Int.J. Behav. Med. (2012) 19:526–534
DOI 10.1007/s12529-011-9205-2

Poor Sleep Quality is Associated with Depressive Symptoms in Patients with Heart Disease

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Published online: 29 November 2011

Abstract
Background Depression in cardiac patients has gained importance due to increased mortality. Although sleep disturbances are a core symptom of depression, the prevalence and patterns of sleep disturbances in heart disease have hardly been examined regarding depression.
Purpose This cross-sectional study aims to examine sleep disturbances and depressive symptoms in consecutively admitted cardiac patients and depressed patients.
Methods Two hundred four inpatients (113 male, 91 female) were examined: 94 cardiac inpatients (mean age 49.3±14.3 years) with different heart diseases and 110 psychiatric inpatients (mean age 41.6±13.0 years) with depressive disorders (DP). A depressive episode according to International Classification of Diseases (ICD)-10 was also diagnosed in 14 of the cardiac patients (DCP). The Pittsburgh Sleep Quality Index (PSQI) and the Beck Depression Inventory (BDI) were used to assess subjective sleep quality and severity of depressive symptoms.
Results Poor sleep quality (PSQI>5) was reported in all comorbid DCP (PSQI 12.00±3.53, BDI 17.86±4.28), in 60% of the 80 non-DCP (PSQI 5.59±3.73, BDI 4.47±3.07), and in 86.4% of the DP (PSQI 11.76±4.77, BDI 27.11±10.54). The cardiac inpatients showed a significant correlation between increased depressive symptoms and the PSQI components subjective *sleep quality* (r=0.40) and *daytime dysfunction* (r=0.34). Both sleep components were significant predictors of self-rated depression (R^2=0.404).
Conclusions Most cardiac patients experience poor sleep quality. Self-reported sleep disturbances in heart disease could serve as predictors of clinical or subclinical comorbid depression outside of a psychiatric setting in cardiology and other fields, and such patients should be referred to consultation-liaison psychiatry or polysomnography where sleep disorders like sleep apnea are suspected.

Keywords Sleep quality · Depression · Coronary heart disease · Chronic heart failure · Comorbidity · Sleep disturbances

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Introduction

Sleep disturbances are very common and have a major impact on physical and mental health. About one third of the general population suffers from insomnia [1]. Yet, complaints of insomnia or hypersomnia lead to a 2.5-fold increase in mental disorders [2]. Sleep disturbances also are a core symptom of depression [3], a disorder with a high point prevalence ranging from 2% to 9% [4]. Insomnia in particular has been identified as a significant predictor of a subsequent depressive disorder [4, 5].

Sleep disturbances also coincide with chronic diseases, depressive disorders and heart disease. Generally, chronic insomnia is found in almost half the patients with chronic disease [6], i.e. with substantially increased mild and severe insomnia on a 2-year follow-up (odds ratios (OR) 2.6 and 8.2 for a current depressive disorder, 2.2 and 3.4 for sub-threshold depression; 1.6 and 2.5 for chronic heart failure, 1.6 and 1.5 for obstructive airways disease). A recent population-based study in the UK showed a clear association between sleep disorders and prior stress and depression as well as prior circulatory diseases including chronic heart failure (CHF) and coronary heart disease (CHD) [7]. These problems are particularly urgent because heart disease and depression are the two leading causes of future disability in developed countries [8].

At present, comorbid depression in cardiovascular disease has gained special attention due to increased morbidity and mortality, especially in the highly prevalent CHF and related cardiopulmonary diseases [9–14]. Depressive symptoms are seen in 24–85% of CHF patients depending on the setting of the study, while 14–26% develop major depression [9]. In a recent British survey, the 12-month prevalence of major depression was 7.9% for patients with CHD (OR 2.3), 9.3% for CHF (OR 1.96), and 15.4% for chronic obstructive pulmonary disease (OR 3.21) [10]. Regarding the type of clinical symptoms, chronic heart disease is often associated not only with depression, but also with fatigue or insomnia [15], thus overlapping with depressive syndromes exhibiting fatigue, insomnia, low mood, loss of interest, loss of energy, weight loss and inability to concentrate [4, 16, 17]. Heart disease and depression also show overlapping biological similarities relevant for pathogenesis like neurohormonal overstimulation of the hypothalamic–pituitary–adrenocortical and sympathetic adrenal medullary systems, impaired heart rate variability and cardiac rhythm disorders, inflammation and hypercoagulability [9, 18–22], all of which are known to correlate with depression, anxiety, sleep disturbances or loss of appetite.

Still, the impact of sleep disturbances on the bidirectional interaction between heart and brain disease remains unclear. Poor sleep or altered sleep duration in CHD patients can cause unfavourable cardiac outcomes [23–26]. While difficulties initiating sleep and a one-item depression measure were identified as variables related to CHD mortality in males [27], other studies did not report positive results on sleep measures and depression scales in cardiovascular disease [28, 29]. However, prior to their acute myocardial infarction (MI), 39% of patients reported insomnia lasting 2 weeks or longer and even 48% in the subgroup with a major depressive episode [30]. By contrast, using sleep questionnaires, Leineweber et al. [24] found no association of poor sleep in female CHD patients with depressive symptoms or coronary risk factors. In fact, only two recent studies point to a link of poor sleep quality in CHD with depression [31] or with the psychological dimension of anger suppression [32].

So far, little is known about the association of sleep quality and depression in cardiac patients. The prevalence and patterns of subjective sleep disturbances in inpatients with heart disease (who often have more than one cardiac diagnosis) have hardly been examined regarding depressive symptoms. In order to better identify cardiac patients at risk of developing a depressive disorder, this study examined: (1) differential patterns of subjective sleep disturbances in patients with either depression, cardiac morbidity or both; (2) whether self-reported poor sleep quality can function as a general predictor of depressive syndromes in heart disease.

Materials and Methods

Subjects

A total of 204 inpatients were recruited, comprising 94 consecutively admitted cardiac patients (mean age, 49.3± 14.3 years) from a cardiological ward and 110 psychiatric patients (mean age, 41.6±13.0 years) treated for a depressive disorder on psychiatric wards specialising in affective disorders: the Departments of Cardiology and Psychiatry of the Technical University Hospital (Aachen), the Alexianer Hospital (Aachen), the EOS Hospital (Münster), all in Germany (Table 1). Exclusion criteria were performance-limiting conditions like amnesia, no fluency in German and inability to concentrate for 1.5 h for the interview and self-rating questionnaires. All patients participated voluntarily within 3 days after admission, were not paid and gave informed consent prior to testing. The study was approved by the local ethics committee.

Measurements

Demographic Characteristics and Clinical Diagnosis

All patients completed a demographic data sheet. Additional clinical data, including diagnosis of heart disease according to International Classification of Diseases (ICD)-10 [33], were taken from medical records. A diagnosis of depressive disorder was confirmed in all psychiatric inpatients by a senior psychiatrist using a clinical interview according to the International Diagnostic Checklist for depression [34] based on ICD-10 [33] and DSM-IV. A depressive episode also was diagnosed in 14 of 94 cardiac patients (DCP) at the time of the survey. All subjects completed the following self-rating questionnaires assessing mood and sleep quality.

Table 1 Characteristics of the total study sample

	Depressive psychiatric inpatients (DP; n=110)		Depressive cardiac inpatients (DCP; n=14)		Non-depressive cardiac inpatients (non-DCP; n=80)	
Male/female (n)	66/44		7/7		18/62	
Age in years (M±SD)	42±13		50±12		49±14	
Diagnostic subgroups[a] (ICD-10)	Individuals treated for:	Percentage, %	Individuals treated for:	Percentage, %	Individuals treated for:	Percentage, %
	Bipolar disorder (F31.xx)	9.1	Chronic ischaemic heart disease (I25.xx)	57.1	Chronic ischaemic heart disease (I25.xx)	46.3
	Depressive episode (F32.xx)	39.1	Heart failure (I50.xx)	14.3	Heart failure (I50.xx)	21.3
	Recurrent depressive disorder (F33.xx)	26.3	Cardiomyopathy (I42.xx)	21.4	Cardiomyopathy (I42.xx)	22.5
	Persistent affective disorder (F34.xx)	3.6	Atrial fibrillation (I48.xx)	28.6	Atrial fibrillation (I48.xx)	31.3
	Adjustment disorder (F43.2)	25.2	Ventral tachycardia/ Arrhythmia (I47.2, I49.xx)	21.4	Ventral tachycardia/ Arrhythmia (I47.2, I49.xx)	18.8
	Personality disorder (F60.xx)	20.0	Angina pectoris (I20.xx)	21.4	Angina pectoris (I20.xx)	25.0
	Substance misuse (F10.xx)	13.6	Myocardial infarction (I21-I22.xx)	14.3	Myocardial infarction (I21-I22.xx)	17.5
	Others (ICD-F)	5.4	Valvular disorders (I34-37.xx)	14.3	Valvular disorders (I34-37.xx)	26.3
			Cardiac valve disease (I39.xx)	7.1	Cardiac valve disease (I39.xx)	8.8
			Others (ICD-I)	14.3	Others (ICD-I)	12.5

[a] Diagnoses within diagnostic groups are not mutually exclusive

Beck Depression Inventory

The Beck Depression Inventory (BDI) [35] is a self-rating instrument for examining depressive symptoms in patients with clinical depression. Patients were asked to rate 21 items (range 0–3) which most closely represented their mental state during the previous week. A total score of >10 indicating mild to moderate depressed syndromes was used as cutoff value.

Pittsburgh Sleep Quality Index

The Pittsburgh Sleep Quality Index (PSQI), a self-rating questionnaire [36], was used to assess sleep quality during the previous 4 weeks in seven component scores (range 0–3): *sleep quality*; *sleep onset latency*; *sleep duration*; *habitual sleep efficiency*; *sleep disturbances*; *use of sleeping medication*; and *daytime dysfunction*. A *global score* of >5 indicates symptoms of disturbed sleep, a higher sum score reflecting poorer subjective sleep quality [37].

Statistical Analyses

All data analyses except calculation of effect sizes were carried out using SPSS for Windows 17.0. A multivariate analysis of variance (MANOVA) with groups (DP, depressive; DCP, depressive cardiac; non-DCP, non-depressive cardiac inpatients) as between-subjects factor was conducted. Covariates were included in MANOVA to control for influences of age, gender and sedatives. $P<0.05$ was considered significant for all comparisons.

Mauchly's sphericity test was conducted prior to MANOVA. Sphericity indicates variance equality of the differences between measurements. If the significance level of Mauchly's test is below $P=0.05$, sphericity cannot be assumed. If Mauchly's sphericity test is significant, then the Greenhouse–Geisser correction is used to adjust the degrees of freedom used to calculate P for the F value. If MANOVA yielded significant results, bias-corrected effect sizes of all sleep components were calculated post hoc to examine the significant ANOVA results in greater detail [38, 39]. In this study, Cohen's d was used as an effect size (ES) to indicate the standardised difference between two means, with ES $0.20<d\leq0.50$ being small, $0.50<d\leq0.80$ medium, and $d\geq0.80$ large [38]. In order to reduce sampling error, ES were corrected using a factor provided by Hedges [39].

Linear regression analysis was performed to determine whether sleep components, age, gender or sedatives (including tranquillizers, anxiolytics and hypnotics) significantly predicted depression across inpatients with heart disease.

Results

Overall Sleep Quality and Depression

Of the 204 patients, poor sleep quality (i.e. PSQI global score >5) was found in 86.4% of DP (PSQI 11.76±4.77, BDI 27.11±10.54), 60% of the 80 non-DCP (PSQI 5.59± 3.73, BDI 4.47±3.07), and all 14 DCP (PSQI 12.00±3.53, BDI 17.86±4.28).

Components of Sleep Quality

Since Mauchly's test of sphericity was significant (Mauchly-W=0.25, approximate χ^2=268.49, P=0.00, df=20), all MANOVA results were adjusted using the Greenhouse–Geisser correction. Table 2 shows the effect of the within-subjects factor components of disturbed sleep (PSQI) and the covariates (sedatives, gender, age).

Differences in sleep quality were examined between the three patient groups. There was a significant between-subjects effect (F=8.99; df=7; P<0.01), with profiles of sleep component scores differing significantly between the three groups. Tests of univariate effects revealed that the three study groups differed significantly on all sleep component scores (Table 2).

None of the covariates included in the analysis had a significant main effect, indicating that differences between the groups were generally not biased by age, gender or sedatives (Table 2, right columns). Only "sedatives" showed a significant interaction with the component score "*use of sleeping medication*" which was expected. Even treating gender as a between-subjects factor did not change the overall picture: There was no significant main effect of gender as between-subjects factor (F=0.62, p=0.74), nor was there a significant interaction with the factor group (F=1.33, p=0.19).

Table 2 Tests for multi- and univariate within-subjects effects

	df	*F*	*P*
Multivariate tests			
Constant term	7	8.97	0.00
Groups	14	8.99	0.00
Gender	7	1.28	0.26
Age	7	1.36	0.23
Sedatives	7	1.70	0.11
Univariate tests (groups)			
Subjective sleep quality	2	29.31	0.00
Sleep latency	2	19.86	0.00
Sleep duration	2	10.09	0.00
Habitual sleep efficiency	2	6.64	0.00
Sleep disturbances	2	13.25	0.00
Use of sleeping medication	2	14.00	0.00
Daytime dysfunction	2	35.07	0.00

Univariate effects are shown only for "groups", since no further significant results were found

Since MANOVA gave partly significant results, effect sizes were calculated to further examine the strengths of the differences in sleep component scores (PSQI) between each of the three groups (Table 3). Unlike non-DCP, DP and DCP both showed poorer sleep quality on all seven component scores (PSQI; Fig. 1).

The effects between the two groups of inpatients with depression (DP, DCP) were strongest for the *sleep disturbances* component (d=0.53). Otherwise, DP and DCP showed mostly similarities on *sleep latency* (d= 0.10). The effect sizes of the remaining components for DP and DCP varied within a range of d=0.15–0.32 (*daytime dysfunction* d=0.15, *habitual sleep efficiency* d=0.16, *subjective sleep quality* d=0.22, *sleep duration* d=0.30, *use of sleeping medication* d=0.32). The effect sizes between DP and DCP compared with non-DCP on all seven components of disturbed sleep (PSQI) were much greater. However, components of *subjective sleep quality, sleep latency, sleep duration* and *sleep disturbances* showed greater effect sizes for DCP vs. non-DCP than for DP vs. non-DCP. On the other hand, components of *habitual sleep efficiency*, *use of sedatives* and *daytime dysfunction* showed greater effect sizes for DP vs. non-DCP than for DCP vs. non-DCP.

Symptoms of Disturbed Sleep as a Predictor for Depression

Stepwise regression analysis showed *subjective sleep quality* (r=0.40) and *daytime dysfunction* (r=0.34) to be most strongly associated with depressive symptoms (BDI) as the dependent variable. Both *subjective sleep quality* and *daytime dysfunction* helped predict depressive syndromes (BDI), with R^2=0.404. Thus, 40.4% of the response variability (depression) was explained by *subjective sleep quality* and *daytime dysfunction*. The remaining five components of disturbed sleep (PSQI) as well as the covariates sedatives, age and gender were not significantly related to depression (BDI).

Discussion

As hypothesized, more than two thirds of the cardiac patients in this study complained of sleep disturbances. (1) Of 204 inpatients, 110 depressed patients (DP; 86.4%) reported poor sleep quality. This confirms previous findings that approximately 90% of inpatients with major depressive disorder exhibit some sleep disturbances [3, 40], supporting the well-known relationship between depression and im-

Table 3 Magnitude of ES between components of disturbed sleep (PSQI) with all of the analysis groups

Outcome measure	Group X vs. Group Y		Bias-corrected ES (Hedges)	Standard error of ES estimate
	Mean±SD	Mean±SD		
Subjective sleep quality				
DP vs. DCP	1.89±0.84	2.07±0.62	−0.22	0.28
DP vs. non-DCP	1.89±0.84	0.98±0.73	1.15	0.16
Non-DCP vs. DCP	0.98±0.73	2.07±0.62	−1.52	0.31
Sleep latency				
DP vs. DCP	1.96±1.01	2.07±1.14	−0.10	0.28
DP vs. non-DCP	1.96±1.01	0.96±0.91	1.03	0.16
Non-DCP vs. DCP	0.96±0.91	2.07±1.14	−1.17	0.30
Sleep duration				
DP vs. DCP	1.56±1.25	1.93±0.92	−0.30	0.28
DP vs. non-DCP	1.56±1.25	0.81±1.01	0.64	0.15
Non-DCP vs. DCP	0.81±1.01	1.93±0.92	−1.11	0.30
Habitual sleep efficiency				
DP vs. DP	1.71±1.29	1.50±1.23	0.16	0.28
DP vs. non-DCP	1.71±1.29	0.94±1.11	0.63	0.15
Non-DCP vs. DP	0.94±1.11	1.50±1.23	−0.50	0.29
Sleep disturbances				
DP vs. DCP	1.36±0.66	1.71±0.61	−0.53	0.29
DP vs. non-DCP	1.36±0.66	0.95±0.57	0.66	0.15
Non-DCP vs. DCP	0.95±0.57	1.71±0.61	−1.31	0.31
Use of sleeping medication				
DP vs. DCP	1.36±1.36	0.93±1.33	0.32	0.28
DP vs. non-DCP	1.36±1.36	0.24±0.66	1.00	0.16
Non-DCP vs. DCP	0.24±0.66	0.93±1.33	−0.87	0.30
Daytime dysfunction				
DP vs. DCP	1.92±0.87	1.79±0.80	0.15	0.28
DP vs. non-DCP	1.92±0.87	0.71±0.75	1.46	0.16
Non-DCP vs. DCP	0.71±0.75	1.79±0.80	−1.40	0.31

Analysis groups: *DP* depressive psychiatric inpatients, *DCP* depressive cardiac inpatients, *Non-DCP* non-depressive cardiac inpatients

pairment of sleep quality. However, self-estimated poor sleep quality was also found (2) in all 14 cardiac patients with a diagnosed depressive episode (DCP), and most importantly, (3) in 60% of the 80 non-depressed cardiac patients (non-DCP) who did not qualify for a clinical diagnosis of depression.

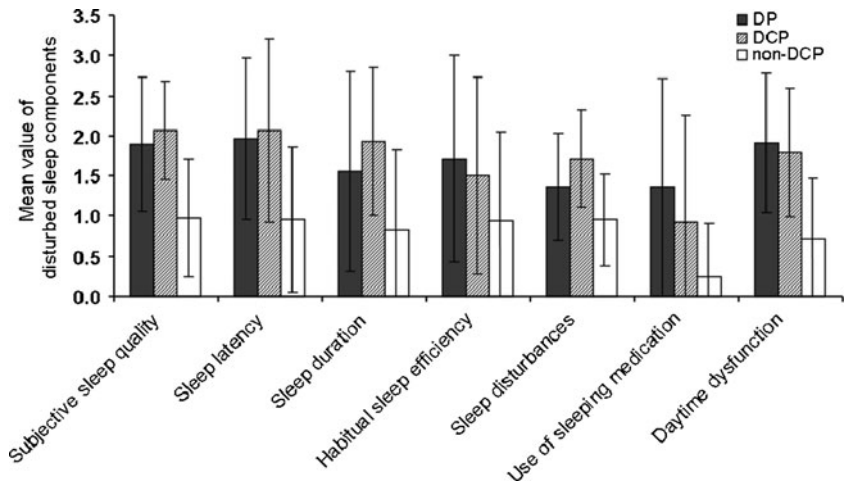

Fig. 1 Mean values of disturbed sleep components measured using the Pittsburgh Sleep Quality Index (PSQI) in depressive inpatients (*DP*), depressive cardiac inpatients (*DCP*) and non-depressive cardiac inpatients (*non-DCP*)

The high rate of 68.9% of poor sleep quality in the *global PSQI score* for inpatients with different heart disease is very similar to findings for CHD inpatients (69.9% of 163 patients) [31], in stable CHF outpatients (73.1% of 170) [41] and our earlier pilot study in a small CHF inpatient sample (68% of 35) [42]. By contrast, only 28% of stable CHD patients in the Heart and Soul Study reported low sleep quality, but this result came from a large cohort of 1,020 outpatients in a different setting who were only given a one-item question on overall sleep quality extracted from the PSQI [32].

The greatest differences in the component scores between depressive inpatients (DP and DCP) and non-DCP were seen for subjective sleep quality and daytime dysfunction. Saleh et al. [31] reported similar findings for 34 depressive and 129 non-depressive CHD patients: Only the global score and the daytime dysfunction component were significantly worse for the depressive CHD patients, not the other PSQI components. Significant decreases of other PSQI component scores in the CHD patients were seen only in the more anxious CHD group, but the patients were not given a diagnostic psychiatric interview [31]. Depression and anxiety symptoms often overlap in cardiac patients, but this study concentrated on depression. Otherwise, there is only one study dealing with psychological distress, i.e. anger suppression (anger-in), a potential psychological risk factor for cardiovascular disease. It was found to be associated with low sleep quality in stable CHD [37].

Moreover, a linear regression analysis of the seven PSQI sleep components and covariates showed that *subjective sleep quality* and *daytime dysfunction* were strong predictors of depressive syndromes in cardiac inpatients (R^2= 0.404). Interestingly, the scores of the same PSQI components were most strongly related to low positive affect in a recent study on repeated ambulatory mood measurements of 96 subjects with major or minor depression and healthy controls [43]. *Subjective sleep quality* and *daytime dysfunction* both fall in line with the combination of three sleep complaints (difficulty falling asleep, waking up repeatedly, awakening tired and fatigued) which were previously identified as predictors for an increased risk of cardiovascular disease [44].

In this study, the comorbidity of depression and heart disease in the DCP group seems to be related to several component scores of poor sleep quality (i.e. *subjective sleep quality, sleep latency, sleep duration, sleep disturbances*) as compared to DP and non-DCP. Likewise, Hayashino et al. [45] showed that poor sleep quality correlated positively not only with depression, but also with the number of comorbid somatic conditions. Moreover, prior insomnia in patients with acute MI who did not meet the criteria for a major depressive episode was related to three times as many depressive symptoms as in non-insomniac MI patients [30]. Therefore, subjective poor sleep quality occurring together with symptoms of clinical or subclinical depression should not be underestimated in cardiac patients with multiple comorbid conditions.

Daytime dysfunction in the sense of daytime sleepiness also represents one major symptom of sleep apnea. Central or obstructive sleep apnea resulting in adverse effects on the cardiovascular system and decreased brain functions with intermittent cerebral hypoxia and hypercapnia represent one of the commonest causes of sleep disturbances in chronic heart disease [46–48]. These sleep-related breathing disorders (SRBDs) may cause excessive daytime sleepiness, sleep fragmentation, increased mortality and impaired quality of life in patients with heart disease [46, 48–50]. Since depression and SRBDs in heart disease affect each other [49–52], sleep apnea represents an independent risk factor for developing depressive syndromes, whereas treating it improves symptoms like daytime sleepiness, depressive mood and quality of life [42, 53, 54]. This study did not specifically examine daytime sleepiness or SRBDs, so an association between SRBD and reported sleep disturbances or depressive symptoms cannot be excluded. Nevertheless, by only assessing the degree of excessive daytime sleepiness in different situations [46, 55], it is not possible to rule out obstructive sleep apnea (OSA) in chronically ill cardiac patients [56, 57] since no correlation was found for daytime sleepiness or self-reported sleep with severe SRBDs despite low objective sleep quality [41].

This study showed gender and age as non-significant covariates. Presumably, this was because only 15% of patients in the cohort were 60 or older, while complaints of sleep disturbances and depression in older patients tend to be commoner in women [3]. However, in chronic cardiac patient cohorts, depressed subjects tend to be not only female [24], but also younger [58]. Still, clinicians need to be aware that elderly adults show a decline in slow-wave sleep with increasing age, and often experience lighter and more fragmented sleep [59].

Regarding medication, this study took into account whether inpatients were given any sedatives at the time of the survey without any effects showing in this cohort. One shortcoming is that it was not possible to include the previous duration of sedatives use or the quantity of somatic, particularly heart medication. A number of cardiovascular drugs are accompanied by psychotropic effects [9, 55, 60], either depressing or sedative, but this was not observed here. Furthermore, the cardiac inpatients were on supervised multidrug therapy, and hence likely to be compliant and under optimised treatment, or the sleep problems could have been worse. On the other hand, DPs showed a more pronounced decline of habitual sleep efficiency, daytime dysfunction and greater use of sleeping

medication compared to the other two groups, most probably reflecting the primary mental disorder and its treatment on the psychiatric ward.

This study has further limitations. First, the sample of inpatients was rather small, although statistics showed all comparisons to be valid. The psychiatric sample included only inpatients with a diagnosed depressive disorder from wards that specialized in treating affective disorders. While this in itself is a strength, the time frame of the study meant recruiting at the university hospital and two other psychiatric hospitals, which may have introduced a potential bias, while the cardiac patients all came from the same place. Second, as already pointed out, the sleep disturbances were assessed only on a subjective basis. While obtaining polysomnographic data of all patients, screening for underlying sleep disorders would have been desirable, it would also have meant greatly expanding the study design. The short questionnaires were used only for a quick clinical screening and to allow further differential diagnoses. Notably, sleep disorders like OSA, restless legs syndrome, primary insomnia, narcolepsy and others are known not only to lower sleep quality but are also associated with depression, although the two-way pathogenesis has not yet been explained sufficiently [61, 62]. Moreover, sleep laboratory measures may correlate with subjective sleep quality but they cannot define it [36]. People usually do not experience their sleep behaviour actively while asleep, but they can experience symptoms of disturbed sleep such as difficulties falling asleep, feeling rested upon awakening, daytime impairments or sleepiness [36]. Thus, self-rated daytime dysfunction still seems the most appropriate indirect way to measure consequences of sleep disturbances. Third, due to the exclusive clinical rating character of the study, additional somatic data like cardiovascular risk factors were not taken into account, so that other comorbid organic factors cannot be ruled out. In particular, the depressive inpatients were not seen by a cardiologist. Further drug effects or psychosocial factors which may relate to the social environment and psychophysiological changes (e.g. low emotional support, social isolation, medical non-compliance, low social-economic status, diet, smoking, drinking) and also correlate with depression and heart disease [9, 63, 64] but that were not included must be considered. These factors would have to be examined in larger, follow-up cardiac studies on depression and sleep disturbances.

In conclusion, there is a need to focus more on subjective sleep disturbances in heart disease. Since the *subjective sleep quality* and *daytime dysfunction* component were specifically identified as predictors of depressive syndromes in cardiac inpatients, clinicians could improve a patient's health by quickly screening for these—although at present there is only insufficient evidence that depression screening and collaborative care indeed improve cardiovascular outcomes [10, 65, 66]. Still, taking a transdiagnostic perspective [67] and assessing sleep patterns in a one-target protocol can help cardiologists and clinicians other than psychiatrists identify promptly or transfer cardiac patients to consultation–liaison psychiatry, e.g. because of suspected depression [9–12, 16, 17] or to further sleep diagnostics like polysomnography, as with suspected OSA due to loud snoring, daytime somnolence and apnea [42, 47–52]. Screening and subsequent early treatment of sleep disturbances in patients with heart disease may reduce comorbid symptoms of depression or associated processes, or even prevent a future depressive disorder altogether.

Acknowledgements We thank the Departments of Psychiatry and Cardiology of the Technical University Hospital (Aachen), the Alexianer Hospital (Aachen) and the EOS Hospital (Münster), Germany, for recruiting patients for this study.

Declaration of Conflicting Interests The authors declare that there were no conflicts of interest.

References

1. Ohayon M. Epidemiology of insomnia: what we know and what we still need to learn. Sleep Med Rev. 2002;6:97–111.
2. Ford DE, Kamerow DB. Epidemiologic study of sleep disturbances and psychiatric disorders. An opportunity for prevention? JAMA. 1989;262:1479–84.
3. Tsuno N, Besset A, Ritchie K. Sleep and depression. J Clin Psychiatry. 2005;66:1254–69.
4. American Psychiatric Association (APA). Diagnostic and statistical manual of mental disorders. 5th edn. DSM-IV-TR. Washington, DC: APA; 2000.
5. Riemann D, Voderholzer U. Primary insomnia: a risk factor to develop depression? J Aff Dis. 2003;76:255–9.
6. Katz DA, McHorney CA. Clinical correlates of insomnia in patients with chronic illness. Arch Intern Med. 1998;158:1099–107.
7. Wallander MA, Johansson S, Ruigómez A, García Rodríguez LA, Jones R. Morbidity associated with sleep disorders in primary care: a longitudinal cohort study. Prim Care Companion J Clin Psychiatry. 2007;9:338–45.
8. Murray CJ, López AD, Murray CJ, López AD. Global mortality, disability, and the contribution of risk factors: Global Burden of Disease Study. Lancet. 1997;349:1436–42.
9. Norra C, Skobel EC, Arndt M, Schauerte P. High impact of depression in heart failure: early diagnosis and treatment options. Int J Cardiol. 2008;125:220–31.
10. Egede LE. Major depression in individuals with chronic medical disorders: prevalence, correlates and association with health resource utilization, lost productivity and functional disability. Gen Hosp Psychiatry. 2007;29:409–16.
11. Glassman AH, Bigger Jr JT, Gaffney M. Psychiatric characteristics associated with long-term mortality among 361 patients having an acute coronary syndrome and major depression: seven-year follow-up of SADHART participants. Arch Gen Psychiatry. 2009;66:1022–9.
12. Lichtman JH, Bigger JT, Blumenthal JA, Frasure-Smith N, Kaufmann PG, Lespérance F, et al. Depression and coronary

heart disease. Recommendations for screening, referral, and treatment. Circulation. 2008;118:1768–75.
13. Osborn DP, Levy G, Nazareth I, Petersen I, Islam A, King MB. Relative risk of cardiovascular and cancer mortality in people with severe mental illness from the United Kingdom's General Practice Research Database. Arch Gen Psychiatry. 2007;64:242–9.
14. Lanfranchi PA, Braghiroli A, Bosimini E, Mazzuero G, Colombo R, Donner CF. Prognostic value of nocturnal Cheyne-Stokes respiration in congestive heart failure. Circulation. 1999;99:1435–40.
15. Juenger J, Schellberg D, Kraemer S, Haunstetter A, Zugck C, Herzog W, et al. Health related quality of life in patients with congestive heart failure: comparison with other chronic diseases and relation to functional variables. Heart. 2002;87:235–41.
16. Goldman LS, Nielsen NH, Champion HC. Awareness, diagnosis, and treatment of depression. J Gen Int Med. 1999;14:569–80.
17. Whooley MA. To screen or not to screen? Depression in patients with cardiovascular disease. J Am Coll Cardiol. 2009;54:891–3.
18. Musselman DL, Evans DL, Nemeroff CB. The relationship of depression to cardiovascular disease: epidemiology, biology, and treatment. Arch Gen Psychiatry. 1998;55:580–92.
19. Otte C, Neylan TC, Pipkin SS, Browner WS, Whooley MA. Depressive symptoms and 24-hour urinary norepinephrine excretion levels in patients with coronary disease: findings from the Heart and Soul Study. Am J Psychiatry. 2005;162:2139–45.
20. Carney RM, Saunders RD, Freedland KE, Stein P, Rich MW, Jaffe AS. Association of depression with reduced heart rate variability in coronary artery disease. Am J Cardiol. 1995;76:562–4.
21. Frasure-Smith N, Lespérance F, Habra M, Talajic M, Khairy P, Dorian P, et al. Elevated depression symptoms predict long-term cardiovascular mortality in patients with atrial fibrillation and heart failure. Circulation. 2009;120:134–40.
22. Frasure-Smith N, Lespérance F, Irwin MR, Talajic M, Pollock BG. The relationships among heart rate variability, inflammatory markers and depression in coronary heart disease patients. Brain Behav Immun. 2009;23:1140–7.
23. Ayas NT, White DP, Manson JE, Stampfer MJ, Speizer FE, Malhotra A, et al. A prospective study of sleep duration and coronary heart disease in women. Arch Intern Med. 2003;163:205–9.
24. Leineweber C, Kecklund G, Janszky I, Akerstedt T, Orth-Gomér K. Poor sleep increases the prospective risk for recurrent events in middle-aged women with coronary disease. The Stockholm Female Coronary Risk Study. J Psychosom Res. 2003;54:121–7.
25. Schwartz SW, Cornoni-Huntley J, Cole SR, Hays JC, Blazer DG, Schocken DD. Are sleep complaints an independent risk factor for myocardial infarction? Ann Epidemiol. 1998;8:384–92.
26. Mezick EJ, Hall M, Matthews KA. Are sleep and depression independent or overlapping risk factors for cardiometabolic disease? Sleep Med Rev. 2011;15:51–63.
27. Mallon L, Broman JE, Hetta J. Sleep complaints predict coronary artery disease mortality in males: a 12-year follow-up study of a middle-aged Swedish population. J Intern Med. 2002;251:207–16.
28. Nicholson A, Fuhrer R, Marmot M. Psychological distress as a predictor of CHD events in men: the effect of persistence and components of risk. Psychosom Med. 2005;67:522–30.
29. Newman AB, Spiekerman CF, Enright P, Lefkowitz D, Manolio T, Reynolds CF, et al. Daytime sleepiness predicts mortality and cardiovascular disease in older adults. The Cardiovascular Health Study Research Group. J Am Geriatr Soc. 2000;48:115–23.
30. Carney RM, Freedland KE, Jaffe AS. Insomnia and depression prior to myocardial infarction. Psychosom Med. 1990;52:603–9.
31. Saleh DK, Nouhi S, Zandi H, Lankarani MM, Assari S, Pishgou B. The quality of sleep in coronary artery disease patients with and without anxiety and depressive symptoms. Indian Heart J. 2008;60:309–12.
32. Caska MC, Hendrickson BE, Wong MH, Ali S, Neylan T, Whooley MA. Anger expression and sleep quality in patients with coronary heart disease: findings from the heart and soul study. Psychosom Med. 2009;71:1–6.
33. World Health Organization. Tenth Revision of the International Classification of Diseases, Chapter (V): Mental and Behavioural Disorders (including disorders of psychological development). Clinical Descriptions and Diagnostic Guidelines. WHO; 1991.
34. Hiller W, Zaudig M, Mombour W. ICD International Diagnostic Checklists for ICD-10 and DSM-IV. Goettingen: Hogrefe & Huber Pub; 1999.
35. Beck AT, Steer RA. Beck Depression Inventory. New York (NY): Harcourt Brace Jovanovich; 1987.
36. Buysse DJ, Reynolds CF, Monk TH, Berman SR, Kupfer DJ. The Pittsburgh Sleep Quality Index: a new instrument for psychiatric practice and research. Psychiatry Res. 1989;28:193–213.
37. Backhaus J, Junghanns K, Broocks A, Riemann D, Hohagen F. Test-retest reliability of the Pittsburgh Sleep Quality Index in primary insomnia. J Psychosom Res. 2002;53:737–40.
38. Cohen J. Statistical power for the behavioural science. 2nd ed. Hillsdale, New York: Erlbaum; 1988.
39. Hedges LV, Olkin I. Statistical methods for meta-analysis. Orlando: Academic; 1985.
40. McCall WV, Reboussin BA, Cohen W. Subjective measurement of insomnia and quality of life in depressed inpatients. J Sleep Res. 2000;9:43–8.
41. Redeker NS, Muench U, Zucker MJ, Walsleben J, Gilbert M, Freudenberger R, et al. Sleep disordered breathing, daytime symptoms, and functional performance in stable heart failure. Sleep. 2010;33:551–60.
42. Skobel EC, Sinha AM, Norra C, Randerath W, Breithardt OA, Breuer C, et al. Effect of cardiac resynchronization therapy on sleep quality, quality of life, and symptomatic depression in patients with chronic heart failure and Cheyne-Stokes respiration. Sleep Breath. 2005;9:159–66.
43. Bower B, Bylsma LM, Morris BH, Rottenberg J. Poor reported sleep quality predicts low positive affect in daily life among healthy and mood-disordered persons. J Sleep Res. 2010;19:323–32.
44. Phillips B, Mannino DM. Do insomnia complaints cause hypertension or cardiovascular disease? J Clin Sleep Med. 2007;15:489–94.
45. Hayashino Y, Yamazaki S, Takegami M, Nakayama T, Sokejima S, Fukuhara S. Association between number of comorbid conditions, depression, and sleep quality using the Pittsburgh Sleep Quality Index: results from a population-based survey. Sleep Med. 2010;11:366–71.
46. Broström A, Strömberg A, Dahlström U, Fridlund B. Sleep difficulties, daytime sleepiness, and health-related quality of life in patients with chronic heart failure. J Cardiovasc Nurs. 2004;19:234–42.
47. Javaheri S, Parker TJ, Liming JD, Corbett WS, Nishiyama H, Wexler L, et al. Sleep apnea in 81 ambulatory male patients with stable heart failure. Types and their prevalences, consequences, and presentations. Circulation. 1998;97:2154–9.
48. Roux F, D'Ambrosio C, Mohsenin V. Sleep-related breathing disorders and cardiovascular disease. Am J Med. 2000;108:396–402.
49. Lanfranchi PA, Somers VK, Braghiroli A, Corra U, Eleuteri E, Giannuzzi P. Central sleep apnea in left ventricular dysfunction: prevalence and implications for arrhythmic risk. Circulation. 2003;107:727–32.
50. Skobel EC, Norra C, Sinha A, Schauerte P. Impact of sleep-related breathing disorders on health-related quality of life in patients with chronic heart failure. Eur J Heart Fail. 2005;7:505–11.
51. Carney RM, Howells WB, Freedland KE, Duntley SP, Stein PK, Rich MW, et al. Depression and obstructive sleep apnea in patients with coronary heart disease. Psychosom Med. 2006;68:443–8.

52. Ehrmann DE, Deldin PJ, Pitt B. Is sleep apnea a potential link between major depressive disorder and cardiovascular disease? Int J Cardiol. 2011;147:1–3.
53. Peppard PE, Szkoł-Coxe M, Hla KM, Young T. Longitudinal association of sleep-related breathing disorder and depression. Arch Intern Med. 2006;166:1709–15.
54. Habukawa M, Uchimura N, Kakuma T, Yamamoto K, Ogi K, Hiejima H, et al. Effect of CPAP treatment on residual depressive symptoms in patients with major depression and coexisting sleep apnea: contribution of daytime sleepiness to residual depressive symptoms. Sleep Med. 2010;11:552–7.
55. Whitney CW, Enright PL, Newman AB, Bonekat W, Foley D, Quan SF. Correlates of daytime sleepiness in 4578 elderly persons: the Cardiovascular Health Study. Sleep. 1998;21:27–36.
56. Kaneko Y, Floras JS, Usui K, Kaneko Y, Floras JS, Usui K, et al. Cardiovascular effects of continuous positive airway pressure in patients with heart failure and obstructive sleep apnea. N Engl J Med. 2003;348:1233–41.
57. Arzt M, Young T, Finn L, Skatrud JB, Ryan CM, Newton GE, et al. Sleepiness and sleep in patients with both systolic heart failure and obstructive sleep apnea. Arch Intern Med. 2006;166:1716–22.
58. Gottlieb SS, Khatta M, Friedmann E, Einbinder L, Katzen S, Baker B, et al. The influence of age, gender, and race on the prevalence of depression in heart failure patients. J Am Coll Cardiol. 2004;43:1542–9.
59. Dijk DJ, Duffy JF, Czeisler CA. Contribution of circadian physiology and sleep homeostasis to age-related changes in human sleep. Chronobiol Int. 2000;17:285–311.
60. Ferentinos P, Kontaxakis V, Havaki-Kontaxaki B, Paparrigopoulos T, Dikeos D, Ktonas P, et al. Sleep disturbances in relation to fatigue in major depression. J Psychosom Res. 2009;66:37–42.
61. Moffaert M. Sleep disorders and depression: the 'chicken and egg' situation. J Psychosom Res. 1994;38:9–13.
62. Froese CL, Butt A, Mulgrew A, Cheema R, Speirs MA, Gosnell C, et al. Depression and sleep-related symptoms in an adult, indigenous, North American population. J Clin Sleep Med. 2008;4:356–61.
63. Hemingway H, Marmot M. Evidence based cardiology: psychosocial factors in the aetiology and prognosis of coronary heart disease. Systematic review of prospective cohort studies. BMJ. 1999;318:1460–7.
64. Havranek EP, Spertus JA, Masoudi FA, Jones PG, Rumsfeld JS. Predictors of the onset of depressive symptoms in patients with heart failure. J Am Coll Cardiol. 2004;44:2333–8.
65. Thombs BD, de Jonge P, Coyne JC, Whooley MA, Frasure-Smith N, Mitchell AJ, et al. Depression screening and patient outcomes in cardiovascular care: a systematic review. JAMA. 2008;300:2161–71.
66. Davidson KW, Rieckmann N, Clemow L, Schwartz JE, Shimbo D, Medina V, et al. enhanced depression care for patients with acute coronary syndrome and persistent depressive symptoms: 1. Coronary Psychosocial Evaluation Studies Randomized Controlled Trial. Arch Intern Med. 2010;170:600–8.
67. Harvey AG, Murray G, Chandler RA, Soehner A. Sleep disturbance as transdiagnostic: consideration of neurobiological mechanisms. Clin Psychol Rev. 2011;31:225–35.