASV group than in the control group (−34±22 events·h^{-1} *versus* −6±14 events·h^{-1}, p<0.001). A similar result was shown for the oxygen desaturation index (−30±23 events·h^{-1} *versus* 0±12 events·h^{-1}, p<0.001) (table 2).

ECG parameters

Baseline QT_c-, $TpTe_c$-interval and TpTe/QT-ratio were comparable between the ASV and the control group (table 3). The decrease in the QT_c-interval within the first 4 weeks after PCI was significantly greater in patients who received ASV therapy than in those in the control group (−5.8% *versus* −1.2%, p=0.041). The $TpTe_c$-interval (−18.0% *versus* −6.8%, p=0.119) and the TpTe/QT-ratio (−17.6% *versus* −6.6%, p=0.252) decreased by trend, more in the ASV group than in the control group (figure 2; table 4). In addition, a subanalysis suggests that in the ASV group, the contribution of the alleviation of obstructive apnoeas to the shortening of the QT_c interval after 4 weeks was greater than the contribution of the alleviation of central apnoeas (supplementary figure S1). After 12 weeks, the decrease in the repolarisation parameters was comparable between the two groups (figure 2; table 4). Within the ASV group, all repolarisation parameters decreased after 12 weeks, whereas in the control group, only $TpTe_c$ was significantly reduced (supplementary table S1). After 4 and 12 weeks, reduction in resting heart rate during

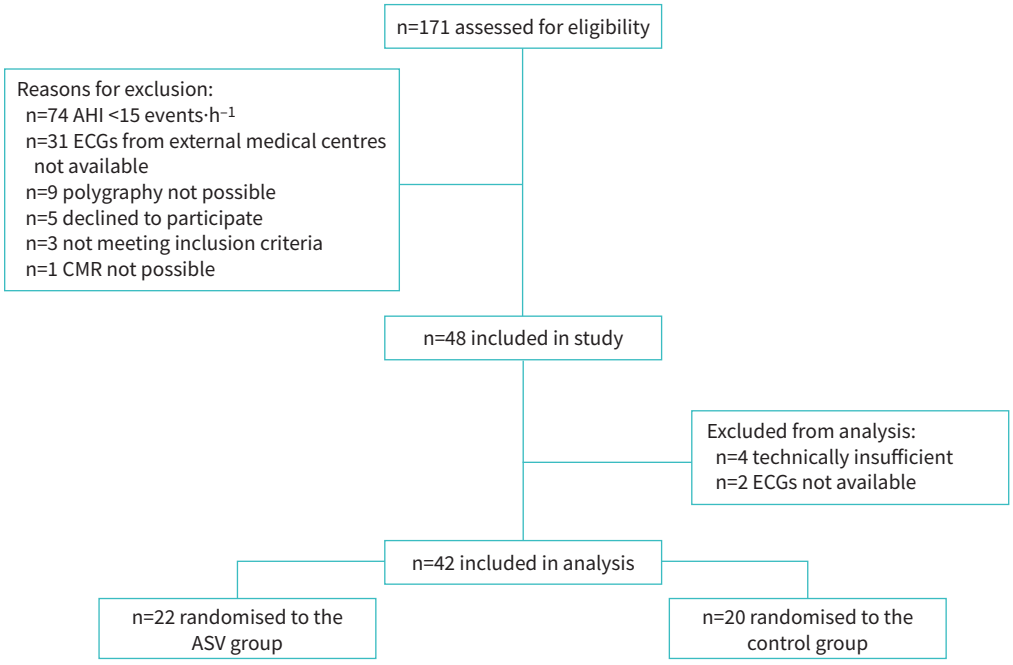

FIGURE 1 Flow of patients throughout the study. AHI: apnoea–hypopnoea index; CMR: cardiac magnetic resonance; ASV: adaptive servoventilation.

the day was numerically greater in the ASV compared to the control group (−9 *versus* −2 beats·min^{-1}, p=0.106; −17 *versus* −10 beats·min^{-1}, p=0.354, respectively; figure 3; table 3). In simple linear regression analyses, no association between time with oxygen saturation <90% (hypoxic load) and the change in QT_c intervals, could be demonstrated (4 weeks after PCI: B-coefficient −0.212, 95% CI −1.52–1.09, p=0.727; 12 weeks after PCI: B-coefficient −0.025, 95% CI −0.88–0.83, p=0.950).

Discussion

This study provides the following insights about the effect of ASV treatment on cardiac repolarisation in patients with AMI and SDB. First, the reduction in the QT_c within 4 weeks differed significantly between the ASV group and the control group. Similar results were yielded for $TpTe_c$, but not for the TpTe/QT ratio. Second, after 12 weeks, no difference was observed between the groups. Third, after 4 and 12 weeks, the deceleration in heart rate was greater by trend in the ASV group compared with the control group.

The QT-interval represents ventricular depolarisation and repolarisation and includes the vulnerable period for re-entry tachycardias. For that reason, many studies have identified a prolongation of the QT-interval as a risk factor for malignant cardiac arrhythmias, which may lead to sudden cardiac death [1, 6]. Similarly, a prolonged TpTe-interval and TpTe/QT ratio have been shown to be associated with ventricular arrhythmias and sudden cardiac death [5, 23, 24]. However, in contrast to QT, TpTe reflects the transmural dispersion of repolarisation, leading to increased vulnerability for the appearance of early afterdepolarisations and TpTe/QT represents the disproportional prolongation of global dispersion [23, 24]. In addition, other studies have shown that patients on β-blockers have a shorter QT_c-interval, particularly at high heart rates, in contrast to those not using them. This antiarrhythmic mechanism could explain the preventive benefit of β-blocker therapy, which also significantly reduces the risk of sudden cardiac death [25–27].

Both SDB and myocardial infarction may lead to prolonged repolarisation [14]. The results of the present study revealed that ASV treatment may reduce QT_c, $TpTe_c$ and TpTe/QT ratio within 12 weeks, but QT_c and the TpTe/QT ratio did not change significantly in the control group. These findings are in accordance with those of Rossi *et al.* [15], who assessed the effect of withdrawal of CPAP therapy on cardiac repolarisation in 41 patients with OSA treated with CPAP for ⩾12 months before the start of the study. The withdrawal for 2 weeks resulted in significantly prolonged QT_c, $TpTe_c$ and TpTe/QT ratios [15]. In the current study, ASV was used instead of CPAP treatment because ASV therapy has an effect, not only on OSA, but also on CSA [16, 28]. In contrast to Rossi *et al.* [15], we assessed patients with myocardial

TABLE 1 Baseline demographics and clinical characteristics (eligible for follow-up)

	Total	ASV	No ASV	p-value
Patients	42	22	20	
Age years	60±10	61±11	59±8	0.528
Body mass index kg·m^{-2}	31.1±6.3	30.2±4.5	32.0±7.8	0.379
Male	33 (79)	17 (77)	16 (80)	0.830
Heart rate beats·min^{-1}	74±15	76±18	71±11	0.406
Systolic blood pressure mmHg	119±16	116±17	122±14	0.250
Diastolic blood pressure mmHg	73±10	71±9	76±10	0.094
Hypertension	25 (60)	12 (55)	13 (65)	0.491
Atrial fibrillation	2 (5)	1 (5)	1 (5)	0.945
Current smoker	23 (55)	12 (55)	11 (55)	0.976
Diabetes mellitus	7 (17)	4 (18)	3 (15)	0.792
Hypercholesterolaemia	21 (50)	12 (55)	9 (45)	0.537
Low-density lipoprotein[#] mg·dL^{-1}	124±30	127±31	121±28	0.570
NT-proBNP[#] pg·mL^{-1}	1113 (479–1905)	1248 (717–1905)	908 (315–2037)	0.435
Left ventricular ejection fraction %	52±10	51±9	53±11	0.445
ST-segment elevation myocardial infarction	34 (81)	17 (77)	17 (85)	0.524
Pain-to-balloon time h	3.7 (2.2–8.2)	3.7 (2.4–9.7)	3.9 (2.2–8.2)	0.935
Culprit lesion				
Left main artery	0 (0)	0 (0)	0 (0)	
LAD artery	30 (71)	15 (68)	15 (75)	0.625
Circumflex artery	15 (36)	8 (36)	7 (35)	0.927
Right coronary artery	26 (62)	12 (55)	14 (70)	0.303
Thrombus aspiration	11 (26)	6 (27)	5 (25)	0.867
Glycoprotein IIb/IIIa inhibitor	8 (19)	4 (18)	4 (20)	0.881
Medication at discharge				
Aspirin	42 (100)	22 (100)	20 (100)	1.000
ADP receptor inhibitor	42 (100)	22 (100)	20 (100)	1.000
β-blocker	37 (88)	19 (86)	18 (90)	0.716
ACE inhibitor	37 (88)	21 (96)	16 (80)	0.122
Statin	42 (100)	22 (100)	20 (100)	1.000

Data are expressed as n, mean±SD, n (%) or median (interquartile range), unless otherwise stated. ASV: adaptive servoventilation; NT-proBNP: N-terminal pro-B-type natriuretic peptide; LAD: left anterior descending; ACE: angiotensin-converting enzyme. [#]: laboratory parameters were measured within the first 24 h after acute myocardial infarction.

TABLE 2 Sleep characteristics

	Total	ASV	No ASV	p-value
Patients	42	22	20	
Baseline				
AHI events·h^{-1}	34±18	34±17	35±18	0.848
Oxygen desaturation index events·h^{-1}	26±19	28±20	24±19	0.493
Minimum oxygen saturation %	81±7	82±4	80±9	0.251
Mean oxygen saturation %	92±2	92±2	92±2	0.796
Time with oxygen saturation <90% %TRT	13±17	14±18	12±17	0.741
Central apnoea index/apnoea index %	47±33	46±34	49±31	0.801
12 weeks after PCI				
ΔAHI[#] events·h^{-1}	−19±23	−34±22	−6±14	**<0.001**
ΔOxygen desaturation index[#] events·h^{-1}	−13±23	−30±23	0±12	**<0.001**
ΔMinimum oxygen saturation[#] %	3±5	4±5	3±6	0.604
ΔMean oxygen saturation[#] %	1±2	1±2	1±2	0.528
ΔTime with oxygen saturation <90% %TRT	−7±14	−7±17	−7±11	0.946

Data are expressed as n or mean±SD, unless otherwise stated. Bold type represents statistical significance. ASV: adaptive servoventilation; AHI: apnoea–hypopnoea index; TRT: total recording time; PCI: percutaneous coronary intervention. [#]: n=9 missing data.

TABLE 3 Absolute changes in cardiac repolarisation parameters and heart rate

	ASV	No ASV	p-value
Patients	22	20	
Pre-PCI[#]			
QT_c ms	412 (390–431)	400 (380–419)	
$TpTe_c$ ms	86 (73–98)	81 (65–95)	
TpTe/QT-ratio	0.21 (0.18–0.25)	0.20 (0.17–0.23)	
Heart rate beats·min^{-1}	74 (63–85)	70 (62–79)	
4 weeks after PCI[¶]			
ΔQT_c ms	−24.1 (−52.1–−9.2)	−4.8 (−18.3–24.0)	**0.041**
Δ$TpTe_c$ ms	−17.6 (−29.5–−1.8)	−4.6 (−14.6–5.1)	0.082
ΔTpTe/QT-ratio	−0.04 (−0.07–0.01)	−0.01 (−0.03–0.01)	0.252
ΔHeart rate beats·min^{-1}	−9 (−39–−1)	−2 (−14–6)	0.106
12 weeks after PCI[+]			
ΔQT_c ms	−29.1 (−41.1–−15.1)	−17.4 (−38.4–9.5)	0.425
Δ$TpTe_c$ ms	−15.7 (−35.4–−4.9)	−16.8 (−30.5–3.5)	0.505
ΔTpTe/QT-ratio	−0.02 (−0.08–0.00)	−0.03 (−0.06–0.02)	0.591
ΔHeart rate beats·min^{-1}	−17 (−41–−1)	−10 (−20–−1)	0.354

Data are expressed as n or median (interquartile range), unless otherwise stated. Bold type represents statistical significance. ASV: adaptive servoventilation; PCI: percutaneous coronary intervention; TpTe: T-peak-to-end. [#]: n=5 missing data; [¶]: n=18 missing data; [+]: n=13 missing data.

infarction. Therefore, treatment with ASV seems to be more reasonable because of the higher number of patients with CSA [29]. In addition, the short-term (4 weeks) and long-term (12 weeks) effects on cardiac repolarisation were evaluated.

ASV therapy led to a greater reduction in heart rate in the treatment group after both 4 and 12 weeks. Bauer *et al*. [30] showed that an impaired ability to decelerate heart rate is a strong predictor of mortality after myocardial infarction and more accurate than LVEF. Since SDB with its arousals, intermittent hypoxia and increased sympathetic activity leads to acute surges in heart rate and blood pressure [8], ASV treatment, which helps to decelerate the heart rate, may eventually contribute to a reduction in mortality.

On the basis of the current results, ASV treatment may have a protective effect on the development of ventricular arrhythmias in the vulnerable early phase after myocardial infarction. This effect may be explained by the fact that patients with SDB already have an increased risk for prolonged repolarisation and the myocardial infarction with its inflammatory processes suddenly adds another risk factor. The

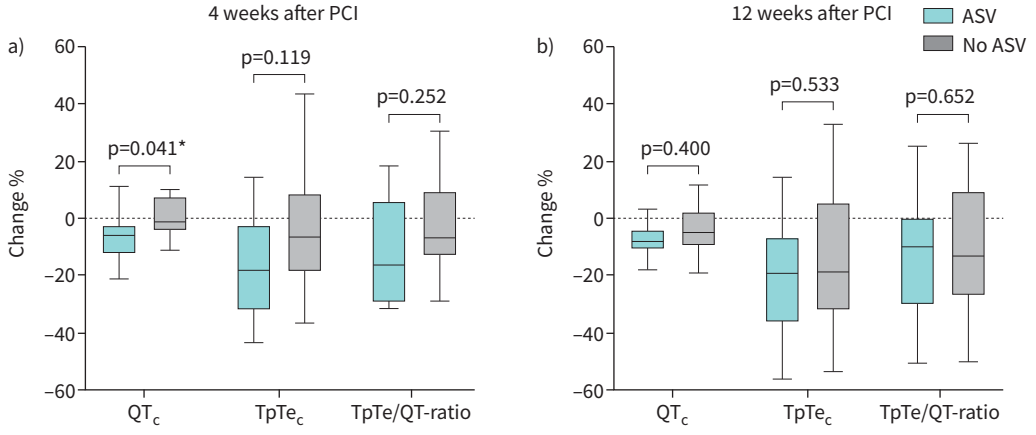

FIGURE 2 a) Changes in cardiac repolarisation parameters 4 weeks after percutaneous coronary intervention (PCI); b) changes in cardiac repolarisation parameters 12 weeks after PCI. Data are presented as median (interquartile range). Percentage values are related to the baseline time. ASV: adaptive servoventilation; TpTe: T-peak-to-end. *: p<0.05.

TABLE 4 Percentage change in cardiac repolarisation parameters

	ASV	No ASV	p-value
Patients	22	20	
4 weeks after PCI[#]			
ΔQT_c %	−5.8 (−12.0–−2.4)	−1.2 (−4.0–6.7)	**0.041**
$\Delta TpTe_c$ %	−18.0 (−32.4–−2.4)	−6.8 (−18.4–8.1)	0.119
ΔTpTe/QT-ratio	−16.7 (−29.8–5.4)	−6.6 (−12.6–9.6)	0.252
12 weeks after PCI[¶]			
ΔQT_c %	−7.6 (−10.5–−3.8)	−4.5 (−9.3–2.5)	0.400
$\Delta TpTe_c$ %	−19.3 (−35.7–−6.8)	−18.8 (−31.7–5.5)	0.533
ΔTpTe/QT-ratio	−9.7 (−30.0–0.5)	−13.0 (−29.7–9.4)	0.652

Data are expressed as n or median (interquartile range), unless otherwise stated. Percentage values are related to the baseline time. Bold type represents statistical significance. ASV: adaptive servoventilation; PCI: percutaneous coronary intervention; TpTe: T-peak-to-end. [#]: n=18 missing data; [¶]: n=13 missing data.

results of the subanalysis, showing a higher impact of obstructive apnoea alleviation on the QT_c time after 4 weeks, are in accordance with the findings of Fisser *et al*. [31], who showed that OSA is associated with spheric cardiac remodelling after AMI, to which the negative intrathoracic pressure swings may contribute. As after 12 weeks, there was no significant difference between the groups, later ASV usage may be less relevant than early use after AMI with respect to cardiac arrhythmias. A reason for this could be that infarct expansion occurs, even after revascularisation of the culprit lesion, early after AMI because of microvascular obstruction and the incomplete cardiac remodelling processes [32, 33]. Furthermore, Buchner *et al*. [12] reported that cardiac remodelling, and consequently improvement in cardiac function after myocardial infarction, is associated with alleviation of sleep apnoea in those with an improved ejection fraction over 12 weeks.

Several mechanisms have been considered to explain the connection between SDB and altered cardiac repolarisation. Repeated inspiratory efforts against collapsed upper airways are associated with large swings in intrathoracic pressure [15]. It is assumed that this pressure, on the one hand, exerts a direct stretching force on the heart and aorta and, on the other hand, leads to changes in the atrial and ventricular volumes [15, 34, 35]. Several studies have shown that this stretch to the cardiac walls and intrathoracic vessels leads to structural and electrical myocardial remodelling [34, 36, 37]. Additionally, intermittent hypoxia is considered a risk factor for cardiac arrhythmia because it increases oxidative stress and sympathetic nervous system activity [22, 38]. In the present study, ASV successfully reduced desaturations, which may have resulted in different short-term effects on cardiac repolarisation. However, an association between hypoxic load and change of QT_c interval was not noted in the ASV group.

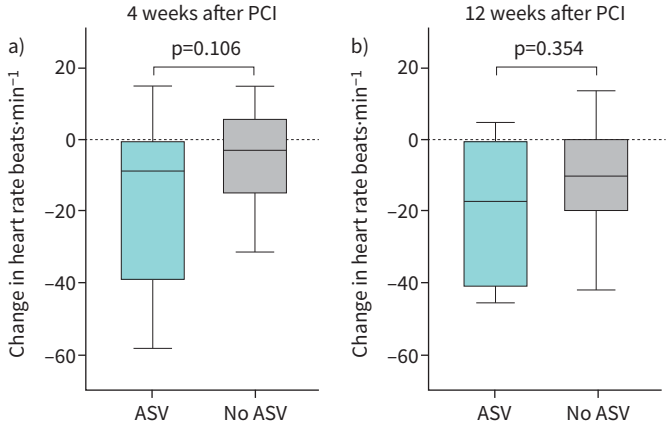

FIGURE 3 a) Changes in heart rate 4 weeks after percutaneous coronary intervention (PCI); b) changes in heart rate 12 weeks after PCI. Data are presented as median (interquartile range).

Strengths and limitations

The strength of this study includes the rapid initiation of ASV treatment within 2.6±1 days after AMI, which may have benefitted the positive short-term effects on cardiac arrhythmias. Furthermore, cardiac repolarisation was assessed after 4 and 12 weeks to represent short- and long-term effects of ASV treatment. Some limitations must be taken into account when interpreting the results. Due to the retrospective design of this ancillary analysis, no causal relationships can be established. A limitation of this study is the small sample size, which limits the statistical power of comparisons between the groups. The number of ECGs per visit varied during the study period. Haemodynamically unstable patients and those with contraindications for cardiac magnetic resonance imaging were excluded and therefore may have resulted in lower repolarisation parameters. As mentioned in the Methods section, the TEAM ASV I trial did not include predominant CSA patients with LVEF ⩽45% after the publication of the SERVE-HF trial; thus, no conclusion on this patient subgroup is possible. Due to the design of the main study, the effect of ASV but not CPAP on cardiac repolarisation was investigated. However, CPAP may have positive effects as well.

Conclusion

In patients with first AMI and SDB, treatment with ASV early after PCI reduced cardiac repolarisation parameters after 4 weeks, but not after 12 weeks, in contrast to patients without ASV. These results suggest that ASV therapy may help to prevent ventricular arrhythmias in the short-term, but not in the long-term period after myocardial infarction. Nevertheless, larger confirmatory randomised trials on early ASV therapy after AMI are needed to gain further insight into the arrhythmia risk of such patients.

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