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Gender-specific changes in quality of life following cardiovascular disease: A prospective study

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Abstract

Gender-specific changes in Quality of Life (QoL) following cardiovascular disease (CVD) were studied in 208 patients to determine whether gender-related differences in postmorbid QoL result from differences in disease severity, premorbid QoL, or different CVD-related recovery. Premorbid data were available from a community-based survey. Follow-ups were done at 6 weeks, 6 months, and 12 months after diagnosis. Results showed that females had worse QoL at all three postmorbid assessments compared to males. However, multivariate analyses adjusting for premorbid gender differences and disease severity showed no significant gender-related differences for physical and psychologic functioning. Therefore, gender differences in QoL following CVD mainly result from premorbid differences in QoL, age, comorbidity, and disease severity at the time of diagnosis, and do not appear to be the consequence of gender-specific recovery. However, in clinical practice it is important to acknowledge the poorer QoL of females following CVD.

Keywords: Gender differences; Cardiovascular disease; Quality of life; Longitudinal

1. Introduction

Over the past decade, gender issues have received increased attention in social and medical sciences. Health surveys reveal that women have a higher prevalence of medical conditions and psychosocial problems, such as depression, compared to men [1–3]. The case of cardiovascular disease (CVD) is a paradoxical one, as CVD is traditionally regarded as a predominantly male disease [4], while recent studies report that women's CVD related morbidity and mortality after CVD exceed that of men [5–7].

The majority of studies on quality of life (QoL) in patients with CVD suggest that women do not cope as well physically and psychosocially as men, as concluded in a large review by Brezinka and Kittel [6]. Female patients score worse on psychosomatic symptoms, depression, anxiety, and sleep disturbances compared to their male counterparts [8]. However, the literature is not consistent, and it remains unclear why gender-related differences in QoL exist among CVD patients. There may be at least three theoretical explanations for a gender-related difference in functioning following CVD. These explanations are hypothetical and probably not mutually exclusive. First, gender-related differences might be a consequence of a more severe CVD pathology among women compared to men. These physiologic differences in disease etiology and disease severity between genders may exist, although they are at the present time not well understood [9]. A second explanation for women's poorer functioning after CVD might be that older women in particular generally have lower premorbid levels of functioning than older men. This may, for example, be due to higher levels of morbidity. Numerous studies indicate that in community samples the prevalence of medical and psychologic conditions is higher in women than in men [1–3]. Hence, differences in QoL after CVD between males and females may be ascribed to existing gender differences in QoL, health status, and social disadvantages before the emergence of CVD [10]. A third, related, explanation for women's poorer functioning after CVD might be that women have worse CVD-related recovery than men independently of premorbid levels of functioning.

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This refers more explicitly to the process of adaptation after CVD has emerged. Although gender differences in risk profiles for CVD have been reported [11–13], little is known about gender-specific adaptation after CVD. In general, smoking, alcohol consumption, and overweight are the most common risk factors for men; whereas psychologic distress, role pressure, and less strenuous exercise are more characteristic of women [14,15]. Therefore, health–behavioral adaptation processes might be prominent for males, whereas psychosocial adaptation processes might be more relevant among females. The effectiveness of these adaptation processes may differ, which might result in gender-related differences in QoL after CVD.

The objective of the present study is to verify empirically these three explanations. The Groningen Longitudinal Aging Study includes premorbid data, and is pre-eminently suitable for studying these explanations of gender-related differences in QoL of CVD patients. We examined gender-specific changes in physical, psychological, and social functioning (QoL) of elderly subjects in the year after diagnosis of CVD. Moreover, we determined whether gender-related differences in postmorbid QoL are a result of differences in disease severity at the time of diagnosis, differences in premorbid QoL, or premorbid social disadvantages, or gender differences in CVD-related recovery. In comparison to most other studies, our study was truly prospective including one *premorbid* and three *postmorbid* assessments. The premorbid measurement was part of a large population based survey in 1993. This design allows an adjustment of possible premorbid gender differences in QoL and other covariates, which were present prior to diagnosis.

2. Methods

This study is part of the Groningen Longitudinal Aging Study (GLAS). Detailed description of data collection is published elsewhere [16–20]. Briefly, in 1993 a total of 5,279 community dwelling elderly people (>57 years) were interviewed providing data on determinants of disease, functional disability, QoL, well-being, and utilization of care.

2.1. New CVD patients

Two cardiovascular events were included as indicators of CVD: acute myocardial infarction (AMI) and congestive heart failure (CHF). From the baseline wave in 1993 until January 1 1998, the 27 general practitioners participating in the Morbidity Registration Network Groningen passed on the names of all patients with a new postbaseline episode of AMI or diagnosis of CHF according to the criteria of the International Classification of Primary Care (ICPC) [21]. AMI was diagnosed if two of the following three symptoms were present: (a) chest pain characteristic of myocardial ischemia and lasting more than 15 min, (b) abnormal ST-T changes or Q waves on an electrocardiogram, or (c) elevation of blood cardiac enzymes (code K75 of the ICPC). CHF was diagnosed if three of the following five symptoms were present: (a) dependent oedema, (b) raised jugular venous pressure or hepatomegaly in the absence of liver disease, (c) signs of pulmonary congestion or pleural effusion, (d) enlarged heart, and (e) dyspnoea in the absence of pulmonary disease (code K77 of the ICPC).

During the enrolment period 207 patients with a new episode of AMI and 293 patients with a first diagnosis of CHF after baseline were recruited. Of these 500 persons with CVD 74 (15%) died before T1 and 49 persons were already participating in one of the other six GLAS cohort studies. Of the 377 potential responders, 68 patients refused participation in the study and 47 patients did not participate for other reasons (see for details: van Jaarsveld et al. [22]), leaving 262 persons for T1 (70%). Of these 262 patients who started the follow-up study, 229 (87%) participated in T2 and 208 (79%) also completed T3. These 208 patients (89 AMI and 119 CHF) are the subjects of this study. The 89 AMI patients include 30 females (34%) and 59 males (66%). The proportion of females is higher among CHF patients: 70 females (59%) and 49 males (41%). The patient's death explained 20 of the 54 cases of dropout. Nonresponse analyses showed that at baseline (T0), participants were significantly younger and had better physical functioning scores, on average, than nonparticipants. No significant differences were found on other socio-demographic or QoL variables at baseline.

2.2. Assessment points

All outcome variables were assessed on four occasions: at baseline (T0: premorbid), 6 weeks (T1), 6 months (T2), and 12 months (T3) after diagnosis. The premorbid assessment took place in 1993 for all participants, while the timing of T1, T2, and T3 depended on the time of diagnosis and occurred between 1993 and 1998. The mean length of the time interval between the baseline interview and the new episode was 26 months and ranged from 1 to 58 months. All the data were collected in face-to-face interviews by well-trained middle-aged women.

2.3. Outcome measures

QoL is conceptualized as relating to three domains of functioning: physical, psychologic, and social.

Physical functioning was assessed with the Groningen Activity Restriction Scale (GARS). GARS comprises 18 ADL (activities of daily living) and IADL (instrumental activities of daily living) items, each with four response categories. Scores may range from 18 (no physical dysfunctioning) to 72 (maximum level of physical dysfunctioning). Examples of GARS items are "Can you dress yourself without any help from others?," and "Can you walk up and down the stairs?" The results of previous studies showed that GARS meets the stochastic cumulative scalability criteria of the Mokken Model [23,24].

Depressive symptoms and feelings of anxiety, indicators of *psychologic functioning*, were assessed with the Hospital Anxiety and Depression Scale (HADS) [25,26]. The HADS depression subscale was originally developed to reveal pos-

sible depressive states in a medical outpatient clinic setting. Items referring to symptoms that may have a physical cause (e.g., insomnia and weight loss) are not included in the scale. Therefore, HADS is considered to have no bias towards depressive symptoms resulting from concurrent general medical conditions [26]. Examples of items are "I still enjoy the things I used to enjoy," "I feel cheerful," and "I get sudden feelings of panic." Both subscales consist of seven items, and the theoretical ranges vary from 0 to 21; higher scores indicate more symptoms. HADS has been validated for an older Dutch population [26].

Social functioning was assessed with two MOS SF-20 subscales: MOS-social functioning and MOS-role functioning [27]. The social functioning subscale measures the extent to which health interferes with normal social activities such as visiting friends, in a one-item question. The role functioning subscale measures the extent to which health interferes with usual daily activities such as housework or paid work. The scale consists of two items: "Does your health keep you from working at a job or doing work around the house?" and "Have you been unable to do certain kinds or amounts of work, housework because of your health?" Both the social and role functioning scales range from 0 to 100. Scores are reversed so that higher scores indicate poorer functioning, corresponding with scores on physical and psychologic functioning. The psychometric properties of the Dutch version of the MOS scales were approved in a pilot study [28].

2.4. Covariates

Disease severity was assessed according to the New York Heart Association (NYHA) classification at T1 (just after diagnosis). This NYHA classification indicates the severity of cardiac symptoms and may range from 1 (mild symptoms) to 4 (severe symptoms).

Premorbid social disadvantages include age, marital status (living with or without a partner), educational level (low, middle, high), and chronic morbidity before the development of CVD (number of chronic conditions), because these were found to be related with cardiac disease status [29]. These characteristics are also associated with gender in our study and may contribute to gender differences in QoL following CVD. The way educational level and comorbidity were measured is reported elsewhere [30].

2.5. Analyses

Differences in QoL between male and female patients were analyzed for significance using *t*-tests on each of the four assessment points. A *P*-value $<.05$ was considered statistically significant. In the introduction, three possible explanations for postmorbid gender differences in QoL were given. To test the first explanation (i.e., a more severe CVD pathology among women than men), gender differences in NYHA classification were studied. The second explanation (i.e., gender-related differences in QoL after CVD may be ascribed to existing differences in QoL and social disadvantages before the emergence of CVD), was analyzed with Multivariate Analyses of Variance (MANOVA) in which gender differences in QoL were adjusted for covariates and levels of premorbid (baseline) QoL. The third explanation (i.e., worse CVD-related recovery in females) was analyzed by studying changes over time with paired *t*-tests (i.e., mean change scores of QoL (T0–T1, T0–T2, and T0–T3) in males vs. females). Again, MANOVA was used to test gender differences in these change scores adjusted for covariates. Specific results on which of the covariates were significant are also presented, including information about interpreting the effects.

3. Results

3.1. Patient characteristics

Table 1 shows the baseline characteristics of the 108 male and 100 female patients. Females were on average older, less educated, more likely to live without a partner, had more chronic conditions at baseline, and reported more cardiac symptoms (i.e., NYHA class III/IV) at T1 (just after diagnosis) compared to males.

All these characteristics were related to QoL in our study, that is, higher age, living without partner, low educational level, and more comorbid conditions at baseline were related to worse QoL at T0, T1, T2, and T3; in addition cardiac symptoms at T1 were related to QoL at T1, T2, and T3 (data not shown). The baseline to CVD time interval was not significantly different between females and males.

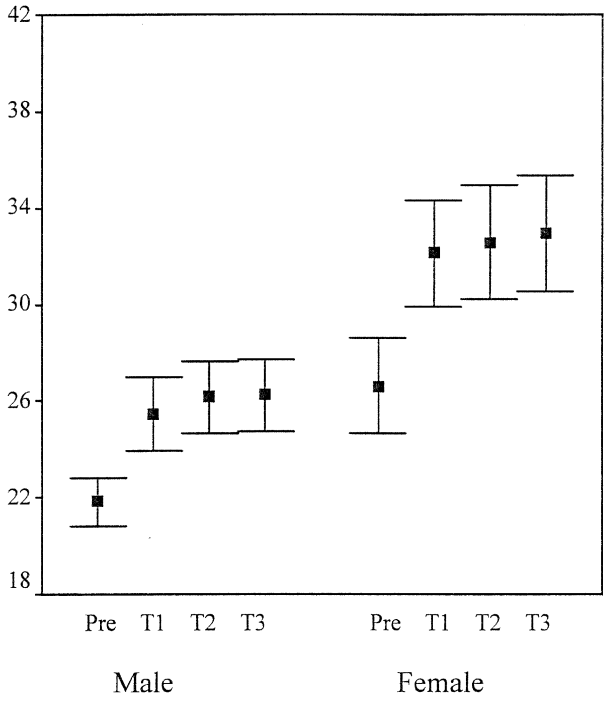
3.2. Gender differences in QoL before and after CVD

Gender differences in physical functioning are shown in Figure 1. Because graphs for the other domains of QoL are comparable, these are not presented. The bars indicate the 95% confidence intervals of the mean scores among males

Table 1
Baseline characteristics of 208 CVD patients

	Males	Females	*P*-value of gender difference
N (%)	108 (52%)	100 (48%)	
Age, mean (SD)	70 (7.6)	74 (7.5)	.001
Number of chronic conditions at baseline			
Mean (SD)	1.4 (1.3)	1.8 (1.5)	.015
Range	0–5	0–7	
Without partner (%)	16	60	<.001
Educational level (%)			
Low	18	50	
Middle	68	55	
High	14	5	<.001
NYHA class at T1 (%)			
I	41	23	
II	17	21	
III	30	49	
IV	12	7	.010
Interval T0–T1, mean (SD)[a]	24.9 (14.8)	28.1 (16.1)	.138

[a]Interval between baseline (T0) and diagnosis of CVD (T1) in months



* Pre = Pre-morbid
T1 = 6 weeks after diagnosis CVD
T2 = 6 months after diagnosis CVD
T3 = 12 months after diagnosis CVD

Higher scores indicate worse physical functioning.

Fig. 1. Change in physical functioning by gender in CVD patients.

Table 2
Gender differences in QoL before and after the development of CVD in older patients

	Male $N = 108$[a]	Female $N = 100$[a]	Unadjusted P-value	Adjusted P-value[b]
Physical functioning				
Premorbid	21.8 (5.1)	26.6 (9.6)	<.001	.126
6 weeks after CVD	25.5 (7.7)	32.1 (10.9)	<.001	.115
6 months after CVD	26.2 (7.5)	32.6 (11.6)	<.001	.432
12 months after CVD	26.2 (7.5)	32.9 (11.8)	<.001	.251
Feelings of anxiety				
Premorbid	3.0 (3.2)	4.7 (4.0)	.002	.045
6 weeks after CVD	4.4 (3.8)	5.3 (3.8)	.118	.273
6 months after CVD	4.1 (3.4)	5.5 (4.2)	.014	.057
12 months after CVD	4.5 (3.5)	5.8 (4.8)	.027	.101
Depressive symptoms				
Premorbid	4.7 (3.7)	5.6 (3.9)	.074	.767
6 weeks after CVD	4.6 (3.5)	5.5 (3.7)	.067	.970
6 months after CVD	5.2 (3.7)	6.4 (4.1)	.026	.462
12 months after CVD	5.1 (3.7)	6.5 (4.5)	.011	.612
Social functioning				
Premorbid	15.7 (22.3)	34.5 (30.5)	<.001	.017
6 weeks after CVD	30.0 (26.1)	48.0 (32.5)	<.001	.006
6 months after CVD	26.3 (24.7)	42.5 (28.2)	<.001	.042
12 months after CVD	26.9 (25.6)	45.5 (29.0)	<.001	.459
Role functioning				
Premorbid	27.0 (39.6)	49.5 (48.4)	<.001	.111
6 weeks after CVD	43.6 (44.3)	64.8 (43.2)	.001	.055
6 months after CVD	39.7 (45.8)	69.4 (39.1)	<.001	.002
12 months after CVD	41.7 (44.0)	72.5 (40.1)	<.001	.009

[a]Figures represent mean (SD).

[b]Scores are adjusted for age, marital status, educational level, and comorbidity index.

T1, T2, and T3 scores are also adjusted for NYHA classification and premorbid level of functioning.

and females and show large individual differences. Results on each domain of QoL are presented separately.

3.3. Physical functioning

In Table 2, mean scores on physical functioning are presented (high scores represent poorer functioning). It is obvious that females, who already had worse premorbid physical functioning (T0) than males, deteriorated even further after their CVD event. At all measurement periods, females reported significantly worse physical functioning than males ($P < .001$, see Table 2). The premorbid level of physical functioning in females (26.6) was as high as men's mean physical functioning at T3 (1 year postmorbid) (26.2). The P-values in the last column of Table 2 indicate whether the gender differences remained significant after adjusting for premorbid characteristics and NYHA class. Clearly, the gender differences on physical functioning were not significant after adjustment for these covariates. Premorbid physical functioning, age, and comorbidity were the significant premorbid covariates. In addition, NYHA class at time of diagnosis added information to explain gender differences at follow-up.

Table 3 shows change scores from baseline. Both males and females showed significant increases in physical dysfunctioning. On average, males deteriorated 4.4 points on the physical functioning scale, while females deteriorated 6.3 during 1-year follow-up. The crude comparison (unadjusted for covariates) of the mean change scores revealed that changes among females were significant larger than changes among males at T1 and T3 ($P < .05$). After adjustment for covariates, these gender differences were no longer significant. Age and NYHA class were the significant covariates. Thus, although substantial gender differences in postmorbid physical functioning were found, indicating poorer adaptation by females, change scores from baseline indicated modest gender differences in CVD-related recovery that were no longer significant after adjustment for age and NYHA class.

3.4. Psychologic functioning

Feelings of anxiety and depressive symptoms were high among females compared to males at all measurement periods (Table 2). Mean premorbid levels of anxiety and depressive symptoms among females (4.7 and 5.6, respectively) were again as high as the mean postmorbid levels among males (4.5 and 5.1, respectively, at T3). Crude gender differences were statistically significant for anxiety at T0, T2, and T3 ($P < .05$) and for depressive symptoms at T2 and T3 ($P < .05$). Gender differences on depressive symptoms at T0 and T1 were border-

Table 3
Gender differences in changes on QoL from baseline functioning

	Male $N = 108$[a]	Female $N = 100$[a]	Unadjusted P-value	Adjusted P-value[b]
Physical functioning				
T0-T1: onset effect	+3.7 (5.5)***	+5.5 (7.3)***	.048	.300
T0–T2: 6-month interval	+4.4 (5.5)***	+6.0 (7.6)***	.096	.737
T0–T3: 12-month interval	+4.4 (5.7)***	+6.3 (7.5)***	.048	.428
Feelings of anxiety				
T0–T1: onset effect	+1.4 (3.8)***	+0.6 (4.2)	.159	.747
T0–T2: 6-month interval	+1.1 (3.5)***	+0.8 (4.1)	.574	.629
T0–T3: 12-month interval	+1.5 (3.7)***	+1.1 (4.2)**	.586	.528
Depressive symptoms				
T0–T1: onset effect	−0.1 (3.6)	−0.1 (3.5)	.958	.559
T0–T2: 6-month interval	+0.5 (4.0)	+0.8 (3.8)*	.629	.266
T0–T3: 12-month interval	+0.4 (4.0)	+0.9 (4.1)*	.368	.254
Social functioning				
T0–T1: onset effect	+14.3 (31.3)***	+13.5 (37.0)***	.862	.580
T0–T2: 6-month interval	+10.6 (28.9)***	+8.0 (35.3)*	.564	.927
T0–T3: 12-month interval	+11.2 (27.7)***	+11.0 (29.7)***	.969	.249
Role functioning				
T0–T1: onset effect	+16.6 (45.8)***	+15.3 (51.0)**	.843	.677
T0–T2: 6-month interval	+12.7 (50.1)*	+19.9 (47.5)***	.302	.129
T0–T3: 12-month interval	+14.7 (50.0)**	+23.0 (45.5)***	.224	.333

[a]Figures represent mean change scores from baseline and (SD). Positive changes indicate a worsening in functioning; negative changes indicate an improvement.

[b]Change scores are adjusted for age, marital status, educational level, comorbidity index, NYHA classification but not for baseline levels.

Paired t-Test on changes within respectively males or females: *P<.05, **P<.01, ***P<.001.

line significant ($P < .10$). T0 differences in anxiety remained significant, even after adjustment for age, marital status, educational level, and comorbidity, while postmorbid gender differences on anxiety and depressive symptoms were no longer significant after adjustment for all covariates. Again, premorbid functioning, age, and comorbidity appeared to be the significant premorbid covariates. In addition NYHA class at T1 was significant in explaining gender differences at follow-up.

Mean changes over time are again presented in Table 3. Different patterns in change over time were identified for anxiety and depressive symptoms for males and females. In males, an immediate increase in feelings of anxiety was identified at T1 ($P < .001$). Among females, a gradual increase in feelings of anxiety was found, which did not attain statistical significance until T3 (see P-values in Table 3 for significant changes within males and females, respectively). Among males, no significant change in depressive symptoms over time was observed, while in females a significant increase in depressive symptoms was found at T2 (6 months after diagnosis, $P < .05$). Although mean changes in anxiety over time were slightly larger for males and mean changes in depressive symptoms were slightly more pronounced among females, both the unadjusted and adjusted gender differences reached no statistical significance. In other words, men and women do not differ significantly on CVD-related recovery in terms of changes in psychologic functioning.

3.5. Social and role functioning

The lower part of Table 2 shows significant gender differences in social and role functioning at both the premorbid and postmorbid assessment periods. Females scored worse on social and role functioning than males. Again, mean premorbid functioning among females was comparable to postmorbid functioning among males. After adjustment for covariates, gender differences remained significant for social functioning at T0, T1, and T2 and for role functioning at T2 and T3 (differences at T1 remained borderline significant with $P < .10$). Premorbid functioning, age, comorbidity, and NYHA class were the significant covariates for both social and role functioning. In addition, marital status was a significant covariate for social functioning and educational level for role functioning.

The change scores on social and role functioning of Table 3 indicated significant changes in functioning among both males and females, without significant differences between males and females. In other words, although premorbid functioning of women is worse, both males and females deteriorated equally with regard to absolute change scores on the scales.

3.6. Covariates

The multivariate analyses included several covariates. Overall, age, comorbidity, NYHA class, and premorbid QoL were significant covariates. Higher age, more comorbid conditions, and higher NYHA class were independently related to worse QoL at T0, T1, T2, and T3. Higher scores on premorbid QoL (indicating worse functioning) were related to worse QoL at T1, T2, and T3. Although marital status and educational level were significantly related to gender and QoL, these were nonsignificant covariates in the multivariate analyses for physical and psychologic functioning. Marital status (i.e., living without a partner) was independently related to worse social functioning and low educational level to worse role functioning at follow-ups. With re-

gard to change scores: higher age and higher NYHA class were related to greater deterioration in physical functioning and larger increase in feelings of anxiety.

4. Discussion

The present study underlines the large impact of CVD on the three domains of QoL (physical, psychologic, and social functioning) for both males and females. One year after diagnosis, QoL is significantly lower than premorbid levels of QoL. Significant gender-related differences were found for all QoL measures, indicating worse QoL among females at all assessment periods. These gender-related differences are substantial, because mean premorbid QoL in females is comparable to mean postmorbid QoL in males on all measures of QoL. The observed gender-related difference of 6.7 units on physical functioning at 1-year follow-up (see Table 2) indicates that, on average, females experience slightly more problems in 6 of the 18 selected physical activities, or a lot more problems in at least three activities compared to males. These observations support the clinical relevance of the gender-related difference in QoL.

In the Introduction, three possible explanations for the lower QoL of females following CVD were presented. Consistent with other studies, we found that females experience more severe symptoms of CVD (i.e., higher NYHA class). In our analyses, we adjusted for NYHA class to study whether postmorbid gender differences in QoL are merely a result of a more severe disease in females or whether differences in QoL are a result of other processes (i.e., explanation 1). Our results showed that higher NYHA class was independently related to worse postmorbid QoL. Thus, the worse postmorbid QoL of females compared to males may indeed be due in part to gender differences in disease severity. We also examined to what extent the poorer baseline characteristics of females (including premorbid levels of QoL, advanced age, and concomitant disease) account for their worse QoL following CVD (i.e., explanation 2). The results show that, after adjusting for these covariates, the postmorbid differences in QoL between males and females largely disappear. Obviously, a large extent of the postmorbid gender differences in QoL is explained by differences in premorbid characteristics. Premorbid level of QoL, age, and comorbidity appeared to be the significant covariates. Higher age and more comorbid conditions were related to worse levels of premorbid QoL. Even after adjusting for premorbid levels of QoL, age, and comorbidity were independently related to postmorbid levels of QoL. The third explanation for worse QoL of females following CVD suggested that women might have a poorer CVD-related recovery. The results using change scores did not back this.

In terms of absolute changes over time, the impact of CVD on both genders seems similar. However, similar absolute changes have to be interpreted carefully. Equal absolute change scores in males and females may not stand for similar changes in experienced disability in everyday life. The mean physical deterioration of females from 27 to 33 on the GARS questionnaire may be of a different order than the observed change in males from 22 to 26. Because premorbid functioning was already worse among females, a further six-point deterioration might have a disproportionately larger impact on females. The effects on psychologic functioning also indicate a somewhat different psychologic impact of CVD on males and females. Clearly, at 6 weeks after diagnosis (T1) levels of anxiety were increased in males, whereas no effect was found in females. In addition, the results indicated that CVD leads to an increase in depressive symptoms, particularly among females. For females, CVD might come even more as a surprise, because CVD is still seen as a predominantly male disease [4]. Therefore, dealing with the consequences of CVD may not at all fit in women's expectations of life, which may result in the specific increase in depressive symptoms and the delayed increase in feelings of anxiety in females.

It might be argued whether the worse scores on QoL among females indicate a true difference in QoL or is a result of report bias. If females generally tend to overreport problems in functioning and males tend to trivialize dysfunctioning, these might lead to biased results. Gender differences in the sensitivity of self-report depression scales are reported in the literature, suggesting separate cutoff scores for males and females [31]. However, a recent study does not support the opinion that females overreport morbidity. In fact, males reported a higher proportion of their conditions in response to a global question, and no evidence was found that females were more likely to report trivial or mental health conditions in response to the global question [32]. Results from a study comparing self-reported disability and performance measures also suggest that both males and females report their disability accurately, and female's higher prevalence of reported functional problems is a reflection of true disability [33]. Thus, it is not likely that the worse scores on each QoL measure among females are caused by a report bias but rather reflect true gender differences in functioning.

This study has several limitations. First, in this study two frequently occurring CVD (congestive heart failure and acute myocardial infarction) are studied together. However, the two cardiovascular events may have very different consequences. Disease-specific analyses of our data (with less power) showed however roughly the same gender differences, but are not presented here due to small sample numbers. Second, analyses pertained to only 208 patients out of the 500 originally diagnosed patients, which might have led to at least some selection bias. However, nonresponse is inevitable in geriatric research, especially when studying CVD. In contrast to most clinical trials, our source population included all incident cases of CVD in a cohort of elderly people. Mortality contributed 25% of the nonresponse ($n = 74$). Nonresponse analyses revealed only minor differences between participants ($n = 208$) and nonparticipants ($n = 292$) in baseline characteristics. Third, the time interval between the

premorbid assessment and CVD diagnosis varied from immediately after the baseline to 58 months (mean: 26.4 months; SD: 15.4). QoL may have changed during the interval. However, the length of the interval was similar for males and females. In addition, the outcomes of our analyses hardly changed when the baseline to CVD time interval was included as covariate in the multivariate models. We, therefore, assume that variation in this interval did not substantially affect the outcomes of the study.

One of the major advantages of the present study is the inclusion of a premorbid measurement. These premorbid data were collected as part of a large population-based survey among elderly in 1993, in which the present study sample participated. Comparison of the premorbid data revealed important issues. When examining only postmorbid assessments, one might conclude that females suffer from harmful consequences of CVD to a higher degree than males. The unadjusted results indeed indicate large gender differences in QoL. However, after adjustment for age, marital status, educational level, NYHA class, and baseline functioning, most gender differences were no longer significant. This suggests that it is not female gender in itself that makes patients more susceptible to the harmful consequences of CVD, but that the less advantageous premorbid characteristics make females more susceptible. On the other hand, although adjusted differences were no longer statistically significant, all gender differences indicate worse adaptation in females. The adaptation to CVD is a complicated process and large individual differences were observed. Also, within male and female patients large individual differences were observed in (changes in) QoL after CVD. The identification of parameters, besides gender, that relate to these individual differences in QoL following CVD is an important issue for further research.

In conclusion, the lower levels of QoL of females are statistically explained by existing differences between males and females. Nevertheless, it is important to acknowledge the worse QoL of females following CVD. In clinical practice, females will have worse QoL, and even though these lower levels are statistically explainable, females represent a vulnerable group with more physical limitations, more distress, and more social limitations and they may need specific attention.

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References

[1] Waldron I. Sex differences in human mortality: the role of genetic factors. Soc Sci Med 1983;17:321–33.
[2] Penning MJ, Strain LA. Gender differences in disability, assistance, and subjective well-being in later life. J Gerontol Ser B-Psychol Sci 1994;49:S202–8.
[3] Beekman ATF, Copeland JR, Prince MJ. Review of community prevalence of depression in later life. Br J Psychiatry 1999;174:307–11.
[4] Sytkowski PA, Dagostino RB, Belanger A, Kannel WB. Sex and time trends in cardiovascular disease incidence and mortality: the Framingham Heart Study, 1950–1989. Am J Epidemiol 1996;143:338–50.
[5] Con AH, Linden W, Thompson JM, Ignaszewski A. The psychology of men and women recovering from coronary artery bypass surgery. J Cardiopulm Rehabil 1999;19:152–61.
[6] Brezinka V, Kittel F. Psychosocial factors of coronary heart disease in women: a review. Soc Sci Med 1996;42:1351–65.
[7] Greenland P, Reicher-Reiss H, Goldbourt U, Behar S. In-hospital and 1-year mortality in 1,524 women after myocardial infarction. Comparison with 4,315 men. Circulation 1991;83:484–91.
[8] Uuskula M. Psychological differences between young male and female survivors of myocardial infarction. Psychother Psychosom 1996; 65:327–30.
[9] Castelli WP. Cardiovascular disease in women. Am J Obstet Gynecol 1988;158:1553–60.
[10] Young RF, Kahana E. Gender, recovery from late life heart attack, and medical care. Women Health 1993;20:11–31.
[11] Eriksson H, Wilhelmsen L, Caidahl K, Svardsudd K. Epidemiology and prognosis of heart failure. Z Kardiol 1991;80:1–6.
[12] Levy D, Larson MG, Vasan RS, Kannel WB, Ho KK. The progression from hypertension to congestive heart failure. JAMA 1996;275:1557–62.
[13] Aronow WS, Ahn C. Incidence of heart failure in 2,737 older persons with and without diabetes mellitus. Chest 1999;115:867–8.
[14] Verbrugge LM. The twain meet: empirical explanations of sex differences in health and mortality. J Health Soc Behav 1989;30:282–304.
[15] Greimel ER, Padilla GV, Grant MM. Gender differences in outcomes among patients with cancer. Psychooncology 1998;7:197–206.
[16] Kempen GIJM, Jelicic M, Ormel J. Personality, chronic medical morbidity, and health-related quality of life among older persons. Health Psychol 1997;16:539–46.
[17] Kempen GIJM, Ormel J, Brilman EI, Relyveld J. Adaptive responses among Dutch elderly: the impact of eight chronic medical conditions on health-related quality of life. Am J Public Health 1997;87:38–44.
[18] Kempen GIJM, Miedema I, van de Bos GAM, Ormel J. Relationship of domain-specific measures of health to perceived overall health among older subjects. J Clin Epidemiol 1998;51:11–8.
[19] Ormel J, Kempen GIJM, Penninx BWJH, Brilman EI, Beekman ATF, van Sonderen E. Chronic medical conditions and mental health in old people: disability and psychosocial resources mediate specific mental health effects. Psychol Med 1997;27:1065–77.
[20] Ormel J, Kempen GIJM, Deeg DJH, Brilman EI, van Sonderen E, Relyveld J. Functioning, well-being, and health perception in late middle-aged and older people: comparing the effects of depressive symptoms and chronic medical conditions. J Am Geriatr Soc 1998;46:39–48.
[21] Lamberts H, Wood M. International classification of primary care. Oxford: Oxford University Press; 1987.
[22] van Jaarsveld CHM, Sanderman R, Miedema I, Ranchor AV, Kempen GIJM. Changes in health-related quality of life in older patients with acute myocardial infarction or congestive heart failure: a prospective study. J Am Geriatr Soc 2001;49:1052–8.

[23] Kempen GIJM, Miedema I, Ormel J, Molenaar W. The assessment of disability with the Groningen Activity Restriction Scale—conceptual framework and psychometric properties. Soc Sci Med 1996;43: 1601–10.

[24] Kempen GIJM, Suurmeijer TPBM. The development of a hierarchical polychotomous ADL-IADL Scale for noninstitutionalized elders. Gerontologist 1990;30:497–502.

[25] Zigmond AS, Snaith RP. The hospital anxiety and depression scale. Acta Psychiatr Scand 1983;67:361–70.

[26] Spinhoven P, Ormel J, Sloekers PPA, Kempen GIJM, Speckens AEM, van Hemert AEM. A validation study of the Hospital and Depression Scale (HADS) in different groups of Dutch subjects. Psychol Med 1997;27:363–70.

[27] Stewart AL, Hays RD, Ware JE. The MOS short-form general health survey: reliability and validity in a patient population. Med Care 1988; 26:724–35.

[28] Kempen GIJM. Het meten van de gezondheidstoestand van ouderen; een toepassing van een Nederlandse versie van de MOS-schaal. Tijdschr Gerontol Geriatr 1992;23:132–40.

[29] Penninx BWJH, Beekman ATF, Honig A, et al. Depression and cardiac mortality—results from a community-based longitudinal study. Arch Gen Psychiatry 2001;58:221–7.

[30] Kempen GI, Brilman EI, Ranchor AV, Ormel J. Morbidity and quality of life and the moderating effects of level of education in the elderly. Soc Sci Med 1999;49:143–9.

[31] Allen-Burge R, Storandt M, Kinscherf DA, Rubin EH. Sex differences in the sensitivity of two self-report depression scales in older depressed inpatients. Psychol Aging 1994;9:443–5.

[32] Macintyre S, Ford G, Hunt K. Do women "over-report" morbidity? Men's and women's responses to structured prompting on a standard question on long standing illness. Soc Sci Med 1999;48:89–98.

[33] Merrill SS, Seeman TE, Kasl SV, Berkman LF. Gender differences in the comparison of self-reported disability and performance measures. J Gerontol Ser A-Biol Sci Med 1997;52A:M19–26.