

# Sleep duration in the first months after ST elevation myocardial infarction: an independent predictor of all-cause mortality

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## Abstract

**Background:** Sleep duration and sleep quality affect patients' general condition and self-reported health status.

**Aim:** The aims of this study were: (1) to describe the clinical characteristic of ST-elevation myocardial infarction (STEMI) patients who sleep too little or too much; and (2) to determine whether sleep duration is independently associated with a higher risk of all-cause mortality.

**Methods:** We enrolled into the study 407 consecutive patients admitted with a diagnosis of STEMI. All patients were asked for sleep duration in the first three months after being discharged from the hospital. According to the sleep duration, we divided patients into three groups: A — the reference category defined as 6–8 sleep hours, B — short sleep with < 6 h, and C — long sleep with > 8 h.

**Results:** The final analysis covered 379 patients (271 males; mean age  $59.4 \pm 10.61$ ). 36 (9.5%) patients slept less than 6 h, 26 (6.9%) slept more than 8 h per night. The all-cause three-year mortality was 1.9% in the reference category, 13.9% in patients who slept less than 6 h, and 30.8% in patients who slept more than 8 h per night ( $p < 0.0001$ ). In the multiple logistic regression analysis, short (OR 10.2, 95% CI 2.1–50,  $p = 0.004$ ) and long sleep duration (OR 33.3, 95% CI 6.8–163.4,  $p < 0.001$ ) were strong and independent predictors of all-cause mortality.

**Conclusions:** Too short and especially too long duration of sleep in the first months after myocardial infarction are strong, independent predictors of all-cause mortality.

**Key words:** sleep duration, myocardial infarction, mortality

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## INTRODUCTION

Sleep duration strongly influences the general condition of patients. A sleep time of between 6 h and 8 h per night may be considered normal and having no negative influence on patient health [1]. On the other hand, too little or too much sleep is associated with adverse health outcomes including hypertension, atrial fibrillation, type 2 diabetes, obesity, dyslipidaemia and/or poor self-rated health [2–7]. All these factors, especially when coexisting, are known to increase the risk of cardiovascular disease (CVD) [7–9]. Thus, as expected,

patients with both sleep excess and deficiency are more likely to suffer from coronary heart disease [10, 11]. Shortened or prolonged sleep length prior to myocardial infarction (MI) is believed to contribute negatively to its occurrence, but whether the time of sleep after the event also affects the general outcome remains unknown. The aim of this study was: (1) to describe the clinical characteristic of ST-elevation myocardial infarction (STEMI) patients who sleep too little (< 6 h) or too much (> 8 h); (2) to determine whether sleep duration is associated with a higher risk of all-cause mortality.

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## METHODS

### Study design, settings and selection of subjects

We enrolled 407 consecutive patients with a confirmed diagnosis of STEMI, admitted to a high-volume, tertiary university hospital with an experienced catheterisation laboratory, who presented to the emergency room within 12 h from the onset of symptoms. Patients' clinical data was collected by interview, physical examination and medical records analysis. The initial screening interview covered cardiovascular risk factors: age, history of hypertension, diabetes mellitus, smoking, dyslipidaemia and having experienced major adverse cardiac events. Hypertension was defined as blood pressure values  $\geq 140/90$  mm Hg or taking antihypertensive drugs. The presence of diabetes was established basing on written diagnosis made previously by a qualified physician. The primary endpoint was all-cause mortality. Survival status was established by telephone contact or analysis of vital status in population registries. Respondent follow-up was carried out as close to three years from the baseline interview. 28 patients, who were lost to follow-up, were not analysed. The study protocol was approved by the Regional Ethics Committee. Informed consent was obtained from every patient before enrolling in the study.

### Assessment of sleep duration

Sleep assessment was conducted using the method described in another prospective cohort study [12]. Prior to hospital discharge, all patients were asked to write down their sleep duration each morning after waking up. All patients were contacted via telephone and asked for mean sleep duration in the first 3 months after discharge from the hospital, based on the written values. Sleep duration was assessed by asking the study participants to give their habitual night sleep time: how many hours do you sleep usually each night? Patient's response: |\_\_|\_\_| hours per night. According to the sleep duration, we divided patients into three groups: group A — the reference category defined as 6–8 sleep hours per night; group B — short sleep, defined as  $< 6$  h per night; and group C — long sleep, defined as  $> 8$  h per night. Groups B and C were both considered as groups with abnormal sleep time.

### Statistical analysis

Continuous data is presented as mean  $\pm$  standard deviation (SD) and was compared using the Mann-Whitney test or Student's t-test. Categorical variables were compared using either the  $\chi^2$  or Fisher exact tests. Multivariable logistic regression analysis including sleep duration, age, sex, history of hypertension, smoking, diabetes mellitus, dyslipidaemia, body mass index (BMI) was performed to identify predictors of all-cause mortality. A p value of less than 0.05 was considered statistically significant, whereas the confidence intervals (CI) were 95%. All analyses were performed using SAS statistical software version 8.02 (SAS Institute, Inc, Cary, NC, USA).

Table 1. Baseline characteristics of the study group (n = 379)

Age [years]	59.4 $\pm$ 10.6
Male sex	271 (71.5%)
BMI [kg/m <sup>2</sup> ]	28.0 $\pm$ 11.2
SBP [mm Hg]	130.6 $\pm$ 25.0
DBP [mm Hg]	78.1 $\pm$ 17.3
HR [bpm]	76.4 $\pm$ 15.5
Diabetes mellitus	48 (12.7%)
Hypertension	195 (51.4%)
Dyslipidaemia	96 (25.3%)
Smoking	191 (50.4%)
Peripheral vascular disease	24 (6.3%)
NYHA class III–IV	9 (2.4%)
COPD/asthma	18 (4.8%)
Prior MI	60 (15.8%)
Prior PTCA	13(3.4%)
Prior CABG	8 (2.1%)
Prior stroke	17 (4.5%)
Sleep duration post discharge [h]	6.5 $\pm$ 1.2

BMI — body mass index; CABG — coronary artery bypass grafting; COPD — chronic obstructive pulmonary disease; DBP — diastolic blood pressure; HR — heart rate; MI — myocardial infarction; NYHA — New York Heart Association; PTCA — percutaneous transluminal coronary angioplasty; SBP — systolic blood pressure

## RESULTS

### Characteristics of study subjects

We included into the final analysis 379 out of 407 patients, in whom we were able to conduct the follow-up. Our population was predominantly male (71.5%; mean age: 59.4  $\pm$  10.6 years). Baseline characteristics are shown in Table 1. The assessment of sleep duration revealed that 36 (9.5%) patients slept for less than 6 h, 26 (6.9%) slept for more than 8 h, and 317 (83.6%) slept between 6 h and 8 h.

Patients with abnormal sleep duration were older (62.7  $\pm$  10.2 vs. 58.7  $\pm$  10.6,  $p = 0.006$ ), and had higher BMI (32.1  $\pm$  25.6 vs. 27.2  $\pm$  4.1,  $p = 0.02$ ) compared to the patients sleeping between 6 h and 8 h. In addition, there was no statistically significant differences in history of diabetes mellitus (21.0% vs. 11.0%,  $p = 0.05$ ), dyslipidaemia (30.7% vs. 24.3%,  $p = 0.37$ ) and hypertension (54.9% vs. 50.8%,  $p = 0.66$ ). Patients with a sleep time of  $< 6$  h and  $> 8$  h combined, had more often experienced MI (25.8% vs. 11.9%,  $p = 0.03$ ), stroke (12.9% vs. 2.8%,  $p = 0.001$ ), and coronary artery bypass grafting (CABG; 6.5% vs. 1.3%,  $p = 0.03$ ) in the past, than the reference group. Other parameters such as the presence of peripheral vascular disease, heart failure (HF) or current smoking did not differ between the groups (Table 2).

After being divided into three groups (A — the reference category defined as sleeping 6–8 h per night, B — short sleep defined as  $< 6$  h per night, and C — long sleep defined

**Table 2.** Comparison between patients sleeping 6 h to 8 h per night and those sleeping less than 6 h and more than 8 h

Characteristics	Sleeping between 6 h and 8 h per night (n = 317)	Sleeping less than 6 h and more than 8 h combined (n = 62)	P
Age [years]	58.7 ± 10.6	62.7 ± 10.2	0.006
Male sex	231 (72.9%)	40 (64.5%)	0.24
Body mass index [kg/m <sup>2</sup> ]	27.2 ± 4.1	32.1 ± 25.6	0.02
Systolic blood pressure [mm Hg]	130.0 ± 25.0	133.9 ± 25.2	0.28
Diastolic blood pressure [mm Hg]	78.0 ± 17.7	78.9 ± 15.3	0.68
Heart rate [bpm]	76.2 ± 15.8	77.0 ± 13.9	0.69
Diabetes mellitus	35 (11.0%)	13 (21.0%)	0.05
Hypertension	161 (50.8%)	34 (54.9%)	0.66
Dyslipidaemia	77 (24.3%)	19 (30.7%)	0.37
Smoking	164 (51.7%)	27 (43.5%)	0.30
Peripheral vascular disease	19 (6.0%)	5 (8.1%)	0.74
Heart failure (III/IV NYHA)	5 (1.6%)	4 (6.5%)	0.06
COPD/asthma	12 (3.8%)	6 (9.7%)	0.09
Prior myocardial infarction	44 (11.9%)	16 (25.8%)	0.03
Prior PTCA	11 (3.5%)	2 (3.2%)	0.78
Prior CABG	4 (1.3%)	4 (6.5%)	0.03
Prior stroke	9 (2.8%)	8 (12.9%)	0.001

CABG — coronary artery bypass grafting; COPD — chronic obstructive pulmonary disease; NYHA — New York Heart Association; PTCA — percutaneous transluminal coronary angioplasty

as > 8 h per night) we could see that patients from the groups sleeping < 6 h were significantly older than those in the reference category ( $65.7 \pm 9.2$  vs.  $58.7 \pm 10.6$ ,  $p = 0.0001$ ) and than those sleeping > 8 h ( $58.3 \pm 10.1$ ,  $p = 0.003$ ), with no difference between groups A and C. A difference in BMI was seen only when we compared the long-sleeping group to the reference category ( $29.6 \pm 4.8$  vs.  $27.2 \pm 4.1$ ,  $p = 0.02$ ); the same trend was seen regarding diabetes (26.9% vs. 11.0%,  $p = 0.04$ ). Dyslipidaemia was present more often in patients with a sleep time of > 8 h, whether compared to those sleeping normally (46.2% vs. 24.3%,  $p = 0.03$ ) or to those sleeping less than 6 h (46.2% vs. 19.4%,  $p = 0.03$ ). Patients with a sleep duration of more than 8 h more often suffered from previous MI than those sleeping 6 h to 8 h (38.5% vs. 11.9%,  $p = 0.002$ ). This trend was not seen for the short sleeping group, and similarly there were no differences in the history of percutaneous coronary angioplasty and CABG, except for a more common history of CABG in the group sleeping less than 6 h than the reference category (8.3% vs. 1.3%,  $p = 0.02$ ). Differences in the occurrence of stroke were seen for both groups with a sleep time deviating from the population mean when compared to the reference category. Both patients sleeping less than 6 h and patients sleeping more than 8 h were more often afflicted with stroke than those sleeping between 6 h and 8 h (11.1% vs. 2.8%,  $p = 0.04$  and 15.4% vs. 2.8%,  $p = 0.007$ ). There were no significant differences in the presence of hypertension, pe-

ripheral vascular disease or HF between the groups. We also observed that they were homogenous regarding mean heart rate and systolic and diastolic blood pressure (Table 3).

### Long-term prognosis

There was a statistically significant increase in 3 year all-cause mortality. The 3-year all-cause mortality rate was 1.9% for the reference category, 13.9% for patients who slept less than 6 h, and 30.8% for patients who slept more than 8 h per night ( $p$  value for trend < 0.0001). The correlation between sleep duration and all-cause mortality was U-shaped (Fig. 1).

In the multiple logistic regression analysis, short (odds ratio [OR] 10.2, 95% CI 2.1–50,  $p = 0.004$ ) and long sleep duration (OR 33.3, 95% CI 6.8–163.4,  $p < 0.001$ ) were strong and independent predictors of all-cause mortality. Also BMI higher from the population median (26.7 kg/m<sup>2</sup>) was a predictor of mortality, with a  $p$  value at the border of statistical significance (OR 4.6, 95% CI 0.9–23.9,  $p = 0.07$ ) (Table 4).

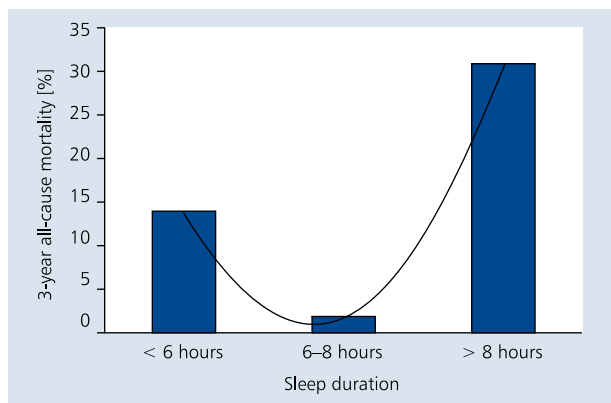
### DISCUSSION

Patients after MI are often on leave from work, undergoing cardiac rehabilitation, paradoxically refraining from or increasing physical activity, or modifying their lifestyle in other ways [13–15]. Regardless of the reason, MI certainly changes the daily routine of most surviving patients, which directly translates into habitual sleep patterns [16]. For some

**Table 3.** Comparison between patients sleeping 6–8 h, less than 6 h, and more than 8 h per night

Characteristics	Sleeping 6–8 h per night (n = 317)	Sleeping less than 6 h per night (n = 36)	Sleeping more than 8 h per night (n = 26)	P value normally sleeping vs. < 6 h	P value normally sleeping vs. > 8 h	P value sleeping < 6 h vs. > 8 h
Age [years]	58.7 ± 10.6	65.7 ± 9.2	58.3 ± 10.1	0.0001	0.83	0.003
Male sex	231 (72.9%)	23 (63.9%)	17 (65.4%)	0.35	0.55	1.00
Body mass index [kg/m <sup>2</sup> ]	27.2 ± 4.1	33.6 ± 32.6	29.6 ± 4.8	0.26	0.02	0.23
Systolic BP [mm Hg]	130.0 ± 25.0	133.8 ± 20.3	134.0 ± 31.1	0.25	0.70	0.97
Diastolic BP [mm Hg]	78.0 ± 17.8	79.2 ± 12.3	78.5 ± 19.0	0.44	0.81	0.47
Heart rate [bpm]	76.2 ± 15.8	77.9 ± 14.4	75.8 ± 13.2	0.55	0.89	0.56
Diabetes mellitus	35 (11.0%)	6 (16.7%)	7 (26.9%)	0.47	0.04	0.36
Hypertension	161 (50.8%)	21 (58.3%)	13 (50.0%)	0.49	0.90	0.61
Dyslipidaemia	77 (24.3%)	7 (19.4%)	12 (46.2%)	0.67	0.03	0.03
Smoking	164 (51.7%)	14 (38.9%)	13 (50.0%)	0.20	0.97	0.44
Peripheral vascular disease	19 (6.0%)	2 (5.6%)	3 (11.5%)	0.79	0.49	0.64
Heart failure (III/IV NYHA)	5 (1.6%)	2 (5.6%)	2 (7.7%)	0.32	0.16	1.00
COPD/asthma	12 (3.8%)	3 (8.3%)	3 (11.5%)	0.40	0.17	0.69
Prior myocardial infarction	44 (11.9%)	6 (16.7%)	10 (38.5%)	0.84	0.002	0.08
Prior PTCA	11 (3.5%)	0 (0.0%)	2 (7.7%)	0.53	0.58	0.17
Prior CABG	4 (1.3%)	3 (8.3%)	1 (3.9%)	0.02	0.84	0.63
Prior stroke	9 (2.8%)	4 (11.1%)	4 (15.4%)	0.04	0.007	0.71
3-year all-cause mortality	5 (1.6%)	5 (13.9%)	8 (30.8%)	0.0002	< 0.0001	0.13

BP — blood pressure; CABG — coronary artery bypass grafting; COPD — chronic obstructive pulmonary disease; NYHA — New York Heart Association; PTCA — percutaneous transluminal coronary angioplasty



**Figure 1.** Three-year all-cause mortality according to sleep duration

patients, it may mean that their sleep time will be prolonged over 8 h per night, for others that it will be shorter than 6 h, no matter how long they slept prior to MI. Both short and, especially, long, sleep duration are predictors of cardiovascular outcomes [1]. This compels us to look for a correlation between sleep duration after MI and the long-term outcome of patients including all-cause mortality.

**Table 4.** Multiple logistic regression analysis for predictors of all-cause mortality

	OR	95% CI	P
Sleep duration < 6 h vs. 6–8 h	10.2	2.1–50	0.004
Sleep duration > 8 h vs. 6–8 h	33.3	6.8–163.4	< 0.001
Age > median			> 0.1
Sex: male vs. female			> 0.1
History of hypertension			> 0.1
Smoking			> 0.1
Diabetes mellitus			> 0.1
Dyslipidemia			> 0.1
Body mass index > median (26.7 kg/m <sup>2</sup> )	4.6	0.9–23.9	0.07

Area under the ROC curve = 0.88; CI — confidence interval; OR — odds ratio

Sleep disorders are highly prevalent in the general population, affecting up to 20% of adults in highly developed countries [17]. Disturbances in the quality of sleep, led by the most common of them, obstructive sleep apnoea syndrome, are known to have an adverse effect on patient health, which is associated with an increased risk of HF, stroke, pulmonary hy-

pertension, ischaemic heart disease, ventricular and supraventricular arrhythmias and sexual dysfunction [18–21]. In this case, the majority of the underlying causes, like sympathetic activation due to hypoxia and hypercapnia, elevated plasma levels of inflammatory markers and cell expression of several adhesion molecules, as well as oxidative stress, seem to be established [19, 22]. The mechanism of how sleep duration by itself, irrespective of sleep quality, affects patients' general condition is more ambiguous. It was previously shown that there is a U-shaped relationship between sleep duration and adverse health outcomes, in our patients' case expressed by the higher 3-year all-cause mortality in both sleeping less than 6 h and more than 8 h than the reference category [1]. In our study, sleep duration deviant from the population mean in 3 months after STEMI was also associated with increased all-cause 3-year mortality.

It seems that different mechanisms are responsible for increases at either end of the curve. Short sleep is associated with metabolic changes, such as increased levels of inflammatory cytokines, leptin, ghrelin, growth hormone and cortisol, which would promote the development of obesity and impaired glucose control, contributing to increased CVD risk [1, 5, 23, 24]. On the other hand, long sleep is considered not as a cause, but more as an outcome, of a patient's general condition. Sleeping more than 8 h per night has been found to be associated with the coexistence of a number of comorbidities and is considered to be their manifestation [25]. This was confirmed in our long-sleeping patients, who had more often a history of diabetes mellitus, dyslipidaemia, or prior MI and stroke.

Multivariable logistic regression analysis proved that sleep duration was an independent predictor of patient outcome. Generally, patients with deviant from the population mean sleep length had more often undergone MI, stroke, or CABG in the past. The history of diabetes mellitus was at the border of statistical significance. Separation into three sleep-duration groups proved some previous findings, and showed also new correlations. As proven in meta-analysis, both too long and too short sleeping groups had more often a history of stroke. Beyond the previously disclosed, we have seen some differences in the presence of dyslipidaemia and diabetes which were driven by the long-sleeping group, while the short sleeping group was proven to be older and more often have a history of CABG. As for diabetes, some studies suggest that short-sleeping patients are twice as likely, and long-sleeping patients three times as likely, as the reference group to develop diabetes [3]. As previously mentioned, in our study this was not seen for those sleeping for less than 6 h.

Our findings about the prevalence of most of the comorbidities are consistent with previous studies [1, 12, 26, 27]. But contrary to some others [28, 29], we did not observe statistically significant differences between groups in the prevalence of hypertension or mean systolic and diastolic blood pressure values. This is particularly interesting, because some authors

have even pulled out far-fetched conclusions that obtaining adequate sleep duration may be considered as a potential part of treatment in patients with hypertension [28].

### *Limitations of the study*

The present study has a relatively small population but concerns an area without a lot of data. The major limitation of the study is no information on pre-infarct sleep time. This data would help to determine whether the STEMI itself is a factor changing sleep duration and therefore affecting prognosis, or whether the more important link is the one between sleep time before STEMI and the outcome. Also, an adequate description of residual confounders, such as depression, comorbidities related to sleep (sleep apnoea or prostate disease), medications, or polypharmacy and an objective method for measuring sleep duration should be considered in further research. But with that said, this study highlights a key patient-centred variable related to quality of life and with high biologic plausibility in linking with poor outcomes. The reference group was defined as 6 h to 8 h based on large population studies, but some data shows that a sleep time of 6 h in some cases may be considered too short, whereas 9 h is in some cases considered normal [1, 3, 10, 30]. In our study group, the abovementioned cut-off criteria (< 6 h, 6–8 h and > 8 h) were shown to be predictive of mortality, but larger population studies have to be performed in order to validate these criteria.

### **CONCLUSIONS**

Patients with sleep duration above 8 h and below 6 h in the 3 months after STEMI are both affected by an excess risk of death and higher prevalence of comorbidities. The quantity of sleep, regardless of the quality, affects patients' general outcome. Sleep length after MI is an important predictor of long-term prognosis in our patients and even may be considered as a novel risk factor of CVD, but whether its modification would contribute positively to the general outcome remains a subject for further investigation. A further study is needed to confirm these results.

*Conflict of interest: none declared*

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# Czas trwania snu w pierwszych miesiącach po zawale serca z uniesieniem odcinka ST: niezależny czynnik ryzyka zgonu

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## Streszczenie

**Wstęp:** Czas trwania snu i jego jakość wpływają na ogólny stan zdrowia pacjentów. Uznaje się, że średni czas trwania snu od 6 do 8 h w ciągu nocy nie wiąże się z negatywnym wpływem na stan zdrowia pacjentów. Z kolei czas trwania snu dłuższy lub krótszy niż przedstawiony powyżej przedział może się wiązać z częstszym występowaniem u pacjentów: nadciśnienia tętniczego, cukrzycy typu 2, otyłości, dyslipidemii i/lub z niższą subiektywną oceną stanu zdrowia. Wszystkie te stany są dobrze znanymi czynnikami ryzyka chorób układu sercowo-naczyniowego. Dlatego też zbyt krótki lub nadmiernie długi czas snu może być łatwym do oceny markerem ryzyka tych chorób (np. choroby niedokrwiennej serca).

**Cel:** Celem niniejszej pracy było opisanie charakterystyki klinicznej pacjentów z zawałem serca z uniesieniem odcinka ST (STEMI), w przypadku których stwierdzono zbyt długi lub zbyt krótki czas trwania snu, oraz ustalenie, czy czas trwania snu jest niezależnym czynnikiem zgonu.

**Metody:** Do badania włączono 407 pacjentów hospitalizowanych z powodu STEMI, którzy zgłosili się do lekarza w ciągu 12 h od wystąpienia objawów. Po okresie hospitalizacji wszyscy pacjenci zostali poproszeni o podanie swojego średniego czasu trwania snu w pierwszych 3 miesiącach po zawale serca. Ze względu na ten parametr przydzielono pacjentów do 3 grup: A — grupa referencyjna (śpiąca 6–8 h), B — krótki czas snu (< 6 h) i C — długi czas snu (> 8 h). Pierwotnym punktem końcowym ocenianym w badaniu była śmiertelność całkowita.

**Wyniki:** Ostateczną analizą objęto 379 pacjentów (271 mężczyźni; średni wiek  $59,4 \pm 10,61$  roku); 36 (9,5%) osób spało < 6 h, a 26 (6,9%) > 8 h. Całkowita, 3-letnia śmiertelność wynosiła 1,9% w grupie A, 13,9% w grupie B i 30,8% w grupie C ( $p < 0,0001$ ). Analiza wieloczynnikowa pokazała, że krótki (OR 10,2; 95% CI 2,1–50;  $p = 0,004$ ) i długi (OR 33,3; 95% CI 6,8–163,4;  $p < 0,001$ ) czas snu są niezależnymi czynnikami ryzyka zgonu.

**Wnioski:** Czas trwania snu powyżej 8 lub poniżej 6 h w pierwszych miesiącach po zawale serca stanowi silny, niezależny czynnik ryzyka śmiertelności całkowitej. Czas trwania snu, niezależnie od jego jakości, wpływa na rokowanie pacjentów.

**Słowa kluczowe:** czas trwania snu, zawał serca, śmiertelność

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