

# Position paper on the importance of psychosocial factors in cardiology: Update 2013

## Positionspapier zur Bedeutung psychosozialer Faktoren in der Kardiologie: Update 2013

### Abstract

**Background:** The rapid progress of psychosomatic research in cardiology and also the increasing impact of psychosocial issues in the clinical daily routine have prompted the Clinical Commission of the German Heart Society (DGK) to agree to an update of the first state of the art paper on this issue which was originally released in 2008.

**Methods:** The circle of experts was increased, general aspects were implemented and the state of the art was updated. Particular emphasis was dedicated to coronary heart diseases (CHD), heart rhythm diseases and heart failure because to date the evidence-based clinical knowledge is most advanced in these particular areas. Differences between men and women and over the life span were considered in the recommendations as were influences of cognitive capability and the interactive and synergistic impact of classical somatic risk factors on the affective comorbidity in heart disease patients.

**Results:** A IA recommendation (recommendation grade I and evidence grade A) was given for the need to consider psychosocial risk factors in the estimation of coronary risks as etiological and prognostic risk factors. Furthermore, for the recommendation to routinely integrate psychosocial patient management into the care of heart surgery patients because in these patients, comorbid affective disorders (e.g. depression, anxiety and post-traumatic stress disorder) are highly prevalent and often have a malignant prognosis. A IB recommendation was given for the treatment of psychosocial risk factors aiming to prevent the onset of CHD, particularly if the psychosocial risk factor is harmful in itself (e.g. depression) or constrains the treatment of the somatic risk factors. Patients with acute and chronic CHD should be offered anti-depressive medication if these patients suffer from medium to severe states of depression and in this case medication with selective reuptake inhibitors should be given. In the long-term course of treatment with implanted cardioverter defibrillators (ICDs) a subjective health technology assessment is warranted. In particular, the likelihood of affective comorbidities and the onset of psychological crises should be carefully considered.

**Conclusions:** The present state of the art paper presents an update of current empirical evidence in psychocardiology. The paper provides evidence-based recommendations for the integration of psychosocial factors into cardiological practice and highlights areas of high priority. The evidence for estimating the efficiency for psychotherapeutic and psychopharmacological interventions has increased substantially since the first release of the policy document but is, however, still weak. There remains an urgent need to establish curricula for physician competence in psychodiagnosis, communication and referral to ensure that current psychocardiac knowledge is translated into the daily routine.

**Keywords:** depression, anxiety, post-traumatic stress disorder, psychotherapy, psychopharmacology

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## Zusammenfassung

**Hintergrund:** Die rasche Weiterentwicklung der psychokardiologischen Forschung, aber auch die wachsende Verankerung psychosozialer Fragestellungen im klinischen Alltag haben die Klinische Kommission der DGK bewogen, einer Aktualisierung und Weiterentwicklung des 2008 erstmals publizierten Positionspapiers zur Bedeutung psychosozialer Faktoren in der Kardiologie zuzustimmen.

**Methoden:** Der Kreis der Autoren wurde vergrößert, allgemeine Aspekte eingefügt und das Wissen in allen Abschnitten auf den heutigen Stand gebracht. Schwerpunkte der Empfehlungen sind die koronare Herzerkrankung, Herzrhythmusstörungen und die Herzinsuffizienz, da hier der Stand der empirischen Evidenz und des klinischen Wissens zu psychosozialen Fragestellungen am weitesten entwickelt ist. Berücksichtigt wurden bei den Empfehlungen Besonderheiten von Frauen und Männern, Unterschiede bzgl. der Lebensspanne, Einflüsse auf die kognitive Leistungsfähigkeit und die interaktive synergistische Bedeutung klassischer Risikofaktoren bei affektiver Komorbidität.

**Ergebnisse:** Eine I-A-Empfehlung (Empfehlungsgrad I, Evidenzgrad A) wurde vergeben für die Aufforderung, psychosoziale Risikofaktoren bei der Einschätzung des KHK-Risikos zu berücksichtigen, die als unabhängige ätiologische und prognostische Risikofaktoren für das Auftreten der koronaren Herzerkrankung (KHK) und für Komplikationen im Behandlungsverlauf der KHK bedeutsam sind. Ferner für die Empfehlung, Patienten mit Herzoperationen von einem interdisziplinären Team zu betreuen, in dem die Möglichkeit besteht, auf psychosoziale Aspekte einzugehen, da bei diesen Patienten komorbide psychische Störungen wie Depressivität, Angst und posttraumatische Belastungsstörung häufig und prognostisch ungünstig sind. Eine I-B-Empfehlung wurde vergeben für die Behandlung psychosozialer Risikofaktoren mit dem Ziel einer Primärprävention der KHK, wenn das Risikomerkmal an sich Krankheitswert hat (z. B. Depression) oder die Behandlung klassischer Risikofaktoren erschwert ist. Eine antidepressive Pharmakotherapie soll Patienten nach akutem Koronarsyndrom sowie in der Phase der chronischen KHK angeboten werden, die an einer mindestens mittelschweren rezidivierenden depressiven Störung leiden. Dabei sollen vorzugsweise Substanzen aus der Gruppe der selektiven Serotoninwiederaufnahmehemmer (SSRI) zum Einsatz kommen. Bei der langfristigen ärztlichen Begleitung von ICD-Patienten sollen die psychosozialen Folgen der ICD-Technologie beachtet und insbesondere relevante Affektstörungen sowie Krisen bei ICD-Patienten erkannt und fachgerecht behandelt werden.

**Schlussfolgerungen:** Das Positionspapier formuliert konkrete Anwendungsfelder mit hoher Priorität für die Einbeziehung psychosozialer Faktoren in die kardiologische Praxis, die eine leitlinienkonforme Evidenz aufweisen. Trotz deutlicher Fortschritte seit der Erstveröffentlichung des Positionspapiers existieren weiterhin Forschungsdefizite für die Bewertung der Wirksamkeit psychotherapeutischer und psychopharmakologischer Konzepte bei kardialen Patienten. Curricula für die Vermittlung von (psycho-)diagnostischer, kommunikativer und differenzialdiagnostischer Kompetenz müssen rasch entwickelt werden, um eine Transmission des aktuellen Wissensstands in die Alltagspraxis zu ermöglichen.

**Schlüsselwörter:** Depression, Angst, posttraumatische Belastungsstörung, Psychotherapie, Psychopharmakologie

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## 1 Preamble

This updated position paper of the “Deutsche Gesellschaft für Kardiologie – Herz- und Kreislaufforschung” (DGK) – on psychosocial aspects of cardiology gives an overview of the current knowledge base and also should ease the decision-making process for physicians, psychotherapists and their patients. Recent studies have been summarised, questions have been resolved, and the unresolved questions portrayed. Recommendations have been given with regard to therapeutic and diagnostic issues. The evidence base for these recommendations is also outlined. The position paper does not replace the doctors’ evaluations of individual patients and the resulting process of diagnosis and therapy.

### Class of recommendation

- Class I: conditions for which there is evidence and/or general agreement that a given procedure or treatment is useful and effective
- Class II: conditions for which there is conflicting evidence and/or a divergence of opinion about the usefulness/efficacy of a procedure or treatment
- Class IIa: weight of evidence/opinion is in favour of usefulness/efficacy
- Class IIb: usefulness/efficacy is less well established by evidence/opinion
- Class III: conditions for which there is evidence and/or general agreement that the procedure/treatment is not useful/effective and in some cases may be harmful.

### Evidence classification

- Level of evidence A: recommendation based on evidence from multiple randomized trials or meta-analyses
- Level of evidence B: recommendation based on evidence from a single randomized trial or nonrandomized studies
- Level of evidence C: recommendation based on expert opinion, case studies, or standards of care.

When assessing evidence regarding diagnostic procedures, large, population-based prospective studies were included.

## 2 Introduction

Psychosocial factors such as depression, anxiety or low social status are associated with increased risk for cardiovascular disease and worsened prognosis after disease onset. As a result of the current evidence base and socio-political relevance, these factors have not only been attracting increasing attention from healthcare providers (cardiologists and GPs) and health payers, but also have been incorporated into the guidelines of large medical associations.

The intention of the present position paper is not to summarise the current state of knowledge on every topic area, as is the case in a meta-analysis or a literature review. Rather, the aim is to formulate relevant conclusions and judgements as an expert panel and to outline the relevance of the relationship between psychosocial

factors and important cardiovascular diseases on the basis of current evidence.

The structure does not equate to the comprehensive scheme of a cardiology textbook. Instead, this paper concentrates on psychosocial hypotheses, which are relevant to cardiology – areas in which there is a critical mass of implementable knowledge. In doing so, the relevance to cardiology remains in focus. If aspects of psychocardiology (such as the role of behavioural therapy with regard to lifestyle changes) have been described in existing position papers or guidelines of the DGK, then the relevant sources have been cited.

### 3 Methods

The present position paper is an update and advancement of the “Position Paper on the Relevance of Psychosocial Factors in Cardiology”, published in 2008. The clinical commission of the DGK agreed to a revision in 2011, since the field has rapidly developed in the past years and the acceptance of psychosocial factors in the daily routine of healthcare providers for cardiac patients has grown remarkably. The circle of authors has expanded, general aspects have been incorporated and the knowledge in all areas has been brought up to date. The experts involved have come to the agreement that at this time there is especially much evidence and clinical knowledge with regard to coronary heart disease, cardiac arrhythmias and heart failure.

## 4 General aspects

### 4.1 Gender effects

Cardiovascular diseases are the most common causes of mortality in both men and women, although higher rates have been observed in women, in whom they manifest at an older age. The incidence rises only moderately after menopause. In older age groups (from 75 years of age) the increase is exponential. This pronounced age effect has consequences for the state of empirical research on the sex specific prognostic value of psychosocial risk factors. Most large, prospective population-based studies involve participants of middle or early old age (typically persons aged 35–75 years). Women in these age groups have lower rates of fatal outcomes, meaning that the number of cases is often too low for valid effect measurement. As a result, the importance of psychosocial risk factors in women is underestimated. A clear exception to this is the Stockholm Women’s Heart Study [1], which has made important contributions to knowledge of psychosocial risk factor constellations in women. In this study, strong evidence was portrayed, highlighting the role of problems related to family and partnerships as well as the multiplicative burden of holding a job and caring for the family in cardiovascular risk (see also [2]).

Evidence suggests that there are sex specific differences in the perception and reporting of complaints. The introceptive perception of complaints, including but not limited to chest pains is more pronounced in women, and in all age groups they are more willing to report complaints than men [3]. On the one hand, complaints may often be a result of mental health related disorders, on the other there is concern that ischaemic heart complaints will be misinterpreted as having functional or psychological causes in daily healthcare practice. Even when the origin of chest pain is somatic, women tend to report increased severity of psychological and physical symptoms on average [4]. In recent years, the importance of stress-induced cardiomyopathy has received recognition. This condition has mostly been observed in women and it often occurs under extremely stressful circumstances [5].

When treating psychosocial aspects of cardiovascular disease conditions, women benefit from different interventions than men. Most recently, the beneficial effect of psychotherapy on the occurrence of cardiac events has been proven for women with CHD [6], [7]. On the other hand, women appear to hardly profit at all from psychotherapy groups involving male and female patients (see [8]).

- **There is concern that ischaemic heart complaints will be misinterpreted as having functional causes. When treating psychosocial aspects of cardiovascular disease conditions, women profit from different interventions than men.**

### 4.2 Partnership and social support

Social support is a protective factor, which is not only important with regard to prevention, but also to the progress of coronary heart disease. Having a healthy relationship plays an important role here. On the other hand a lack of social support is a risk factor for adverse outcomes: in a meta-analysis, Barth et al. [9] showed that this factor has a significant and substantial influence on all-cause mortality (Hazard Ratio 1.59; 95% confidence interval 1.21–2.08) even after controlling for somatic risk factors. The breaking of a long-term relationship due to the death of a partner is also a risk factor, and more so for men than women. This was first shown in the classic “broken heart” study by Parkes et al. [10], and the results were successfully replicated in several studies. An elevated mortality risk has been described not only within the first months after the event, but already within the first days [11].

Occasionally it is recommended that partners be integrated into heart patients’ interventions in order to provide emotional support and to aid in coping with the disease (by helping with encouraging healthy lifestyle behaviours, adherence to treatment and control visits). The aim here is to maximise and strengthen resource usage, whilst on the other hand avoiding dysfunctional relationships. However, evidence to prove the effective-

ness of such partner and family orientated interventions is sparse.

Long-term relationships can, however, promote the continuation of unhealthy lifestyle habits (such as smoking and consumption of alcohol) [12]. It is also important to note that the partners of heart patients are often made to feel insecure by the disease and thus feel helpless. As a result, as has been shown with regard to ICD patients, the partner often reports higher levels of depression and anxiety than the patient [13]. Naturally, this can lead to both persons being negatively influenced by communication tainted by avoidance and feelings of anxiety [14]. "Care-giving" for a family member who is suffering from cancer has recently been identified as a cardiovascular risk factor [15].

### 4.3 Age specific aspects

Heart diseases affect people from different age groups differently. As a result of substantially improved healthcare, a growing number of children and adolescents born with heart conditions reach adulthood. With regard to this patient group, particular psychosocial aspects must be considered – in particular the high prevalence of anxiety and depressive disorders [16] or developmental problems after complex surgery undertaken during childhood [17]. Specialised centres for adolescents are helpful in ensuring a healthy transition into adulthood by supervising the increasing independence of the patient from the family. The guidelines of the American College of Cardiology recommend that this process should begin at 12 years of age [18].

In midlife adulthood, psychosocial interventions to improve behaviour related to cardiovascular risk and reintegration into the labour market are important. In older age groups the importance of coping with reduced physical performance, stamina and cognitive resources step into the foreground. At the same time, chronic heart diseases with comparable prognoses to progressed tumours are increasingly prevalent in this group and require palliative care.

- **In patients with progressive heart disease conditions, palliative care is often appropriate and should be considered.**

Evidence from cross-sectional and cohort studies suggest that early psychosocial stressors are effective across large periods of life, and that they have a significant effect on heart disease prognosis. Stressors and lack of positive factors *in utero* and in early childhood may determine the risk of cardiac events which occur decades later [19]. Stressful life experiences in childhood, adolescence and early adulthood have been associated with increased CHD incidence in the late decades of life [20].

### 4.4 Cognitive disorders and dementia

Cognitive disorders occur in patients with cardiac diseases in approximately 25–50% of cases [21], [22], [23]. In

particular, patients with severe heart insufficiency and those who have experienced open heart surgery often present with notably slowed mental processing and memory deficits [24]. The worsening of higher cognitive functions relevant to daily life and performance in memory and concentration do not initially become apparent through direct communication with the patient because the patient can often compensate the deficits in the context of conversation. Problems with communication and understanding as well as problems adhering to treatment should give impetus to assess the cognitive status of patients. Patients with cardiac diseases, especially those with heart failure, suffer from an almost two-fold increased risk of dementia [25].

- **Problems with communication and understanding as well as problems adhering to treatment highlight the necessity of assessing the cognitive status of patients.**

It is being discussed whether these cognitive impairments may have cardiac, cerebrovascular, or other somatic and psychosocial causes. All in all, one may assume a multifactorial ethiopathogenesis. This may result in impaired quality of life, increased rates of sick leave, and even an inability to work or find employment. This, in turn, may lead to massive economic costs. Additionally, cognitive dysfunction is an independent predictor of mortality [26]. Improvements in cognitive abilities after heart transplantation and cardiac resynchronisation therapy could be achieved using biventricular pacemakers and sport-based interventions.

### 4.5 Communication, adherence and health behaviour

#### Communication

Patient-centred communication has positive effects on patient satisfaction, adherence, utilisation of healthcare and in some cases also the success of treatment [27]. In addition, targeted training can lead to improvements [28].

The basic elements of patient-centred communication are [29]:

- **Waiting:** A pause of 1 to 3 seconds should be allowed after patients' questions or comments.
- **Repetition:** Essential sentences or key words from the patients' last comment should be repeated.
- **Reflection:** If patients speak of their emotions and moods (e.g. anxiety and insecurity), these should be acknowledged.
- **Summarization:** Important aspects of the consultation should be summarized by the physician, in order to signal that the patient has been understood and to avoid misunderstandings.

**Table 1: Appropriate interventions with regard to the stages of behaviour change**

Phase	Description	Possible interventions
Pre-contemplation	The individual has no intention to take action within the next 6 months and is generally unaware or under aware of the problem.	Should aim to create dissonance, for example by giving individual justifications for quitting smoking or losing weight.
Contemplation	The individual intends to take action within the next 6 months. He or she is aware that a problem exists but has not yet made a commitment to take action.	Motivational talks, psycho-education
Preparation	The individual intends to take action within the next 30 days and has taken some behavioural steps in this direction.	Motivational talks, psycho-education, creation of a behaviour plan, plan changes in small steps, offer concrete help, and possibly also registering with an intervention program.
Action	The individual changes his or her overt behaviour for less than 6 months.	Short interventions based on behavioural therapy, smoking cessation courses, sport programs. The patient should be encouraged and asked about problems achieving the relevant goals.
Maintenance	The individual changes his or her overt behaviour for more than 6 months and works to prevent relapse and consolidate the gains attained.	Inquiry, recognition, strengthening, dealing with potential problems. The possibility of relapse and strategies for use in that scenario should be discussed. Offer to get in contact with the doctor again.

## Adherence and health related behaviour

Prochaska and Di Clemente's "transtheoretical model" [30] describes five ideal stages of quitting smoking, which have been found to be similar with regard to other addictions. It may also be used as an analogue for other unhealthy behaviours. The described process begins with a smoker with no motivation to change and ends with a non-smoker who must maintain abstinence.

The first three stages are hardly different from a layperson's perspective – the motivation to change is built here. The actual change in behaviour takes place in the fourth stage. Different strategies for advice and therapy are relevant for different stages (see Table 1).

Frustration may result from therapeutic strategies, which are indiscriminately applied, e.g. when all smokers in a rehabilitation clinic are automatically assigned to a therapy group designed to get participants to quit smoking straight away. According to the stage model, patients who are in the pre-contemplation stage will be at best bored, and in the worst case cause them to completely reject the therapy, which in turn will negatively affect the motivation of other participants. If therapy begins with an analysis of the stage of the process of behaviour change, then it is possible to recognize which persons already have motivation to change, and in which areas motivation can be built or strengthened. Although the theoretical base for this is sound, a systematic Cochrane data analysis [31] has shown that stage-based interventions which were conceptualized according to the transtheoretical model did not perform significantly better than those which were not model-based.

## 4.6 Health related quality of life

Over the last two decades, health related Quality of Life (QoL) has become a relevant parameter to measure the success and progression of the therapy of cardiac patients. Partially due to pressure from regulatory authorities, QoL has become a secondary endpoint next to objective outcomes in particular with regard to therapies, which bring no proven improvement in prognosis or involve serious psychological burden. Early studies, which measured the QoL of cardiac patients were often hampered by methodological shortcomings. Now it is universally accepted that health related QoL is by definition a subjective judgment by the patient and that it is a multidimensional construct. It measures the ability to achieve daily tasks and feelings regarding at least physical, mental and social areas of cognition. The measurement of QoL is generally conducted using self-reported questionnaires, which are either generic or disease-specific. In either case, scores may be built to represent various dimensions of health and sometimes the building of sum scores is allowed. Various reviews [32], [33], [34], [35] have assessed the metric quality and relevance of these instruments to heart patients. Whether a QoL instrument is suitable will largely depend on the question at hand. In any case, validated questionnaires with standardised methods of interpretation should be used. On top of this, one must ensure that the questionnaire is specific enough and sensitive to change, in order to ensure that treatment effects can be effectively measured. These criteria are more likely to be fulfilled by disease specific instruments; however, generic instruments

allow comparisons between patients with different diseases.

Patients with cardiovascular diseases often report impaired QoL when compared to healthy controls. This is determined mostly by the functional severity of the heart disease (e.g. NYHA class, number of ICD shocks), and much less by objective indicators of cardiac or neuroendocrine status. In addition, the psychological status (especially levels of depression) affects QoL. Several studies have shown that impaired QoL is predictive of cardiac events, although it is possible that this is caused by disease severity and affective comorbidities, which themselves are associated with QoL and prognosis [36]. The effect of therapies on the QoL of patients with heart disease is often disappointing with respect to expectations.

## 5 Psychosocial aspects of “classical” cardiovascular risk factors

### 5.1 Hypertension

When considered as a disease of its own, arterial hypertension affects 10–50% of the population, depending on age group. Acute stressors can cause a short-term rise in blood pressure, as seen in “white-coat hypertension”. However, these short-term changes appear to pose no extra risk for the manifestation of arterial hypertension. Chronic stressors have been associated with permanently elevated blood pressure levels [37], especially when stress related coping mechanisms are inadequate. This appears to also be the case with more specific psychological factors such as anxiety, depression, feelings of suppressed anger and hostility as well as alexithymia. Jonas et al. [38] identified anxiety and depression to be predictors of hypertension requiring treatment which manifests 7–16 years later. Furthermore, associations have been shown between post-traumatic stress disorder and elevated systolic and diastolic blood pressure levels [39], [40]. Social networks seem to also have an effect on the manifestation of high blood pressure: the lower the levels of social networking, the higher the risk of incident hypertension [41]. Sleep quality is a sensitive indicator of a lifestyle full of conflict and could recently be shown to be related to a significantly elevated risk for incident hypertension, especially in persons aged <65 years [42].

Clinical symptoms of hypertensive crisis such as retrosternal pressure, dyspnoea or strong vegetative symptoms could lead to a vicious circle of anxiety and continued high blood pressure, due to thoughts about the vital threat posed by the disease. Psychosocial interventions involving, for example, methods of relaxation or biofeedback are capable of reducing blood pressure levels, although the reported effect strengths are relatively small [43], [44].

### 5.2 Hypercholesterinaemia

Psychosocial factors related to depression have been associated with hypercholesterolemia. In a meta-analysis including over 45,000 cases, Shin et al. (2008) [45] came to the conclusion that depression is inversely associated with overall cholesterol levels in persons of both genders. Furthermore, evidence is increasingly showing that very low cholesterol levels are associated with self-harm, violence and suicidal tendencies [46], [47]. The evidence with regard to other factors of the lipid metabolism (especially LDL, HDL and triglycerides) remains inconclusive. A recent investigation by Tedders et al. [48] in around 8,500 persons found a U-shaped relationship between LDL concentrations and depression. Van Reed Dordland et al. [49] found depressive persons to have lower levels of HDL and an elevated triglyceride levels. The importance of these findings remains unclear. The finding that depressed patients have lower cholesterol levels contradicts the evidence that they have an increased risk for CHD. It is possible that depressed patients have an altered composition of lipoproteins. However, research in this area has also led to contradicting results [50], [51].

### 5.3 Smoking

Persons with comorbid psychological disorders (e.g. depression) and low socioeconomic status smoke more and find it harder to quit. For this reason, they require special attention [52]. Systematic strategies for communication have been developed for giving advice and conducting anamneses, which take account of the individual level of motivation and the severity of the addiction [53] (see section 4.5.). Motivational interviewing is more suited for patients with no intention to quit [53], [54]. Advice given per telephone has been shown to be effective in this respect [55]. In Germany, for example, the “Rauchtelefon” (smoker telephone) service is offered by the German Cancer Research Centre (<http://www.dkfz.de/de/rauchtelefon/>) [56]. After 6 months, the rate of abstinence will rise according to the amount of time which has been invested [53], if this exceeds 30 minutes then the effectiveness of the therapy is roughly doubled in comparison to a time investment of under three minutes.

- **Every smoker should be given a clear recommendation to quit. The motivation to change should be built up and practical help should be offered.**

### 5.4 Diabetes mellitus

The effectiveness of diabetes therapy is largely determined by the ability of the patient to conquer the emotional and cognitive challenges posed, and thus apply what they are taught to their daily lives. Interventions to educate and train patients are indispensable, since success relies not only on acceptance of the disease and extensive knowledge, but also on the ability to treat oneself [57].

Psychosocial burdens and psychological symptoms such as depression, anxiety, eating disorders and addiction (e.g. tobacco and alcohol) are large barriers to the success of treatment and require adequate diagnosis and therapy. Depression increases not only the risk of incident type 2 diabetes mellitus [58], but also the morbidity and mortality in patients with either type 1 or type 2 diabetes [59]. Patients with diabetes have a roughly doubled risk of developing depressive symptoms than healthy controls [58], [60]. Psychosomatic primary healthcare, psychotherapy and antidepressant medication have been shown to be effective in treating depressive symptoms in patients with diabetes [61]. A collaborative treatment package for depressive diabetes patients (some of whom have comorbid CHD) has been shown to reduce HbA1c and LDL concentrations in addition to depressive symptoms [62]. Anxiety disorders (including disease specific forms like pathological fear of hypoglycaemia and fear of injections) worsen QoL and metabolic regulation in patients with type 1 and 2 diabetes [63]. Primary psychosomatic therapy, sometimes in combination with antidepressive medication, offers effective therapy options [57]. Eating disorders with psychological causes can also have a negative effect on the metabolism and the prognosis of diabetes patients (e.g. via "insulin purging", when doses of insulin are not taken on purpose with the aim of losing weight) [64]. Psychotherapy and, in patients with obesity, programs to help manage weight are effective in improving symptoms and metabolism regulation [65].

## 5.5 Obesity/malnutrition

Psychosocial factors have a strong influence on eating habits and alcohol consumption. Anxiety and personality disorders like depression resulting from both work or private life can lead to a higher intake of calories and reduced physical activity and may thus lead to an increase in Body Mass Index (BMI) [66]. Obesity can lead to a negative self-esteem, which may in turn lead to the development of depression, which is associated with increases in body weight. So-called binge eating (BE) is a special form of eating disorder, which is characterized by episodic over-eating without any undertakings to regulate weight thereafter – this generally leads to weight gain obesity [65]. Obesity plays an important role in the metabolic syndrome (abdominal obesity, high blood pressure, dyslipidaemia and insulin resistance). As BMI increases, so does the risk for CHD and myocardial infarction, whereby comorbid depression has a synergistic effect [67]. Nonetheless, patients with chronic CHD or heart failure with a moderately increased BMI (BMI <30) have been shown to have a better prognosis [68]. Amongst therapies for persons with a high BMI, professional programs to help patients manage weight, which focus on eating habits and lifestyle changes, are the most important. If a patient has a comorbid depressive disorder then this should be taken into account by the therapy. If several psychotherapeutic interventions have been ineffective for a patient, then the possibility of bariatric sur-

gery should be considered, and a psychotherapist should be actively involved in the discussion.

## 5.6 Physical inactivity

In patients with CHD, cardiovascular training not only reduces all-cause mortality by 27% and cardiac mortality by 31% [69], but also reduces the burden of risk factors. Active motivation to increase physical activity is a central pillar of rehabilitation and thus also secondary prevention in cardiac patients. However, only a third of patients in Europe utilize the possibilities on offer [70]. There is a need for programs, which encourage and support stable long-term behaviour changes.

Cardiovascular training is also considered an effective treatment for depressive disorders [71] and is applicable to older patients [72]. So far there is, however, only indirect evidence of this antidepressive effect in patients with CHD [73] since targeted, randomized studies have yet to be carried out. An antidepressive effect was found in a meta-analysis of studies of patients with various chronic diseases, in which the subgroup with cardiovascular diseases had a particularly strong effect [74]. Patients with depression often have much difficulty in initiating and maintaining cardiovascular training due to a lack of motivation. Group training programs, which are easily reachable and take place at regular times, may be particularly helpful when the antidepressive effect of sport and social support are combined.

The following strategies have been proven to be effective at motivating heart patients in clinical practice to do regular cardiovascular fitness training [75], [76]:

- Questioning of the patients' attitudes (subjective disease theory), knowledge and requirements (e.g. previous sport experience), in order to individualize recommendations.
- Offering stadium specific interventions (see [72], [73]) after evaluation of the patient through discussions (e.g. heart sport groups).

## 6 Coronary heart disease

### 6.1 Psychosocial risk factors and comorbidity

Psychosocial factors are not only associated with an increased CHD incidence, but also with inferior QoL and increased risk of future cardiac events in patients with established CHD. Several plausible, psychobiological mechanisms for these observations have been identified, such as changes in the autonomic nervous system and the endocrine and inflammatory systems [77], [78]. Behavioural mechanisms include unhealthy nutrition, lower physical activity and other harmful behaviours such as smoking and excessive alcohol consumption [79], [80]. Low social status is one of the primary psychosocial risk factors, as measured by school and professional educa-



tion, family income or a perceived low position in the professional hierarchy. These factors are associated with an increased risk for CHD related mortality (relative risk [RR]: 1.3 to 2.0) [80], [81]. Chronic stress at work may result from a combination of high expectations and low decisional control (demand-control model) or a combination of a large workload and relatively low rewards (effort-reward imbalance model) [82]. These conditions are associated with an increased coronary risk in men [77], [81]. With regard to women, however, there have been no clear results [83]. Being engaged in work-shifts, including working at night, for several years or working excessive over hours is also associated with increased risk for CHD [84]. For women, family conflicts as well as the combination of having to work and care for the family increase their risk (RR: 2.9 resp. 4.0) [2]. Social isolation and a lack of social support are associated with unfavourable prognoses in patients already diagnosed with CHD (RR: 1.5; 3.0) [9] (see Part 4.2.).

A person with “type D personality” has a chronic tendency toward negative feelings such as depression, anxiety and irritability (negative affectivity). According to recent meta-analyses, when combined with a strong social inhibition, type D personality predicts a notably worsened prognosis in persons with CHD [85], [86]. Despite this, the most recent studies have been unable to replicate these results in patients with heart failure or mixed cardiac diagnoses [87], [88], [89].

Not only major depression, but also subsyndromal depressive symptoms increase the risk for the development of CHD (RR: 1.6 to 1.9) [90] as well as for an untoward course once the disease is established (OR: 1.6 to 2.4) [90]. The syndrome known as “vital exhaustion”, which involves mainly symptoms of tiredness and greatly impaired drive has also been found to increase the risk of new cardiac events (e.g. myocardial infarction or cardiac death) by a factor of 1.7 [91].

- **Patients with CHD should be screened for depression. A positive screening should be followed by further diagnostic measures and therapy, where appropriate.**

The prevalence of depression following a clinical manifestation of CHD ranges from 20 to 50%. Amongst these cases, 15–20% have clinically significant disorders such as episodes of major depression. The remaining cases may have depressive adjustment disorders or dysthymia. In about half of the patients, depressive symptoms remit following routine cardiologic treatment. In the other half, symptoms persist for months or years if specific psychotherapeutic or pharmacologic interventions are not applied [92]. Overall, patients with severe mental disorders – notably schizophrenia – have a greatly reduced life expectancy; and cardiovascular diseases seem to be the main cause [93].

New meta-analyses have shown that acute, severe anxiety disorders are associated with increased CHD incidence (RR: 1.3) [94] as well as poorer prognosis after myocardial infarction [95]. However, in CHD patients who have not yet experienced an acute coronary syndrome, or have a

normal ejection fraction, anxiety is associated with improved CHD prognosis [96]. Hostility and anger form the etiopathologic core of the “type A personality”, which was largely propagated between 1965 and 2000. New meta-analyses have shown that the presence of these factors is associated with a mild yet statistically significant increased risk in both healthy persons and those with CHD (HR: 1.2) [97]. The “type A personality” concept has not proved to be a valid indicator of prognosis [98].

After an acute cardiac event, some patients develop symptoms of recurring intrusive memories, feelings of numbness or alterations in arousal and reactivity (e.g., hypervigilance), which in some patients may be serious enough to warrant a diagnosis of post-traumatic stress disorder (PTSD). Such symptoms are associated with severe impairments of QoL and increase the risk for further cardiac events [99].

## 6.2 Psychological factors and risk of acute coronary syndromes

Acute mental stressors may act as triggers of acute coronary syndromes [100], stress-induced cardiomyopathy [5] or ventricular tachycardia. These stressors include exposure to a natural catastrophe such as an earthquake [101] as well as personal stressors, e.g. defeat or other serious life events [102].

In the pre-hospital phase of an acute myocardial infarction, psychological factors are decisive contributors to delay in rapidly receiving cardiologic therapy (decision behaviour). The reasons for delay in treatment caused by patients’ behaviour are still incompletely understood. Therefore, it is difficult to create specific prevention strategies. One risk factor associated with a poor prognosis of an ongoing acute myocardial infarction is the tendency for *denial*. This attitude may dispose patients with threatening symptoms of an imminent infarct, such as long-lasting angina pectoris, to not correctly interpret their severity [103]. As a result, the patients delay their decision to seek medical care.

- **Patients who survived an acute coronary syndrome should be provided with strategies to reduce the length of the prehospital phase of any new coronary event.**

## 6.3 Psychosocial interventions for primary and secondary prevention

Intervention studies in cardiac primary prevention have to date almost exclusively assessed behavioural and somatic risk factors [104]. The interventions have mostly been “multidisciplinary”, involving various techniques such as nutrition advice, physical exercise and psychosocial therapy. The effects of psychosocial components cannot be formally isolated from these studies. A clear understanding of the effectiveness of primary intervention measures to facilitate coping with psychosocial stressors is thus currently not possible.

A larger number of studies have been conducted to determine the effectiveness of psychosocial interventions for secondary prevention. Most studies reported positive effects on quality of life, health behaviour, and somatic risk profile, whilst several reported a protective effect on cardiovascular morbidity and mortality [8], [105]. Men appear to profit more from the interventions than women. Programs, which were initiated at least two months after the cardiac event showed stronger effects on the rate of future events than those initiated immediately after [8]. The meta-analysis by Whalley et al. [105] specifically investigated the effect of psychosocial interventions in the secondary prevention of CHD. The positive effects of these interventions on quality of life, depressive symptoms and anxiety could be affirmed, as well as an effect on cardiac mortality. However, the incidence of reinfarction or the need for invasive therapy did not change between groups [105].

Several controlled studies have investigated the effectiveness of psychotherapeutic interventions in treating significant depressive syndromes in CHD patients. Positive, yet moderate effects on depressive symptoms were reported in most of the studies, which evaluated various techniques such as cognitive behavioural therapy [106], [107], [108], [109], interpersonal therapy [110], and "collaborative treatment" [62], [111]. This conclusion was also shared by a recent Cochrane review [112]. The ENRICHD study [106] aimed to investigate the effect of psychotherapy on mortality and reinfarction rates. However, no differences were shown for these main outcomes between the study groups. Nonetheless, methodological issues have been raised, indicating that these results do not prove the ineffectiveness of the intervention. New approaches explicitly aim for an improvement in stress-coping and social support via group psychotherapy. Two current randomized studies showed significant improvements in cardiac prognosis, particularly for women [6], [7].

Secondary analyses of major intervention studies suggest that successfully treating depression lowers mortality, whereas non-response is associated with an increased cardiac risk [113], [114]. However, the difficulties when treating depression in coronary patients should not be underestimated. Whilst it is possible that individualized approaches may be more likely to improve prognosis [109]; the most suitable form of treatment has yet to be determined [115]. Currently, a German study (SPIRR-CAD) [116] is evaluating a tiered psychotherapy in depressive CHD patients.

## 6.4 Psychopharmacotherapy of patients with CHD

Although CHD and depression are two of the most important diseases from a worldwide perspective, only a small number of controlled, randomized studies have been conducted, which evaluate the effectiveness of antidepressant medication in patients with CHD. A meta-analysis of data available at the beginning of 2010 is based on

three studies with a total of 355 patients and a comparable number of control participants (whereby changes in depression severity served as the outcome criterion) [110], [117], [118]. Analyses of these data suggested that the pharmacological therapy was moderately superior to placebo. The analysis was flawed by the fact that the patient groups were not homogenous. Thus, it is possible that patients who respond well to the treatments were not identified.

Specific factors need to be considered when analysing the incidence and course of depression in CHD patients. Subgroup analyses of the aforementioned studies demonstrated an increased benefit for the pharmacological therapy of the post infarction depression in patients who already had a moderate or severe depressive episode prior to the acute coronary syndrome, or in those who suffered from a severe recurrent depression [117], [119]. In the chronic phase of CHD, patients with a comorbid severe recurrent depressive disorder profited from antidepressant pharmacotherapy [109], [110]. These findings refer to the pharmacotherapy with the selective serotonin reuptake inhibitors (SSRI) Sertraline (post infarction depression) and Citalopram (depression in chronic CHD patients) – similar effects can also be expected from other medicaments of this substance category.

With regard to patients with CHD, SSRIs and the substance Mirtazapin have most commonly been evaluated [120]. In these studies, the use of these medications was not associated with an increase in the incidence of relevant, cardiac related adverse drug reactions (ADR). However, the group-sizes were not large enough to detect rare side-effects. Examples of these rare but important ADRs include the induction of heart rhythm disorders with a danger of sudden cardiac death and of an increased bleeding risk. The evaluation of the associated risk of heart rhythm disorders whilst under treatment with antidepressants is difficult, since both the underlying disease (depression) and the treatment (antidepressants) can increase the risk [121]. Correcting for the effect of depression, Whang et al. [122] found a positive relationship between the use of antidepressant medication and sudden cardiac death (Hazard Ratio: 3.3); 61% of the participants took SSRIs. However, this observation refers only to women. Other investigators observed a reduction in the incidence of cardiac events when under treatment with SSRIs. A post-hoc analysis of the ENRICHD-data demonstrated a relative mortality risk of 0.59 for those who had been treated with the SSRI Sertraline [123].

Since SSRIs interfere with the serotonin metabolism of blood platelets, the use of these substances may lead to an increased bleeding risk in these patients, who are mostly also undergoing antiplatelet therapy. Nonetheless, it is still unclear whether the risk is genuinely elevated, since both positive [124] as well as negative results [125] have been published.

The use of tri- and tetracyclic antidepressants (TCA) has been associated with relevant cardiac side-effects [126], which may be explained by the chinidin-like, adrenergic and anticholinergic effects of these agents. As a result,

these substances should not be used for the pharmacotherapy of CHD patients, or should only be administered after a careful risk/benefit analysis. Although the antidepressant Venlafaxin is widely used, there are no randomized or controlled studies evaluating this therapy in patients with comorbid heart disease. In elderly depressed patients a relevant rise in blood pressure was observed in 24% of normotensive and 54% of hypertensive patients [127]; as a result, caution is necessary. There is too little data to allow any recommendations with regard to hypericum extract (St. John's wort). This substance can cause relevant changes in plasma concentrations and the clinical effect of other medicaments (such as anticoagulants and immunosuppressants).

Despite the lack of data, clinicians must make decisions about the initiation of antidepressant pharmacotherapy. When considering this, the potential benefits (such as remission of depression, reduction of the risk of a recurrence, reduction of suicide risk) and the potential harm (such as ADRs including the risk of sudden cardiac death) must be accounted for. Limitations of use, contraindications and warning notices, in particular the notes of regulatory authorities on Citalopram and Escitalopram from 2011, must be considered. ECGs are recommended to evaluate the QT interval prior to the initiation of therapy with SSRIs and during the maintenance phase. In addition, interactions of antidepressants with other medicaments (e.g. Amiodoron or Sotalol) must be considered. It remains unclear whether a pharmacological antidepressant therapy in patients with CHD improves the cardiac prognosis [123], [128]. It has, however, repeatedly been shown that patients whose depression cannot be improved by antidepressant therapy have an unfavourable cardiac prognosis [114], [129] and require special attention.

- **If pharmacological antidepressant therapy is indicated in a CHD patient, selective serotonin reuptake inhibitors are recommended. Contraindications and warning notices regarding these substances must be carefully considered.**
- **Tri- and tetracyclic antidepressants (TCA) should not be used for pharmacological therapy of CHD patients, or should only be administered after a careful risk/benefit analysis.**

## 6.5 Mental health and heart surgery/heart transplantation

The majority of studies on mental health comorbidities in patients undergoing heart operations focus on coronary bypass operations and heart transplantations. There is very little data with regard to operations on the aorta, heart valves and corrections of congenital heart disease in adulthood. The consequences of heart operations during childhood on patients and their relatives will not be covered here.

The psychological stress of an aorto-coronary bypass operation is highest during the time period before the oper-

ation and then decreases during postoperative recovery. This is accompanied by an improvement in quality of life, which, in most studies, is large, stable over time and leads to higher levels as compared to the preoperative state. The value of these observations is nonetheless limited, since the preoperative quality of life is strongly influenced by the fear of the intervention and also often by depressed mood. Minimally invasive techniques seem to have little advantage over standard methods with regard to psychological stress.

The negative effect of depressed mood on quality of life and psychosocial functioning after a bypass operation has been clearly proven. Furthermore, there is evidence that preoperative depressed mood or the presence of a depressive disorder predict an increase of subsequent cardiovascular morbidity and mortality [130], [131], [132]. Despite improved surgical techniques and shorter operation times, neuropsychiatric syndromes (adjustment disorder, delirium) are detectable in up to a third of patients for several hours or days. It appears that a longer duration of acute symptoms may be a risk factor for postoperative mental disorders, such as post-traumatic stress disorder (PTSD). Cognitive and memory deficits are still detectable in about 20% of patients after several months. Cognitive deficits that persist for over a year have only been observed in a small subgroup, and a causal relationship between cognitive deficits and heart operations has yet to be proven [133]. With regard to heart valve operations, comorbid depression [134] and heart related anxiety [135] during the postoperative phase are associated with reduced quality of life. As a result, these patients deserve particular attention.

Also, comorbid mental health conditions such as depression, anxiety and PTSD are common in patients before and after heart transplantation. The strongest symptom severity and the lowest health related quality of life are reported whilst waiting for a donor heart. In the first years after transplantation the psychosocial situation of the majority of patients improves dramatically. In the long run, however, medical complications often lead to an increase in anxiety and depression. Poor adherence to treatment regimens is often a symptom of a mental disorder. High levels of depression predict increased mortality after transplantation [136], [137]. Approximately 10% of patients develop PTSD after transplantation, which is associated with increased mortality [138] and reduced quality of life [139].

- **Before and after heart transplantation, a careful mental health evaluation and, where appropriate, treatment are mandatory.**

Partners of patients undergoing heart surgery often suffer from anxiety, PTSD and depression, in particular when patients spend long periods of time in an intensive care unit [140], had undergone a heart transplantation [141] or were subjected to long periods of bridging with an artificial heart before transplantation [142]. The partners of patients often experience psychological stress which often significantly exceeds that of the patients' themselves.

Programs which offer psychosocial support to patients before cardiac operations are generally positively assessed. However, evaluations of symptom severity and parameters of disease progression show both positive and negative results. It is recommended that patients who are awaiting heart transplantation be supervised by an interdisciplinary team, in which it is possible to address psychosocial problems. Generally speaking, a diagnosed mental health disorder does not contraindicate heart transplantation; it is an indication for an appropriate therapy. After transplantation, a re-evaluation of mental health symptoms should be carried out.

## 7 Cardiac arrhythmias

### 7.1 Psychosocial factors, triggers and comorbidity

Supraventricular tachycardia (SVT) often leads to considerable anxiety and thus impaired quality of life. On the one hand, spontaneously terminating SVTs can be misinterpreted as panic attacks [143], on the other hand, palpitations and mild sinus tachycardias should not be diagnosed as SVTs requiring treatment in patients with anxiety, in particular those with panic disorders. To date, potential psychological triggers of SVT have been inadequately investigated.

In contrast to this, depression and anxiety (in men) and hostility have been shown to predict both new and recurrent atrial fibrillation in selected studies. Patients often report that anxiety and stress triggers this arrhythmia [144]. The data currently available is not adequate enough to generalize these findings, however. The quality of life of atrial fibrillation patients is lower in comparison to healthy controls [145]. Research focusing psychosocial aspects of AF patients is still in its beginnings.

The implantable cardioverter/defibrillator (ICD) is a standard therapy in patients who have survived a life-threatening cardiac arrhythmia. This therapy is also an option in the primary prevention of select patients with a high risk for cardiac arrhythmia. Coping with the presence of a permanent ICD requires many adaptations even in the normal patient. These include the experience of dependence on the device, the behaviour of relatives, complications in sex life, anticipation of shocks, and questions about the end of life [146].

- **The mental health consequences of ICD therapy should be given particular attention.**

Complications, as well as appropriate and disproportionate shocks are not uncommon and often burden the patient greatly. There is a current position paper on the handling of shock applications (including psychosocial aspects) in ICD patients by the European Heart Rhythm Association (EHRA) [147].

As detailed in a systematic review [148], the prevalence of mental health disorders in ICD carriers vary widely according to methodology. The estimated prevalence of

depression varies from 11% to 28% (when using structured interviews). For depressive symptoms the prevalence was found to range from 5% to 41% (using self-administered questionnaires). A similarly strong dependence on the methods of data collection and diagnosis arise when looking at anxiety: 11 to 26% of ICD carriers suffer from an anxiety disorder (using structured interviews) and 8% to 63% suffered from symptoms of anxiety (using self-administered questionnaires). Phobic anxiety is 10 times more common in ICD patients than in the general population (prevalence rates vary from 0.2% to 2.6%) and the prevalence tends to increase further as the disease progresses [149].

It is important to consider mental health comorbidities in the long term care of ICD patients. For example, depression is an established risk factor for adequate shock applications and all-cause mortality [150], [151]. PTSD has also been shown to be a relevant predictor of all-cause mortality in ICD patients [152].

### 7.2 Psychosocial interventions in patients with implantable cardioverter-defibrillators (ICD)

Pedersen et al. [153] and Salmoirago-Blotcher & Ockene [154] conducted systematic reviews investigating the results of psychotherapeutic intervention studies in ICD patients, which aimed to treat anxiety and depression. Both reviews shared the conclusion that no meaningful, empirically based interpretations can be made of the studies due to methodological shortcomings (small numbers of cases, lack of randomization, high drop-out rates, low participation rates).

It is also still unclear at what time the interventions should be initiated. The majority of intervention studies included patients at the time of implantation. This period of time is perhaps too early to influence psychological parameters. Patients generally tend to experience a phase of adjustment to the implantation, which they mostly cope with effectively on their own [155]. Intervening too soon could have adverse consequences [156]. Telephone-based follow-up care using elements of behavioural therapy which is offered to ICD patients aged over 65 years leads to increased levels of self-reported anxiety. Psychotherapeutic treatment concepts should therefore be reserved for patients with psychological comorbidities or traumatic sequelae of aversive experiences [157].

### 7.3 Psychopharmacological treatment of patients with severe cardiac arrhythmias

To our knowledge there are no interpretable data from controlled trials on the safety of psychopharmacological treatments in patients with pronounced cardiac arrhythmias. Since these patients often have severe cardiac dysfunction, the correspondent warning notices and contraindications, in particular those for tricyclic antidepressants,

the SSRIs Citalopram and Escitalopram, as well as the antipsychotic Haloperidol, should especially be considered. Psychotropic drugs known to elongate the QT interval should be avoided ([158], <http://www.torsades.org/>). Drug interactions through the cytochrome P450 system, which lead to increased concentrations of potentially arrhythmogenic substances, must be considered. The antidepressant Mirtazapine and the antipsychotic Olanzapin lower the risk of adverse effects in patients with cardiac arrhythmias. Special caution is to be taken when combining antidepressants with antipsychotics from the Phenothiazine group, since this combination can lead to significantly elongated QT intervals and has been associated with sudden cardiac death in certain situations [159].

- **When giving psychopharmacological therapy to patients with pronounced cardiac arrhythmias, the dangers of drug interactions and QTc elongation must be considered.**

## 8 Chronic heart failure

### Depression and quality of life

The prevalence of depressive disorders is 2 to 4 times higher in patients with chronic heart failure in comparison to the general population. Even mild depressive symptoms significantly increase the risk of hospitalization and mortality in this group [160].

Depression in patients with heart failure can easily go unnoticed in clinical practice, since symptoms such as impaired concentration, tiredness, lack of drive and sleep can themselves result from the cardiac disease. On top of this, patients tend to not share this information and are often reclusive. Patients with heart failure often suffer from significantly impaired quality of life [161]. Self-reported physical and mental quality of life scores tend to be lower as the clinical severity of heart failure (as measured by the NYHA class) declines [162]. Comorbid mental disorders lead to further worsening of quality of life [163].

- **Patients with chronic heart failure should be screened for depression. If screened positive, patients should receive further diagnostics and therapy, if appropriate.**

### 8.1 Illness behaviour (adherence)

Patients with heart failure show reduced adherence to required changes in their life style and prescribed medication. Only 20–60% of patients take the medicines prescribed by their doctor reliably [164], [165]. Other therapeutic recommendations such as the restriction of intake of water and sodium, weighing oneself daily, increase in physical activity, abstinence from smoking as well as reductions in alcohol consumption are often followed insufficiently [165]. A lack of adherence is an important cause of cardiac decompensation, repeated hospitalizations and doctor consultations as well as loss

of productivity at work [165], [166]. Mental disorders such as anxiety, depression and cognitive dysfunction are associated with worsened adherence and prognosis. These disorders make it harder for patients to appraise their health status quickly and to react appropriately [167].

### 8.2 Psychosocial interventions and psychotropic medication

Several courses of action have been proposed in order to improve this situation. A meta-analysis by McAllister et al. showed that educational programs to improve disease coping reduced the relative risk of renewed hospitalizations by 34% [168]. To overcome memory problems, reminders of dosages on drug packages are effective in helping patients with cognitive impairment to reduce future hospitalizations and the risk of death [166].

The disease progression can be positively influenced by regular control visits, for example via telephone monitoring. Recent studies, however, have failed to report any positive effect on survival (TIM-HF und TELE-HF) [169]. In contrast to this, Angermann and her colleagues report that a collaborative care program adapted to patients' needs did not only increase quality of life, but also improved survival in the intervention group [170].

Physical training is recommendable, depending on the disease state of individual patients [171]. An important problem which needs addressing here is lack of adherence to exercise recommendations [172]. It also seems appropriate to involve and advise family members of patients. Patients with chronic heart failure and depression or anxiety disorders should be offered psychoeducation as a part of primary psychosomatic health care. If this does not suffice, then further treatment options should be considered such as stress management training and psychotherapy [173]. Psychopharmacological therapy should only be used with special attention to the cardiovascular disease, potential undesirable side effects and known interactions with other medicines. Regular ECG and lab controls are necessary. The use of tricyclic antidepressants should be avoided due to their potential proarrhythmic effects. SSRIs are favourable, since they have been shown to have few cardiovascular side effects, even though an FDA alert warned that larger doses of Citalopram (>40 mg) have the potential of inducing Torsade de Pointes tachycardias and QT elongations. However, it appears that SSRIs may not be superior to placebo therapy, as was suggested by the SADHART-CHF trial [174]. Also, the survival of patients was not affected in that trial. Further controlled trials are needed in order to assess the effect of antidepressant pharmacotherapy and psychotherapy or a combination of both in patients with heart failure.

## 9 Functional heart complaints

Heart-related symptoms such as palpitations or chest pain in cardiac patients do not necessarily result from an organic disease, but may instead be functional in nature. Such functional heart complaints are not only a problem in the differential diagnosis and course of heart diseases, but also affect the doctor-patient relationship in general. Furthermore, functional symptoms lead to subjective impairment which is often more pronounced than the one caused by chronic CHD [175]. The causes of these symptoms may be extra-cardiac somatic diseases (e.g. reflux disease, chest wall syndrome etc.), but also may be psychogenic, which is the case in 7–17% of primary care patients with chest pain [176]. The reported prevalence of mental disorders is as high as 50% and above in selected patient samples from cardiac emergency care or invasive diagnostics, particularly in patients in whom no cardiac substrate for their complaints could be verified [176]. The diagnostic categories of the ICD-10 do not cover these syndromes thoroughly. As a result, care-givers classify them in different categories, such as anxiety disorders (e.g. panic disorder), depression with heart-related symptoms or somatoform disorders (such as heart-related hypochondria or somatoform autonomic dysfunctions of the cardiovascular system).

Since patients generally assume that they suffer from an organic illness and tend to demand diagnostic clarification, this may lead to an exaggerated use of medical diagnostics with high costs, yet without seminal results. Although it is often necessary to exclude somatic pathology, this alone does not prove the psychogenic nature of the patient's complaints. A positive diagnosis of the underlying mental or psychosomatic disorder should therefore be attempted. In the absence of new clinical information, repeated somatic tests or procedures are generally contraindicated. A few small studies could show that psycho-education and psychotherapy are effective in these patients [177], [178], [179]. It also seems that antidepressant medication improves symptoms [180], whilst short educational interventions by nurses show no effect [181]. Evidence from large, controlled trials is still lacking.

- **Functional heart complaints are present in both men and women with or without organic heart diseases. Besides excluding a somatic illness they require a positive diagnosis of underlying psychopathology.**

## 10 Practical consequences

### 10.1 For general practitioners and practicing cardiologists

General practitioners and cardiologists who treat patients in the long term have the opportunities to recognize psychosocial risks and to raise the issue. When a patient develops a new heart disease, they can estimate the need

for secondary prevention in the form of extra psychosocial offers and can provide concrete advice on further options. While taking the patient's history, they should ask not only for classical risk factors, but should also for personal and environmental factors such as chronic stress at the workplace or at home. It is important to determine whether there are signs of negative affectivity, especially anxiety or depressed mood, or whether the patient has a low social status or shows signs of having an inadequate social network. In order to increase the sensitivity of the diagnostic process, screening methods can be employed. Self-rating questionnaires have been found useful for this purpose, for example the "Patient Health Questionnaire" [182], which helps to identify depression, anxiety or somatoform disorders, or the Hospital Anxiety and Depression Scale [183] for identifying depression and anxiety.

Such screening should be followed by defined diagnostic and therapeutic measures to be used when specific symptom thresholds are exceeded. The primary aim is to reduce the excessive rate of undiagnosed and untreated psychological comorbidities in cardiology patients. In addition to reducing the subjective burden of the disease, these measures might be able to improve the cardiac prognosis. It is possible to create a stable, long-term and trusting working relationship using simple conversational techniques (see 4.5). If the patient suffers from a severe mental disorder such as severe depression, personality disorder or heavy addiction, then he/she should be referred to a suitable specialist.

In order to improve the implementation of the aforementioned measures, we believe it would be wise to expand the regulations on curricula for specialty training in cardiologists to include "competences in the diagnosis and therapy of psychosocial factors in cardiology". Professional training courses have been developed and implemented to teach these competences (<http://www.akademie.dgk.org/>). The medical reimbursement schedule should also reflect these services.

### 10.2 In the acute care hospital

In acute care hospitals, the diagnosis and treatment of cardiac symptoms are of primary importance. Psychosocial issues receive less attention, since the daily routine involves complex procedures, time pressure and since patients do not stay in the clinic for a sufficiently long time. Nonetheless, it seems that adequate diagnosis and therapy of relevant psychological comorbidities are necessary.

Psychosomatic or psychiatric consultation services are generally only available in university hospitals (and even here only on a limited basis) and in some specialized hospitals. The situation could be improved if the primary care-givers acquired basic competences in psychocardiology; appropriate training curricula are available for this purpose (see above). The following aims should be worked towards when implementing psychocardiological knowledge in acute-care hospitals:

- The ability to recognize psychological and psychosomatic disorders and problems even in patients with complex cardiac diseases (diagnostic competence)
- The ability to offer limited consultations, for example during ward rounds or medical briefings before discharge. This may help to support coping, overcome emotional crises provide patients with information and advice. Family members and partners should be involved (communicative competence).
- The ability to decide on indications for treatment, referrals to specialized outpatient or inpatient psychotherapy or for involving a psychosomatic or psychiatric consultation service (competence in differential diagnosis and therapy indication).

In the past years, psychosomatic inpatient units for cardiology patients with severe mental comorbidities have been developed. These encompass not only cardiac diagnosis and treatment but simultaneously also individually tailored psychotherapeutic interventions and physiotherapy. The complexity of the diseases and treatments requires excellent cardiological and psychotherapeutic competences as well as regular team conferences and supervision. Such services are currently only available in select specialized institutions.

### 10.3 Rehabilitation

During cardiac rehabilitation, which is often offered after a coronary event or cardiac surgery, there is an opportunity to focus on identifying and possibly modifying comorbidities or behaviours, which have been shown to have a negative effect on cardiac outcomes. By screening all patients at the beginning of rehabilitation, one can improve the detection of mental disorders which are associated with an increased risk for cardiac events [173]. In order to detect depression and anxiety, various screening instruments have been developed (e.g. the Hospital Anxiety and Depression Scale (HADS)). If a patient reports high distress levels then further evaluation by clinical interview should be conducted. If a clinically relevant depression or anxiety disorder is detected then these should be treated according to guidelines.

Problems with concentration and memory should ideally be clarified by a neuropsychologist in order to prevent poor adherence and train patients with coping skills relevant to their daily lives. Psycho-educational services (in the form of lectures or group discussions) during rehabilitation should be aimed towards teaching patients about factors which help them to cope with their disease: for example the importance of stable social relationships, stress management and the ability to relax. Explaining and taking the stigma out of psychological issues supports the acceptance of psychological services for individuals and groups.

In addition to diagnosing psychological and neuropsychological problems and preventive psychoeducation, change-oriented group interventions are also indicated. Such measures sharpen the awareness of risk factors such as

smoking, unhealthy levels of alcohol consumption and physical inactivity, and also build the first motivations to look into health promoting behavioural changes.

If there is any need for further psychological or neuropsychological treatment, then the patient should be referred to a professional institution whilst still in the rehabilitation phase. In practice (depending on the disorder and local service availability) this involves a search for psychiatrists, physicians specialized in psychosomatic medicine, psychotherapists or specialized pain or memory clinics located close to the patient's home. Unfortunately, it is not yet possible to satisfactorily provide a seamless transition to psychotherapeutic care [184].

These wide ranging psychosocial services require multidisciplinary exchange within interprofessional teams consisting of psychologists, neuropsychologists, social workers, ergotherapists and cardiologists in the cardiac rehabilitation facilities. This would require a ratio of 1 psychologist per 40 patients. Currently, German health insurance companies and pension funds cover a ratio of 1:80 [185]. At least one psychologist per institution should be an approved psychotherapist and additional psychotherapeutic qualifications in physician team members are desirable.

## 11 Summary of recommendations

Psychosocial risk factors should be taken account of when assessing the risk for CHD. There are both social factors to consider, such as low social status, a lack of social support, death of a partner and loneliness; as well as psychological factors (depression, depressed mood, vital exhaustion, anxiety, hostility and post-traumatic stress disorder). These factors are independent, etiological risk factors for the development of CHD and negatively affect prognosis for complications during treatment. *Recommendation level: I, Evidence grade A*

The doctor-patient interactions should follow the principles of patient-centred communication. Age and sex-specific psychosocial aspects should be considered. *Recommendation level: I, Evidence grade C*

Treatment of psychosocial risk factors with the aim of primary prevention of CHD should only be conducted when the risk factor itself is a diagnosable disorder (for example depression) or when the factor worsens classical risk factors. *Recommendation level: I, Evidence grade B*

Individually tailored treatment services should be recommended to all patients with CHD. These may include education, exercise and movement therapy, motivational support with regard to healthy behaviours, relaxation methods and stress management. *Recommendation level: IIa, Evidence grade B*

Psychotherapy should be offered to CHD patients with affective comorbidity in order to improve quality of life. *Recommendation level: IIa, Evidence grade A*

Psychotherapeutic strategies should be adapted to the specific needs of the CHD patient. Sex specific aspects

should be taken account of. *Recommendation level: IIb, Evidence grade B*

Antidepressant pharmacotherapy of depression after an acute coronary syndrome should be offered to patients with at least moderate, recurring depressive disorders. In this case, selective serotonin reuptake inhibitors (SSRIs) are preferable. *Recommendation level: I, Evidence grade B*

Patients with chronic CHD with at least moderate depression should be offered antidepressant medication from the serotonin reuptake inhibitor (SSRI) group, especially when suffering a relapse. *Recommendation level: I, Evidence grade B*

The long-term care of ICD patients should consider the psychosocial consequences of ICD technology. In particular, relevant affective disorders or crises should be detected and treated according to guidelines. Specific recommendations for psychotherapeutic interventions are not currently available. *Recommendation level: I, Evidence grade B*

Patients undergoing cardiac surgery should be cared for by an interdisciplinary team, offering the opportunity to address psychosocial issues, since these patients often suffer from mental disorders such as depression, anxiety and post-traumatic stress disorder, which worsen prognosis. *Recommendation level: I, Evidence grade A*

Patients with chronic heart failure and comorbid depression should receive offers within the framework of primary psychosomatic healthcare. If these do not suffice, then psychotherapy should be considered as further treatment option. *Recommendation level: IIa, Evidence grade B*

Patients with heart failure and comorbid depression should only be treated with pharmacological therapy after a careful risk-benefit analysis, since no benefit has been shown thus far for antidepressants in these patients. *Recommendation level: IIb, Evidence grade B*

Patients with relevant cardiovascular diseases should not be treated with tricyclic antidepressants. *Recommendation level: III, Evidence grade B*

## 12 Planned date for an update of the position paper

Considering the pace of scientific developments of this subject matter, it is necessary to continually review the contents of this position paper. Randomized therapy trials are currently being conducted to assess the effect of psychotherapy and antidepressants on depressed mood in patients with coronary heart disease and heart failure. Results of these trials should be available in three years' time. Relevant results are also expected from other areas of cardiology (e.g. patients with atrial fibrillation) as well as perspectives from new psychopathological concepts. An update of this position paper is planned for March, 2017.

## Notes

### First authorship shared

Ladwig KH and Lederbogen F contributed equally to this work.

### Conflicts of interests

The expert group wrote this paper without any financial support or scientific information from any third party. There were no co-operations with any industry partners.

### German version

The German version of this position paper has been published in the journal "Der Kardiologe" [186].

## References

1. Orth-Gomér K, Wamala SP, Horsten M, Schenck-Gustafsson K, Schneiderman N, Mittleman MA. Marital stress worsens prognosis in women with coronary heart disease: The Stockholm Female Coronary Risk Study. *JAMA*. 2000 Dec;284(23):3008-14. DOI: 10.1001/jama.284.23.3008
2. Low CA, Thurston RC, Matthews KA. Psychosocial factors in the development of heart disease in women: current research and future directions. *Psychosom Med*. 2010 Nov;72(9):842-54. DOI: 10.1097/PSY.0b013e3181f6934f
3. Ladwig KH, Marten-Mittag B, Formanek B, Dammann G. Gender differences of symptom reporting and medical health care utilization in the German population. *Eur J Epidemiol*. 2000 Jun;16(6):511-8. DOI: 10.1023/A:1007629920752
4. Büchner B, Kleiber C, Stanske B, Herrmann-Lingen C. Stress und Herzkrankheit bei Frauen Geschlechtsspezifische Risiken, Bewältigungsprobleme und Behandlungsansätze [Stress and heart disease in women]. *Herz*. 2005 Aug;30(5):416-28. DOI: 10.1007/s00059-005-2703-8
5. Wittstein IS, Thiemann DR, Lima JA, Baughman KL, Schulman SP, Gerstenblith G, Wu KC, Rade JJ, Bivalacqua TJ, Champion HC. Neurohumoral features of myocardial stunning due to sudden emotional stress. *N Engl J Med*. 2005 Feb;352(6):539-48. DOI: 10.1056/NEJMoa043046
6. Orth-Gomér K, Schneiderman N, Wang HX, Walldin C, Blom M, Jernberg T. Stress reduction prolongs life in women with coronary disease: the Stockholm Women's Intervention Trial for Coronary Heart Disease (SWITCHD). *Circ Cardiovasc Qual Outcomes*. 2009 Jan;2(1):25-32. DOI: 10.1161/CIRCOUTCOMES.108.812859
7. Gulliksson M, Burell G, Vessby B, Lundin L, Toss H, Svärdsudd K. Randomized controlled trial of cognitive behavioral therapy vs standard treatment to prevent recurrent cardiovascular events in patients with coronary heart disease: Secondary Prevention in Uppsala Primary Health Care project (SUPRIM). *Arch Intern Med*. 2011 Jan;171(2):134-40. DOI: 10.1001/archinternmed.2010.510
8. Linden W, Phillips MJ, Leclerc J. Psychological treatment of cardiac patients: a meta-analysis. *Eur Heart J*. 2007 Dec;28(24):2972-84. DOI: 10.1093/eurheartj/ehm504
9. Barth J, Schneider S, von Känel R. Lack of social support in the etiology and the prognosis of coronary heart disease: a systematic review and meta-analysis. *Psychosom Med*. 2010 Apr;72(3):229-38. DOI: 10.1097/PSY.0b013e3181d01611



10. Parkes CM, Benjamin B, Fitzgerald RG. Broken heart: a statistical study of increased mortality among widowers. *Br Med J*. 1969 Mar;1(5646):740-3. DOI: 10.1136/bmj.1.5646.740
11. Mostofsky E, Maclure M, Sherwood JB, Tofler GH, Muller JE, Mittleman MA. Risk of acute myocardial infarction after the death of a significant person in one's life: the Determinants of Myocardial Infarction Onset Study. *Circulation*. 2012 Jan;125(3):491-6. DOI: 10.1161/CIRCULATIONAHA.111.061770
12. Hemminki K, Jiang Y. Cancer risks among long-standing spouses. *Br J Cancer*. 2002 Jun;86(11):1737-40. DOI: 10.1038/sj.bjc.6600302
13. Pedersen SS, Sears SF, Burg MM, Van Den Broek KC. Does ICD indication affect quality of life and levels of distress? *Pacing Clin Electrophysiol*. 2009 Feb;32(2):153-6. DOI: 10.1111/j.1540-8159.2008.02196.x
14. Titscher G, Schöppel C. Die Bedeutung der Paarbeziehung für Genese und Verlauf der koronaren Herzkrankheit. Frankfurt/Main: Verlag für Akamedische Schriften; 2000.
15. Ji J, Zöller B, Sundquist K, Sundquist J. Increased risks of coronary heart disease and stroke among spousal caregivers of cancer patients. *Circulation*. 2012 Apr;125(14):1742-7. DOI: 10.1161/CIRCULATIONAHA.111.057018
16. Bromberg JI, Beasley PJ, D'Angelo EJ, Landzberg M, DeMaso DR. Depression and anxiety in adults with congenital heart disease: a pilot study. *Heart Lung*. 2003 Mar-Apr;32(2):105-10. DOI: 10.1067/mhl.2003.26
17. Massaro AN, El-Dib M, Glass P, Aly H. Factors associated with adverse neurodevelopmental outcomes in infants with congenital heart disease. *Brain Dev*. 2007 Aug;30(7):437-46. DOI: 10.1016/j.braindev.2007.12.013
18. Warnes CA, Williams RG, Bashore TM, Child JS, Connolly HM, Dearani JA, del Nido P, Fasules JW, Graham TP Jr, Hijazi ZM, Hunt SA, King ME, Landzberg MJ, Miner PD, Radford MJ, Walsh EP, Webb GD. ACC/AHA 2008 Guidelines for the Management of Adults with Congenital Heart Disease: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (writing committee to develop guidelines on the management of adults with congenital heart disease). *Circulation*. 2008 Dec;118(23):e714-833. DOI: 10.1161/CIRCULATIONAHA.108.190690
19. Barker DJ, Gluckman PD, Godfrey KM, Harding JE, Owens JA, Robinson JS. Fetal nutrition and cardiovascular disease in adult life. *Lancet*. 1993 Apr 10;341(8850):938-41. DOI: 10.1016/0140-6736(93)91224-A
20. Dong M, Giles WH, Felitti VJ, Dube SR, Williams JE, Chapman DP, Anda RF. Insights into causal pathways for ischemic heart disease: adverse childhood experiences study. *Circulation*. 2004 Sep;110(13):1761-6. DOI: 10.1161/01.CIR.0000143074.54995.7F
21. Clark AP, McDougall G. Cognitive impairment in heart failure. *Dimens Crit Care Nurs*. 2006 May-Jun;25(3):93-100. DOI: 10.1097/00003465-200605000-00001
22. Dickson VV, Tkacs N, Riegel B. Cognitive influences on self-care decision making in persons with heart failure. *Am Heart J*. 2007 Sep;154(3):424-31. DOI: 10.1016/j.ahj.2007.04.058
23. Vogels RL, Scheltens P, Schroeder-Tanka JM, Weinstein HC. Cognitive impairment in heart failure: a systematic review of the literature. *Eur J Heart Fail*. 2007 May;9(5):440-9. DOI: 10.1016/j.ejheart.2006.11.001
24. Kindermann I, Fischer D, Karbach J, Link A, Walenta K, Barth C, Ukena C, Mahfoud F, Köllner V, Kindermann M, Böhm M. Cognitive function in patients with decompensated heart failure: the Cognitive Impairment in Heart Failure (CogImpair-HF) study. *Eur J Heart Fail*. 2012 Apr;14(4):404-13. DOI: 10.1093/eurjhf/hfs015
25. Qiu C, Winblad B, Marengoni A, Klarin I, Fastbom J, Fratiglioni L. Heart failure and risk of dementia and Alzheimer disease: a population-based cohort study. *Arch Intern Med*. 2006 May;166(9):1003-8. DOI: 10.1001/archinte.166.9.1003
26. Sachs GA, Carter R, Holtz LR, Smith F, Stump TE, Tu W, Callahan CM. Cognitive impairment: an independent predictor of excess mortality: a cohort study. *Ann Intern Med*. 2011 Sep;155(5):300-8. DOI: 10.7326/0003-4819-155-5-201109060-00007
27. Finset A. Research on person-centred clinical care. *J Eval Clin Pract*. 2011 Apr;17(2):384-6. DOI: 10.1111/j.1365-2753.2010.01608.x
28. Lewin SA, Skea ZC, Entwistle V, Zwarenstein M, Dick J. Interventions for providers to promote a patient-centred approach in clinical consultations. *Cochrane Database Syst Rev*. 2001;(4):CD003267. DOI: 10.1002/14651858.CD003267
29. Adler RH, Herzog W, Joraschky P, Köhle K, Langewitz W, Söllner W, Wesiack W, editors. Uexküll Psychosomatische Medizin. Theoretische Modelle und klinische Praxis. 7. Aufl. München: Urban & Fischer; 2011.
30. Prochaska JO, DiClemente CC. Stages and processes of self-change of smoking: toward an integrative model of change. *J Consult Clin Psychol*. 1983 Jun;51(3):390-5. DOI: 10.1037/0022-006X.51.3.390
31. Cahill K, Lancaster T, Green N. Stage-based interventions for smoking cessation. *Cochrane Database Syst Rev*. 2010;(11):CD004492. DOI: 10.1002/14651858.CD004492.pub4
32. Thompson DR, Yu CM. Quality of life in patients with coronary heart disease-I: assessment tools. *Health Qual Life Outcomes*. 2003;1:42. DOI: 10.1186/1477-7525-1-42
33. Eurich DT, Johnson JA, Reid KJ, Spertus JA. Assessing responsiveness of generic and specific health related quality of life measures in heart failure. *Health Qual Life Outcomes*. 2006;4:89. DOI: 10.1186/1477-7525-4-89
34. Garin O, Ferrer M, Pont A, Rué M, Kotzeva A, Wiklund I, Van Ganse E, Alonso J. Disease-specific health-related quality of life questionnaires for heart failure: a systematic review with meta-analyses. *Qual Life Res*. 2009 Feb;18(1):71-85. DOI: 10.1007/s11136-008-9416-4
35. Dyer MT, Goldsmith KA, Sharples LS, Buxton MJ. A review of health utilities using the EQ-5D in studies of cardiovascular disease. *Health Qual Life Outcomes*. 2010;8:13. DOI: 10.1186/1477-7525-8-13
36. Fallor H, Störk S, Schowalter M, Steinbüchel T, Wollner V, Ertl G, Angermann CE. Is health-related quality of life an independent predictor of survival in patients with chronic heart failure? *J Psychosom Res*. 2007 Nov;63(5):533-8. DOI: 10.1016/j.jpsychores.2007.06.026
37. Sparrenberger F, Cicheler FT, Ascoli AM, Fonseca FP, Weiss G, Berwanger O, Fuchs SC, Moreira LB, Fuchs FD. Does psychosocial stress cause hypertension? A systematic review of observational studies. *J Hum Hypertens*. 2009 Jan;23(1):12-9. DOI: 10.1038/jhh.2008.74
38. Jonas BS, Franks P, Ingram DD. Are symptoms of anxiety and depression risk factors for hypertension? Longitudinal evidence from the National Health and Nutrition Examination Survey I Epidemiologic Follow-up Study. *Arch Fam Med*. 1997 Jan-Feb;6(1):43-9. DOI: 10.1001/archfami.6.1.43
39. Abouzeid M, Kelsall HL, Forbes AB, Sim MR, Creamer MC. Posttraumatic stress disorder and hypertension in Australian veterans of the 1991 Gulf War. *J Psychosom Res*. 2012 Jan;72(1):33-8. DOI: 10.1016/j.jpsychores.2011.08.002

40. Kinzie JD, Riley C, McFarland B, Hayes M, Boehnlein J, Leung P, Adams G. High prevalence rates of diabetes and hypertension among refugee psychiatric patients. *J Nerv Ment Dis.* 2008 Feb;196(2):108-12. DOI: 10.1097/NMD.0b013e318162aa51
41. Kaplan MS, Nunes A. The psychosocial determinants of hypertension. *Nutr Metab Cardiovasc Dis.* 2003 Feb;13(1):52-9. DOI: 10.1016/S0939-4753(03)80168-0
42. Wang Q, Xi B, Liu M, Zhang Y, Fu M. Short sleep duration is associated with hypertension risk among adults: a systematic review and meta-analysis. *Hypertens Res.* 2012 Oct;35(10):1012-8. DOI: 10.1038/hr.2012.91.
43. Linden W, Moseley JV. The efficacy of behavioral treatments for hypertension. *Appl Psychophysiol Biofeedback.* 2006 Mar;31(1):51-63. DOI: 10.1007/s10484-006-9004-8
44. Dickinson HO, Campbell F, Beyer FR, Nicolson DJ, Cook JV, Ford GA, Mason JM. Relaxation therapies for the management of primary hypertension in adults. *Cochrane Database Syst Rev.* 2008 Jan 23;(1):CD004935. DOI: 10.1002/14651858.CD004935.pub2
45. Shin JY, Suls J, Martin R. Are cholesterol and depression inversely related? A meta-analysis of the association between two cardiac risk factors. *Ann Behav Med.* 2008 Aug;36(1):33-43. DOI: 10.1007/s12160-008-9045-8
46. Golomb BA. Cholesterol and violence: is there a connection? *Ann Intern Med.* 1998 Mar;128(6):478-87. DOI: 10.7326/0003-4819-128-6-199803150-00009
47. Garland MR, Hallahan B, McNamara M, Carney PA, Grimes H, Hibbeln JR, Harkin A, Conroy RM. Lipids and essential fatty acids in patients presenting with self-harm. *Br J Psychiatry.* 2007 Feb;190:112-7. DOI: 10.1192/bjp.bp.105.019562
48. Tedders SH, Fokong KD, McKenzie LE, Wesley C, Yu L, Zhang J. Low cholesterol is associated with depression among US household population. *J Affect Disord.* 2011 Dec;135(1-3):115-21. DOI: 10.1016/j.jad.2011.06.045
49. van Reedt Dortland AK, Giltay EJ, van Veen T, van Pelt J, Zitman FG, Penninx BW. Associations between serum lipids and major depressive disorder: results from the Netherlands Study of Depression and Anxiety (NESDA). *J Clin Psychiatry.* 2010 Jun;71(6):729-36. DOI: 10.4088/JCP.08m04865blu
50. Lehto SM, Ruusunen A, Niskanen L, Tolmunen T, Voutilainen S, Viinamäki H, Kaplan GA, Kauhanen J. Elevated depressive symptoms and compositional changes in LDL particles in middle-aged men. *Eur J Epidemiol.* 2010 Jun;25(6):403-9. DOI: 10.1007/s10654-010-9457-1
51. Hummel J, Westphal S, Weber-Hamann B, Gilles M, Lederbogen F, Angermeier T, Luley C, Deuschle M, Kopf D. Serum lipoproteins improve after successful pharmacologic antidepressant treatment: a randomized open-label prospective trial. *J Clin Psychiatry.* 2011 Jul;72(7):885-91. DOI: 10.4088/JCP.09m05853blu
52. Köllner V. Tabakentwöhnung - eine Aufgabe für die ärztliche Psychotherapie? [Tobacco control - a task of medical psychotherapy?]. *Arzt Psychother.* 2011;6(4):217-22.
53. Fiore MC, Baker TB. Clinical practice. Treating smokers in the health care setting. *N Engl J Med.* 2011 Sep;365(13):1222-31. DOI: 10.1056/NEJMcp1101512
54. Lai DT, Cahill K, Qin Y, Tang JL. Motivational interviewing for smoking cessation. *Cochrane Database Syst Rev.* 2010;(1):CD006936. DOI: 10.1002/14651858.CD006936.pub2
55. Stead LF, Hartmann-Boyce J, Perera R, Lancaster T. Telephone counselling for smoking cessation. *Cochrane Database Syst Rev.* 2013 Aug 12;8:CD002850. DOI: 10.1002/14651858.CD002850.pub3
56. Pötschke-Langer M, Schütz J, Kohn R, Lindinger P. Das Rauchertelefon des Deutschen Krebsforschungszentrums. *Psychotherapie im Dialog.* 2008;9(4):378-81. DOI: 10.1055/s-0028-1090066
57. Kulzer B, Albus C, Herpertz S, Kruse J, Lange K, Lederbogen F, Petrak F. Praxisleitlinie der DGK: Psychosoziales und Diabetes mellitus. *Diabetologie.* 2010;5:S139-45. DOI: 10.1055/s-0030-1262634
58. Mezuk B, Eaton WW, Albrecht S, Golden SH. Depression and type 2 diabetes over the lifespan: a meta-analysis. *Diabetes Care.* 2008 Dec;31(12):2383-90. DOI: 10.2337/dc08-0985
59. Chida Y, Hamer M. An association of adverse psychosocial factors with diabetes mellitus: a meta-analytic review of longitudinal cohort studies. *Diabetologia.* 2008 Dec;51(12):2168-78. DOI: 10.1007/s00125-008-1154-1
60. Barnard KD, Skinner TC, Peveler R. The prevalence of co-morbid depression in adults with Type 1 diabetes: systematic literature review. *Diabet Med.* 2006 Apr;23(4):445-8. DOI: 10.1111/j.1464-5491.2006.01814.x
61. van der Feltz-Cornelis CM, Nuyen J, Stoop C, Chan J, Jacobson AM, Katon W, Snoek F, Sartorius N. Effect of interventions for major depressive disorder and significant depressive symptoms in patients with diabetes mellitus: a systematic review and meta-analysis. *Gen Hosp Psychiatry.* 2010 Jul-Aug;32(4):380-95. DOI: 10.1016/j.genhosppsych.2010.03.011
62. Katon WJ, Lin EH, Von Korff M, Ciechanowski P, Ludman EJ, Young B, Peterson D, Rutter CM, McGregor M, McCulloch D. Collaborative care for patients with depression and chronic illnesses. *N Engl J Med.* 2010 Dec;363(27):2611-20. DOI: 10.1056/NEJMoa1003955
63. Anderson RJ, Grigsby AB, Freedland KE, de Groot M, McGill JB, Clouse RE, Lustman PJ. Anxiety and poor glycemic control: a meta-analytic review of the literature. *Int J Psychiatry Med.* 2002;32(3):235-47. DOI: 10.2190/KLGD-4H8D-4RYL-TWQ8
64. Goebel-Fabbri AE, Fikkan J, Franko DL, Pearson K, Anderson BJ, Weinger K. Insulin restriction and associated morbidity and mortality in women with type 1 diabetes. *Diabetes Care.* 2008 Mar;31(3):415-9. DOI: 10.2337/dc07-2026
65. Deutsche Gesellschaft für Psychosomatische Medizin und Psychotherapie; Deutsches Kollegium für Psychosomatische Medizin, editors. Diagnostik und Therapie der Essstörungen. S3-Leitlinie. Registernummer 051/026. Stand: 12.12.2010. AWMF; 2010. Available from: <http://www.awmf.org/leitlinien/detail/II/051-026.html>
66. Zhao G, Ford ES, Li C, Strine TW, Dhingra S, Berry JT, Mokdad AH. Serious psychological distress and its associations with body mass index: findings from the 2007 Behavioral Risk Factor Surveillance System. *Int J Public Health.* 2009 Jun;54 Suppl 1:30-6. DOI: 10.1007/s00038-009-0004-3
67. Ladwig KH, Marten-Mittag B, Löwel H, Döring A, Wichmann HE. Synergistic effects of depressed mood and obesity on long-term cardiovascular risks in 1510 obese men and women: results from the MONICA-KORA Augsburg Cohort Study 1984-1998. *Int J Obes (Lond).* 2006 Sep;30(9):1408-14. DOI: 10.1038/sj.ijo.0803285
68. Logue J, Murray HM, Welsh P, Shepherd J, Packard C, Macfarlane P, Cobbe S, Ford I, Sattar N. Obesity is associated with fatal coronary heart disease independently of traditional risk factors and deprivation. *Heart.* 2011 Apr;97(7):564-8. DOI: 10.1136/hrt.2010.211201
69. Jolliffe JA, Rees K, Taylor RS, Thompson D, Oldridge N, Ebrahim S. Exercise-based rehabilitation for coronary heart disease. *Cochrane Database Syst Rev.* 2001;(1):CD001800. DOI: 10.1002/14651858.CD001800

70. Piepoli MF, Corrà U, Benzer W, Bjarnason-Wehrens B, Dendale P, Gaita D, McGee H, Mendes M, Niebauer J, Zwisler AD, Schmid JP; Cardiac Rehabilitation Section of the European Association of Cardiovascular Prevention and Rehabilitation. Secondary prevention through cardiac rehabilitation: from knowledge to implementation. A position paper from the Cardiac Rehabilitation Section of the European Association of Cardiovascular Prevention and Rehabilitation. *Eur J Cardiovasc Prev Rehabil*. 2010 Feb;17(1):1-17. DOI: 10.1097/HJR.0b013e3283313592
71. Mead GE, Morley W, Campbell P, Greig CA, McMurdo M, Lawlor DA. Exercise for depression. *Cochrane Database Syst Rev*. 2009;(3):CD004366. DOI: 10.1002/14651858.CD004366.pub4
72. Babyak M, Blumenthal JA, Herman S, Khatri P, Doraiswamy M, Moore K, Craighead WE, Baldeewicz TT, Krishnan KR. Exercise treatment for major depression: maintenance of therapeutic benefit at 10 months. *Psychosom Med*. 2000 Sep-Oct;62(5):633-8.
73. Blumenthal JA, Babyak MA, Carney RM, Huber M, Saab PG, Burg MM, Sheps D, Powell L, Taylor CB, Kaufmann PG. Exercise, depression, and mortality after myocardial infarction in the ENRICH trial. *Med Sci Sports Exerc*. 2004 May;36(5):746-55.
74. Herring MP, Puetz TW, O'Connor PJ, Dishman RK. Effect of exercise training on depressive symptoms among patients with a chronic illness: a systematic review and meta-analysis of randomized controlled trials. *Arch Intern Med*. 2012 Jan;172(2):101-11. DOI: 10.1001/archinternmed.2011.696
75. Schlicht W, Kanning M, Bös K. Psychosoziale Interventionen zur Beeinflussung des Risikofaktors Bewegungsmangel. Theoretische Modelle und praktische Evidenzen. Frankfurt: VAS; 2003. (Statuskonferenz Psychokardiologie; 10).
76. European Association of Cardiovascular Prevention and Rehabilitation Committee for Science Guidelines, EACPR; Corrà U, Piepoli MF, Carré F, Heuschmann P, Hoffmann U, Verschuren M, Halcox J, Giannuzzi P, Saner H, Wood D, Piepoli MF, Corrà U, Benzer W, Bjarnason-Wehrens B, Dendale P, Gaita D, McGee H, Mendes M, Niebauer J, Zwisler AD, Schmid JP. Secondary prevention through cardiac rehabilitation: physical activity counselling and exercise training: key components of the position paper from the Cardiac Rehabilitation Section of the European Association of Cardiovascular Prevention and Rehabilitation. *Eur Heart J*. 2010 Aug;31(16):1967-74. DOI: 10.1093/eurheartj/ehq236
77. Rozanski A, Blumenthal JA, Davidson KW, Saab PG, Kubzansky L. The epidemiology, pathophysiology, and management of psychosocial risk factors in cardiac practice: the emerging field of behavioral cardiology. *J Am Coll Cardiol*. 2005 Mar;45(5):637-51. DOI: 10.1016/j.jacc.2004.12.005
78. Dimsdale JE. Psychological stress and cardiovascular disease. *J Am Coll Cardiol*. 2008 Apr;51(13):1237-46. DOI: 10.1016/j.jacc.2007.12.024
79. Hamer M, Molloy GJ, Stamatakis E. Psychological distress as a risk factor for cardiovascular events: pathophysiological and behavioral mechanisms. *J Am Coll Cardiol*. 2008 Dec;52(25):2156-62. DOI: 10.1016/j.jacc.2008.08.057
80. Stringhini S, Sabia S, Shipley M, Brunner E, Nabi H, Kivimäki M, Singh-Manoux A. Association of socioeconomic position with health behaviors and mortality. *JAMA*. 2010 Mar;303(12):1159-66. DOI: 10.1001/jama.2010.297
81. Woodward M, Brindle P, Tunstall-Pedoe H; SIGN group on risk estimation. Adding social deprivation and family history to cardiovascular risk assessment: the ASSIGN score from the Scottish Heart Health Extended Cohort (SHHEC). *Heart*. 2007 Feb;93(2):172-6. DOI: 10.1136/hrt.2006.108167
82. Siegrist J, Dragano N. Psychosoziale Belastungen und Erkrankungsrisiken im Erwerbsleben : Befunde aus internationalen Studien zum Anforderungs-Kontroll-Modell und zum Modell beruflicher Gratifikationskrisen [Psychosocial stress and disease risks in occupational life. Results of international studies on the demand-control and the effort-reward imbalance models]. *Bundesgesundheitsblatt Gesundheitsforschung Gesundheitsschutz*. 2008 Mar;51(3):305-12. DOI: 10.1007/s00103-008-0461-5
83. Eller NH, Netterstrøm B, Gyntelberg F, Kristensen TS, Nielsen F, Steptoe A, Theorell T. Work-related psychosocial factors and the development of ischemic heart disease: a systematic review. *Cardiol Rev*. 2009 Mar-Apr;17(2):83-97. DOI: 10.1097/CRD.0b013e318198c8e9
84. Virtanen M, Ferrie JE, Singh-Manoux A, Shipley MJ, Vahtera J, Marmot MG, Kivimäki M. Overtime work and incident coronary heart disease: the Whitehall II prospective cohort study. *Eur Heart J*. 2010 Jul;31(14):1737-44. DOI: 10.1093/eurheartj/ehq124
85. Denollet J, Schiffer AA, Spek V. A general propensity to psychological distress affects cardiovascular outcomes: evidence from research on the type D (distressed) personality profile. *Circ Cardiovasc Qual Outcomes*. 2010 Sep;3(5):546-57. DOI: 10.1161/CIRCOUTCOMES.109.934406
86. Grande G, Romppel M, Barth J. Association between type D personality and prognosis in patients with cardiovascular diseases: a systematic review and meta-analysis. *Ann Behav Med*. 2012 Jun;43(3):299-310. DOI: 10.1007/s12160-011-9339-0
87. Pelle AJ, Pedersen SS, Schiffer AA, Szabó B, Widdershoven JW, Denollet J. Psychological distress and mortality in systolic heart failure. *Circ Heart Fail*. 2010 Mar;3(2):261-7. DOI: 10.1161/CIRCHEARTFAILURE.109.871483
88. Grande G, Romppel M, Vesper JM, Schubmann R, Glaesmer H, Herrmann-Lingen C. Type D personality and all-cause mortality in cardiac patients—data from a German cohort study. *Psychosom Med*. 2011 Sep;73(7):548-56. DOI: 10.1097/PSY.0b013e318227a9bc
89. Coyne JC, Jaarsma T, Luttik ML, van Sonderen E, van Veldhuisen DJ, Sanderma R. Lack of prognostic value of type D personality for mortality in a large sample of heart failure patients. *Psychosom Med*. 2011 Sep;73(7):557-62. DOI: 10.1097/PSY.0b013e318227ac75
90. Nicholson A, Kuper H, Hemingway H. Depression as an aetiologic and prognostic factor in coronary heart disease: a meta-analysis of 6362 events among 146 538 participants in 54 observational studies. *Eur Heart J*. 2006 Dec;27(23):2763-74. DOI: 10.1093/eurheartj/ehl338
91. Williams JE, Mosley TH Jr, Kop WJ, Couper DJ, Welch VL, Rosamond WD. Vital exhaustion as a risk factor for adverse cardiac events (from the Atherosclerosis Risk In Communities [ARIC] study). *Am J Cardiol*. 2010 Jun;105(12):1661-5. DOI: 10.1016/j.amjcard.2010.01.340
92. Thombs BD, Bass EB, Ford DE, Stewart KJ, Tsilidis KK, Patel U, Fauerbach JA, Bush DE, Ziegelstein RC. Prevalence of depression in survivors of acute myocardial infarction. *J Gen Intern Med*. 2006 Jan;21(1):30-8. DOI: 10.1111/j.1525-1497.2005.00269.x
93. Newcomer JW, Hennekens CH. Severe mental illness and risk of cardiovascular disease. *JAMA*. 2007 Oct;298(15):1794-6. DOI: 10.1001/jama.298.15.1794
94. Roest AM, Martens EJ, de Jonge P, Denollet J. Anxiety and risk of incident coronary heart disease: a meta-analysis. *J Am Coll Cardiol*. 2010 Jun;56(1):38-46. DOI: 10.1016/j.jacc.2010.03.034

95. Roest AM, Martens EJ, Denollet J, de Jonge P. Prognostic association of anxiety post myocardial infarction with mortality and new cardiac events: a meta-analysis. *Psychosom Med.* 2010 Jul;72(6):563-9. DOI: 10.1097/PSY.0b013e3181dbff97
96. Meyer T, Buss U, Herrmann-Lingen C. Role of cardiac disease severity in the predictive value of anxiety for all-cause mortality. *Psychosom Med.* 2010 Jan;72(1):9-15. DOI: 10.1097/PSY.0b013e3181c64fc0
97. Chida Y, Steptoe A. The association of anger and hostility with future coronary heart disease: a meta-analytic review of prospective evidence. *J Am Coll Cardiol.* 2009 Mar;53(11):936-46. DOI: 10.1016/j.jacc.2008.11.044
98. Myrtek M. Meta-analyses of prospective studies on coronary heart disease, type A personality, and hostility. *Int J Cardiol.* 2001 Jul;79(2-3):245-51. DOI: 10.1016/S0167-5273(01)00441-7
99. Spindler H, Pedersen SS. Posttraumatic stress disorder in the wake of heart disease: prevalence, risk factors, and future research directions. *Psychosom Med.* 2005 Sep-Oct;67(5):715-23. DOI: 10.1097/01.psy.0000174995.96183.9b
100. Nawrot TS, Perez L, Künzli N, Munters E, Nemery B. Public health importance of triggers of myocardial infarction: a comparative risk assessment. *Lancet.* 2011 Feb 26;377(9767):732-40. DOI: 10.1016/S0140-6736(10)62296-9
101. Leor J, Kloner RA. The Northridge earthquake as a trigger for acute myocardial infarction. *Am J Cardiol.* 1996 Jun 1;77(14):1230-2. DOI: 10.1016/S0002-9149(96)00169-5
102. Wilbert-Lampen U, Leistner D, Greven S, Pohl T, Sper S, Völker C, Güthlin D, Plasse A, Knez A, Küchenhoff H, Steinbeck G. Cardiovascular events during World Cup soccer. *N Engl J Med.* 2008 Jan;358(5):475-83. DOI: 10.1056/NEJMoa0707427
103. Ladwig KH, Gärtner C, Walz LM, Smenes KR, Ronel J. Die innere Barriere: der Beitrag gesundheitspsychologischer Konzepte zur Erklärung der prahospitalen Verzögerungszeit beim akuten Myokardinfarkt: Eine systematische Literaturanalyse des aktuellen Wissensstandes [The inner barrier: how health psychology concepts contribute to the explanation of prehospital delays in acute myocardial infarction: a systematic analysis of the current state of knowledge]. *Psychother Psychosom Med Psychol.* 2009 Dec;59(12):440-5. DOI: 10.1055/s-2008-1067576
104. Graham I, Atar D, Borch-Johnsen K, Boysen G, Burell G, Cifkova R, Dallongeville J, De Backer G, Ebrahim S, Gjelsvik B, et al. European guidelines on cardiovascular disease prevention in clinical practice: full text. Fourth Joint Task Force of the European Society of Cardiology and other societies on cardiovascular disease prevention in clinical practice (constituted by representatives of nine societies and by invited experts). *Eur J Cardiovasc Prev Rehabil.* 2007 Sep;14 Suppl 2:S1-113. DOI: 10.1097/01.hjr.0000277983.23934.c9
105. Whalley B, Rees K, Davies P, Bennett P, Ebrahim S, Liu Z, West R, Moxham T, Thompson DR, Taylor RS. Psychological interventions for coronary heart disease. *Cochrane Database Syst Rev.* 2011;(8):CD002902. DOI: 10.1002/14651858.CD002902.pub3
106. Berkman LF, Blumenthal J, Burg M, Carney RM, Catellier D, Cowan MJ, Czajkowski SM, DeBusk R, Hosking J, Jaffe A, Kaufmann PG, Mitchell P, Norman J, Powell LH, Raczynski JM, Schneiderman N; Enhancing Recovery in Coronary Heart Disease Patients Investigators (ENRICH). Effects of treating depression and low perceived social support on clinical events after myocardial infarction: the Enhancing Recovery in Coronary Heart Disease Patients (ENRICH) Randomized Trial. *JAMA.* 2003 Jun;289(23):3106-16. DOI: 10.1001/jama.289.23.3106
107. Barth J, Paul J, Härter M, Bengel J. Inpatient psychotherapeutic treatment for cardiac patients with depression in Germany: short term results. *GMS Psychosoc Med.* 2005;2:Doc04. Available from: <http://www.egms.de/en/journals/psm/2005-2/psm000013.shtml>
108. Freedland KE, Skala JA, Carney RM, Rubin EH, Lustman PJ, Dávila-Román VG, Steinmeyer BC, Hogue CW Jr. Treatment of depression after coronary artery bypass surgery: a randomized controlled trial. *Arch Gen Psychiatry.* 2009 Apr;66(4):387-96. DOI: 10.1001/archgenpsychiatry.2009.7
109. Davidson KW, Rieckmann N, Clemow L, Schwartz JE, Shimbo D, Medina V, Albanese G, Kronish I, Hegel M, Burg MM. Enhanced depression care for patients with acute coronary syndrome and persistent depressive symptoms: coronary psychosocial evaluation studies randomized controlled trial. *Arch Intern Med.* 2010 Apr 12;170(7):600-8. DOI: 10.1001/archinternmed.2010.29
110. Lespérance F, Frasere-Smith N, Koszycki D, Laliberté MA, van Zyl LT, Baker B, Swenson JR, Ghatavi K, Abramson BL, Dorian P, Guertin MC; CREATE Investigators. Effects of citalopram and interpersonal psychotherapy on depression in patients with coronary artery disease: the Canadian Cardiac Randomized Evaluation of Antidepressant and Psychotherapy Efficacy (CREATE) trial. *JAMA.* 2007 Jan;297(4):367-79. DOI: 10.1001/jama.297.4.367
111. Rollman BL, Belnap BH, LeMenager MS, Mazumdar S, Houck PR, Counihan PJ, Kapoor WN, Schulberg HC, Reynolds CF 3rd. Telephone-delivered collaborative care for treating post-CABG depression: a randomized controlled trial. *JAMA.* 2009 Nov;302(19):2095-103. DOI: 10.1001/jama.2009.1670
112. Baumeister H, Hutter N, Bengel J. Psychological and pharmacological interventions for depression in patients with coronary artery disease. *Cochrane Database Syst Rev.* 2011;(9):CD008012. DOI: 10.1002/14651858.CD008012.pub3
113. Carney RM, Blumenthal JA, Freedland KE, Youngblood M, Veith RC, Burg MM, Cornell C, Saab PG, Kaufmann PG, Czajkowski SM, Jaffe AS; ENRICH Investigators. Depression and late mortality after myocardial infarction in the Enhancing Recovery in Coronary Heart Disease (ENRICH) study. *Psychosom Med.* 2004 Jul-Aug;66(4):466-74. DOI: 10.1097/01.psy.0000133362.75075.a6
114. de Jonge P, Honig A, van Melle JP, Schene AH, Kuyper AM, Tulner D, Schins A, Ormel J; MIND-IT Investigators. Nonresponse to treatment for depression following myocardial infarction: association with subsequent cardiac events. *Am J Psychiatry.* 2007 Sep;164(9):1371-8. DOI: 10.1176/appi.ajp.2007.06091492
115. Deutsche Gesellschaft für Psychiatrie, Psychotherapie und Nervenheilkunde (DGPPN); Bundesärztekammer (BÄK); Kassenärztliche Bundesvereinigung (KBV), et al. S3-Leitlinie/Nationale VersorgungsLeitlinie Unipolare Depression. Langfassung. 2009. Available from: [http://www.etracker.de/Inkcnt.php?et=WmgYP3&url=http://versorgungsleitlinien.de/themen/depression/pdf/s3\\_nv1\\_depression\\_lang.pdf&linkname=s3\\_nv1\\_depression\\_lang](http://www.etracker.de/Inkcnt.php?et=WmgYP3&url=http://versorgungsleitlinien.de/themen/depression/pdf/s3_nv1_depression_lang.pdf&linkname=s3_nv1_depression_lang)
116. Albus C, Beutel ME, Deter HC, Fritzsche K, Hellmich M, Jordan J, Juenger J, Krauth C, Ladwig KH, Michal M, Mueck-Weymann M, Petrowski K, Pieske B, Ronel J, Soellner W, Waller C, Weber C, Herrmann-Lingen C. A stepwise psychotherapy intervention for reducing risk in coronary artery disease (SPIRR-CAD) - rationale and design of a multicenter, randomized trial in depressed patients with CAD. *J Psychosom Res.* 2011 Oct;71(4):215-22. DOI: 10.1016/j.jpsychores.2011.02.013

117. Glassman AH, O'Connor CM, Califf RM, Swedberg K, Schwartz P, Bigger JT Jr, Krishnan KR, van Zyl LT, Swenson JR, Finkel MS, Landau C, Shapiro PA, Pepine CJ, Mardekian J, Harrison WM, Barton D, McIvor M; Sertraline Antidepressant Heart Attack Randomized Trial (SADHEART) Group. Sertraline treatment of major depression in patients with acute MI or unstable angina. *JAMA*. 2002 Aug 14;288(6):701-9. Erratum in: *JAMA* 2002 Oct 9;288(14):1720. DOI: 10.1001/jama.288.6.701
118. Strik JJ, Honig A, Lousberg R, Lousberg AH, Cherix EC, Tuynman-Qua HG, Kuijpers PM, Wellens HJ, Van Praag HM. Efficacy and safety of fluoxetine in the treatment of patients with major depression after first myocardial infarction: findings from a double-blind, placebo-controlled trial. *Psychosom Med*. 2000 Nov-Dec;62(6):783-9.
119. Glassman AH, Bigger JT, Gaffney M, Shapiro PA, Swenson JR. Onset of major depression associated with acute coronary syndromes: relationship of onset, major depressive disorder history, and episode severity to sertraline benefit. *Arch Gen Psychiatry*. 2006 Mar;63(3):283-8. DOI: 10.1001/archpsyc.63.3.283
120. Honig A, Kuyper AM, Schene AH, van Melle JP, de Jonge P, Tulner DM, Schins A, Crijns HJ, Kuijpers PM, Vossen H, Lousberg R, Ormel J; MIND-IT investigators. Treatment of post-myocardial infarction depressive disorder: a randomized, placebo-controlled trial with mirtazapine. *Psychosom Med*. 2007 Sep-Oct;69(7):606-13. DOI: 10.1097/PSY.0b013e31814b260d
121. Narayan SM, Stein MB. Do depression or antidepressants increase cardiovascular mortality? The absence of proof might be more important than the proof of absence. *J Am Coll Cardiol*. 2009 Mar;53(11):959-61. DOI: 10.1016/j.jacc.2008.12.009
122. Whang W, Kubzansky LD, Kawachi I, Rexrode KM, Kroenke CH, Glynn RJ, Garan H, Albert CM. Depression and risk of sudden cardiac death and coronary heart disease in women: results from the Nurses' Health Study. *J Am Coll Cardiol*. 2009 Mar;53(11):950-8. DOI: 10.1016/j.jacc.2008.10.060
123. Taylor CB, Youngblood ME, Catellier D, Veith RC, Carney RM, Burg MM, Kaufmann PG, Shuster J, Mellman T, Blumenthal JA, Krishnan R, Jaffe AS; ENRICH Investigators. Effects of antidepressant medication on morbidity and mortality in depressed patients after myocardial infarction. *Arch Gen Psychiatry*. 2005 Jul;62(7):792-8. DOI: 10.1001/archpsyc.62.7.792
124. de Abajo FJ, García-Rodríguez LA. Risk of upper gastrointestinal tract bleeding associated with selective serotonin reuptake inhibitors and venlafaxine therapy: interaction with nonsteroidal anti-inflammatory drugs and effect of acid-suppressing agents. *Arch Gen Psychiatry*. 2008 Jul;65(7):795-803. DOI: 10.1001/archpsyc.65.7.795
125. Kim DH, Daskalakis C, Whellan DJ, Whitman IR, Hohmann S, Medvedev S, Kraft WK. Safety of selective serotonin reuptake inhibitor in adults undergoing coronary artery bypass grafting. *Am J Cardiol*. 2009 May;103(10):1391-5. DOI: 10.1016/j.amjcard.2009.01.348
126. Roose SP, Laghrissi-Thode F, Kennedy JS, Nelson JC, Bigger JT Jr, Pollock BG, Gaffney A, Narayan M, Finkel MS, McCafferty J, Gergel I. Comparison of paroxetine and nortriptyline in depressed patients with ischemic heart disease. *JAMA*. 1998 Jan;279(4):287-91. DOI: 10.1001/jama.279.4.287
127. Johnson EM, Whyte E, Mulsant BH, Pollock BG, Weber E, Begley AE, Reynolds CF. Cardiovascular changes associated with venlafaxine in the treatment of late-life depression. *Am J Geriatr Psychiatry*. 2006 Sep;14(9):796-802. DOI: 10.1097/01.JGP.0000204328.50105.b3
128. van Melle JP, de Jonge P, Honig A, Schene AH, Kuyper AM, Crijns HJ, Schins A, Tulner D, van den Berg MP, Ormel J; MIND-IT investigators. Effects of antidepressant treatment following myocardial infarction. *Br J Psychiatry*. 2007 Jun;190:460-6. DOI: 10.1192/bjp.bp.106.028647
129. Glassman AH, Bigger JT Jr, 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 Sep;66(9):1022-9. DOI: 10.1001/archgenpsychiatry.2009.121
130. Connerney I, Shapiro PA, McLaughlin JS, Bagiella E, Sloan RP. Relation between depression after coronary artery bypass surgery and 12-month outcome: a prospective study. *Lancet*. 2001 Nov 24;358(9295):1766-71. DOI: 10.1016/S0140-6736(01)06803-9
131. Blumenthal JA, Lett HS, Babyak MA, White W, Smith PK, Mark DB, Jones R, Mathew JP, Newman MF; NORG Investigators. Depression as a risk factor for mortality after coronary artery bypass surgery. *Lancet*. 2003 Aug 23;362(9384):604-9. DOI: 10.1016/S0140-6736(03)14190-6
132. McKenzie LH, Simpson J, Stewart M. A systematic review of pre-operative predictors of post-operative depression and anxiety in individuals who have undergone coronary artery bypass graft surgery. *Psychol Health Med*. 2010 Jan;15(1):74-93. DOI: 10.1080/13548500903483486
133. Selnes OA, McKhann GM, Borowicz LM Jr, Grega MA. Cognitive and neurobehavioral dysfunction after cardiac bypass procedures. *Neurol Clin*. 2006 Feb;24(1):133-45. DOI: 10.1016/j.ncl.2005.10.001
134. Rimington H, Weinman J, Chambers JB. Predicting outcome after valve replacement. *Heart*. 2010 Jan;96(2):118-23. DOI: 10.1136/hrt.2008.160010
135. Aicher D, Holz A, Feldner S, Köllner V, Schäfers HJ. Quality of life after aortic valve surgery: replacement versus reconstruction. *J Thorac Cardiovasc Surg*. 2011 Aug;142(2):e19-24. DOI: 10.1016/j.jtcvs.2011.02.006
136. Zipfel S, Schneider A, Wild B, Löwe B, Jünger J, Haass M, Sack FU, Bergmann G, Herzog W. Effect of depressive symptoms on survival after heart transplantation. *Psychosom Med*. 2002 Sep-Oct;64(5):740-7. DOI: 10.1097/01.PSY.0000031575.42041.6A
137. Havik OE, Sivertsen B, Relbo A, Hellesvik M, Grov I, Geiran O, Andreassen AK, Simonsen S, Gullestad L. Depressive symptoms and all-cause mortality after heart transplantation. *Transplantation*. 2007 Jul;84(1):97-103. DOI: 10.1097/01.tp.0000268816.90672.a0
138. Dew MA, Kormos RL, Roth LH, Murali S, DiMartini A, Griffith BP. Early post-transplant medical compliance and mental health predict physical morbidity and mortality one to three years after heart transplantation. *J Heart Lung Transplant*. 1999 Jun;18(6):549-62. DOI: 10.1016/S1053-2498(98)00044-8
139. Köllner V, Einsle F, Schade I, Maulhardt T, Gulielmos V, Joraschky P. Psychosoziale Belastung und Lebensqualität bei Patienten nach Herz- oder Lungentransplantation [The influence of anxiety, depression and post-traumatic stress disorder on quality of life after thoracic organ transplantation]. *Z Psychosom Med Psychother*. 2003;49(3):262-74.
140. Young E, Eddleston J, Ingleby S, Streets J, McJanet L, Wang M, Glover L. Returning home after intensive care: a comparison of symptoms of anxiety and depression in ICU and elective cardiac surgery patients and their relatives. *Intensive Care Med*. 2005 Jan;31(1):86-91. DOI: 10.1007/s00134-004-2495-y
141. Dew MA, Myaskovsky L, DiMartini AF, Switzer GE, Schulberg HC, Kormos RL. Onset, timing and risk for depression and anxiety in family caregivers to heart transplant recipients. *Psychol Med*. 2004 Aug;34(6):1065-82. DOI: 10.1017/S0033291703001387

142. Bunzel B, Laederach-Hofmann K, Wieselthaler G, Roethy W, Wolner E. Mechanical circulatory support as a bridge to heart transplantation: what remains? Long-term emotional sequelae in patients and spouses. *J Heart Lung Transplant*. 2007 Apr;26(4):384-9. DOI: 10.1016/j.healun.2007.01.025
143. Medi C, Kalman JM, Freedman SB. Supraventricular tachycardia. *Med J Aust*. 2009 Mar;190(5):255-60.
144. McCabe PJ. Psychological distress in patients diagnosed with atrial fibrillation: the state of the science. *J Cardiovasc Nurs*. 2010 Jan-Feb;25(1):40-51. DOI: 10.1097/JCN.0b013e3181b7be36
145. Thrall G, Lane D, Carroll D, Lip GY. Quality of life in patients with atrial fibrillation: a systematic review. *Am J Med*. 2006 May;119(5):448.e1-19.
146. Ladwig KH, Ischinger NF, Ronel J, Kolb C. Umgang mit ICD-Patienten an ihrem Lebensende: Einstellungen, Wissen und Verhalten von Ärzten und Patienten. Eine kritische Literaturanalyse [Treating ICD patients at the end of their lives: attitudes, knowledge, and behavior of doctors and patients. A critical literature analysis]. *Herzschrittmacherther Elektrophysiol*. 2011 Sep;22(3):151-6. DOI: 10.1007/s00399-011-0138-x
147. Braunschweig F, Boriani G, Bauer A, Hatala R, Herrmann-Lingen C, Kautzner J, Pedersen SS, Pehrson S, Ricci R, Schaliij MJ. Management of patients receiving implantable cardiac defibrillator shocks: recommendations for acute and long-term patient management. *Europace*. 2010 Dec;12(12):1673-90. DOI: 10.1093/europace/euq316
148. Magyar-Russell G, Thombs BD, Cai JX, Baveja T, Kuhl EA, Singh PP, Montenegro Braga Barroso M, Arthurs E, Roseman M, Amin N, Marine JE, Ziegelstein RC. The prevalence of anxiety and depression in adults with implantable cardioverter defibrillators: a systematic review. *J Psychosom Res*. 2011 Oct;71(4):223-31. DOI: 10.1016/j.jpsychores.2011.02.014
149. Cho EN, von Känel R, Marten-Mittag B, Ronel J, Kolb C, Baumert J, Ladwig KH. Determinants and trajectory of phobic anxiety in patients living with an implantable cardioverter defibrillator. *Heart*. 2012 May;98(10):806-12. DOI: 10.1136/heartjnl-2011-301204
150. Whang W, Albert CM, Sears SF Jr, Lampert R, Conti JB, Wang PJ, Singh JP, Ruskin JN, Muller JE, Mittleman MA; TOVA Study Investigators. Depression as a predictor for appropriate shocks among patients with implantable cardioverter-defibrillators: results from the Triggers of Ventricular Arrhythmias (TOVA) study. *J Am Coll Cardiol*. 2005 Apr;45(7):1090-5. DOI: 10.1016/j.jacc.2004.12.053
151. Tzeis S, Kolb C, Baumert J, Reents T, Zrenner B, Deisenhofer I, Ronel J, Andrikopoulos G, Ladwig KH. Effect of depression on mortality in implantable cardioverter defibrillator recipients—findings from the prospective LICAD study. *Pacing Clin Electrophysiol*. 2011 Aug;34(8):991-7. DOI: 10.1111/j.1540-8159.2011.03081.x
152. Ladwig KH, Baumert J, Marten-Mittag B, Kolb C, Zrenner B, Schmitt C. Posttraumatic stress symptoms and predicted mortality in patients with implantable cardioverter-defibrillators: results from the prospective living with an implanted cardioverter-defibrillator study. *Arch Gen Psychiatry*. 2008 Nov;65(11):1324-30. DOI: 10.1001/archpsyc.65.11.1324
153. Pedersen SS, van den Broek KC, Sears SF Jr. Psychological intervention following implantation of an implantable defibrillator: a review and future recommendations. *Pacing Clin Electrophysiol*. 2007 Dec;30(12):1546-54. DOI: 10.1111/j.1540-8159.2007.00905.x
154. Salmoirago-Blotcher E, Ockene IS. Methodological limitations of psychosocial interventions in patients with an implantable cardioverter-defibrillator (ICD) A systematic review. *BMC Cardiovasc Disord*. 2009;9:56. DOI: 10.1186/1471-2261-9-56
155. Irvine J, Firestone J, Ong L, Cribbie R, Dorian P, Harris L, Ritvo P, Katz J, Newman D, Cameron D, Johnson S, Bilanovic A, Hill A, O'Donnell S, Sears S Jr. A randomized controlled trial of cognitive behavior therapy tailored to psychological adaptation to an implantable cardioverter defibrillator. *Psychosom Med*. 2011 Apr;73(3):226-33. DOI: 10.1097/PSY.0b013e31820afc63
156. Crössmann A, Schulz SM, Kühlkamp V, Ritter O, Neuser H, Schumacher B, Bauer W, Pauli P. A randomized controlled trial of secondary prevention of anxiety and distress in a German sample of patients with an implantable cardioverter defibrillator. *Psychosom Med*. 2010 Jun;72(5):434-41. DOI: 10.1097/PSY.0b013e3181d9bcec
157. Jordan J, Titscher G, Kirsch H. Behandlungsmanual zur Psychotherapie von akuten und posttraumatischen Belastungsstörungen nach ICD-Mehrfachschicks [Treatment manual for psychotherapy of acute and posttraumatic stress disorders after multiple ICD shocks]. *Herzschrittmacherther Elektrophysiol*. 2011 Sep;22(3):189-201. DOI: 10.1007/s00399-011-0148-8
158. Wenzel-Seifert K, Wittmann M, Haen E. QTc prolongation by psychotropic drugs and the risk of Torsade de Pointes. *Dtsch Arztebl Int*. 2011 Oct;108(41):687-93. DOI: 10.3238/arztebl.2011.0687
159. Honkola J, Hookana E, Malinen S, Kaikkonen KS, Junttila MJ, Isohanni M, Kortelainen ML, Huikuri HV. Psychotropic medications and the risk of sudden cardiac death during an acute coronary event. *Eur Heart J*. 2012 Mar;33(6):745-51. DOI: 10.1093/eurheartj/ehr368
160. Rutledge T, Reis VA, Linke SE, Greenberg BH, Mills PJ. Depression in heart failure: a meta-analytic review of prevalence, intervention effects, and associations with clinical outcomes. *J Am Coll Cardiol*. 2006 Oct;48(8):1527-37. DOI: 10.1016/j.jacc.2006.06.055
161. Hobbs FD, Kenkre JE, Roalfe AK, Davis RC, Hare R, Davies MK. Impact of heart failure and left ventricular systolic dysfunction on quality of life: a cross-sectional study comparing common chronic cardiac and medical disorders and a representative adult population. *Eur Heart J*. 2002 Dec;23(23):1867-76. DOI: 10.1053/euhj.2002.3255
162. Juenger J, Schellberg D, Kraemer S, Haunstetter A, Zugck C, Herzog W, Haass M. Health related quality of life in patients with congestive heart failure: comparison with other chronic diseases and relation to functional variables. *Heart*. 2002 Mar;87(3):235-41. DOI: 10.1136/heart.87.3.235
163. Faller H, Störk S, Schuler M, Schowalter M, Steinbüchel T, Ertl G, Angermann CE. Depression and disease severity as predictors of health-related quality of life in patients with chronic heart failure – a structural equation modeling approach. *J Card Fail*. 2009 May;15(4):286-292.e2. DOI: 10.1016/j.cardfail.2008.10.022
164. Dickstein K, Cohen-Solal A, Filippatos G, McMurray JJ, Ponikowski P, Poole-Wilson PA, Strömberg A, van Veldhuisen DJ, Atar D, Hoes AW, Keren A, Mebazaa A, Nieminen M, Priori SG, Swedberg K; ESC Committee for Practice Guidelines (CPG). ESC Guidelines for the diagnosis and treatment of acute and chronic heart failure 2008: the Task Force for the Diagnosis and Treatment of Acute and Chronic Heart Failure 2008 of the European Society of Cardiology. Developed in collaboration with the Heart Failure Association of the ESC (HFA) and endorsed by the European Society of Intensive Care Medicine (ESICM). *Eur Heart J*. 2008 Oct;29(19):2388-442. DOI: 10.1093/eurheartj/ehn309
165. van der Wal MH, Jaarsma T, van Veldhuisen DJ. Non-compliance in patients with heart failure; how can we manage it? *Eur J Heart Fail*. 2005 Jan;7(1):5-17. DOI: 10.1016/j.ejheart.2004.04.007
166. Laufs U, Rettig-Ewen V, Böhm M. Strategies to improve drug adherence. *Eur Heart J*. 2011 Feb;32(3):264-8. DOI: 10.1093/eurheartj/ehq297

167. Cutler DM, Everett W. Thinking outside the pillbox – medication adherence as a priority for health care reform. *N Engl J Med*. 2010 Apr;362(17):1553-5. DOI: 10.1056/NEJMp1002305
168. McAlister FA, Lawson FM, Teo KK, Armstrong PW. A systematic review of randomized trials of disease management programs in heart failure. *Am J Med*. 2001 Apr 1;110(5):378-84. DOI: 10.1016/S0002-9343(00)00743-9
169. Koehler F, Winkler S, Schieber M, Sechtem U, Stangl K, Böhm M, de Brouwer S, Perrin E, Baumann G, Gelbrich G, Boll H, Honold M, Koehler K, Kirwan BA, Anker SD. Telemedicine in heart failure: pre-specified and exploratory subgroup analyses from the TIM-HF trial. *Int J Cardiol*. 2012 Nov 29;161(3):143-50. DOI: 10.1016/j.ijcard.2011.09.007
170. Angermann CE, Störk S, Gelbrich G, Faller H, Jahns R, Frantz S, Loeffler M, Ertl G; Competence Network Heart Failure. Mode of action and effects of standardized collaborative disease management on mortality and morbidity in patients with systolic heart failure: the Interdisciplinary Network for Heart Failure (INH) study. *Circ Heart Fail*. 2012 Jan;5(1):25-35. DOI: 10.1161/CIRCHEARTFAILURE.111.962969
171. Piepoli MF, Conraads V, Corrà U, Dickstein K, Francis DP, Jaarsma T, McMurray J, Pieske B, Piotrowicz E, Schmid JP, Anker SD, Solal AC, Filippatos GS, Hoes AW, Gielen S, Giannuzzi P, Ponikowski PP. Exercise training in heart failure: from theory to practice. A consensus document of the Heart Failure Association and the European Association for Cardiovascular Prevention and Rehabilitation. *Eur J Heart Fail*. 2011 Apr;13(4):347-57. DOI: 10.1093/eurjhf/hfr017
172. Conraads VM, Deaton C, Piotrowicz E, Santaularia N, Tierney S, Piepoli MF, Pieske B, Schmid JP, Dickstein K, Ponikowski PP, Jaarsma T. Adherence of heart failure patients to exercise: barriers and possible solutions: a position statement of the Study Group on Exercise Training in Heart Failure of the Heart Failure Association of the European Society of Cardiology. *Eur J Heart Fail*. 2012 May;14(5):451-8. DOI: 10.1093/eurjhf/hfs048
173. Bundesärztekammer; Kassenärztliche Bundesvereinigung; Arbeitsgemeinschaft der Wissenschaftlichen Medizinischen Fachgesellschaften, editors. Nationale VersorgungsLeitlinie Chronische Herzinsuffizienz. Langfassung. Version 1.5. 2012.
174. O'Connor CM, Jiang W, Kuchibhatla M, Silva SG, Cuffe MS, Callwood DD, Zakhary B, Stough WG, Arias RM, Rivelli SK, Krishnan R; SADHART-CHF Investigators. Safety and efficacy of sertraline for depression in patients with heart failure: results of the SADHART-CHF (Sertraline Against Depression and Heart Disease in Chronic Heart Failure) trial. *J Am Coll Cardiol*. 2010 Aug;56(9):692-9. DOI: 10.1016/j.jacc.2010.03.068
175. Albus C, Herrmann-Lingen C. Funktionelle Störungen in der Kardiologie. *Psychosomatik und Konsiliarpsychiatrie*. 2007 Feb;1(2):118-22. DOI: 10.1007/s11800-007-0024-x
176. Deutsche Gesellschaft für Allgemeinmedizin und Familienmedizin (DEGAM), editor. Brustschmerz. DEGAM-Leitlinie Nr. 15. AWMF-Registernummer 053-023. Stand Januar 2011. Düsseldorf: omikron publishing; 2011. Available from: <http://www.awmf.org/leitlinien/detail/ll/053-023.html>
177. Mayou RA, Bryant BM, Sanders D, Bass C, Klimes I, Forfar C. A controlled trial of cognitive behavioural therapy for non-cardiac chest pain. *Psychol Med*. 1997 Sep;27(5):1021-31. DOI: 10.1017/S0033291797005254
178. Mayou R, Sprigings D, Birkhead J, Price J. A randomized controlled trial of a brief educational and psychological intervention for patients presenting to a cardiac clinic with palpitation. *Psychol Med*. 2002 May;32(4):699-706. DOI: 10.1017/S0033291702005536
179. Potts SG, Lewin R, Fox KA, Johnstone EC. Group psychological treatment for chest pain with normal coronary arteries. *QJM*. 1999 Feb;92(2):81-6. DOI: 10.1056/NEJM199405193302003
180. Cannon RO 3rd, Quyyumi AA, Mincemoyer R, Stine AM, Gracely RH, Smith WB, Geraci MF, Black BC, Uhde TW, Waclawiw MA. Imipramine in patients with chest pain despite normal coronary angiograms. *N Engl J Med*. 1994 May;330(20):1411-7. DOI: 10.1056/NEJM199405193302003
181. Sanders D, Bass C, Mayou RA, Goodwin S, Bryant BM, Tyndel S. Non-cardiac chest pain: why was a brief intervention apparently ineffective? *Psychol Med*. 1997 Sep;27(5):1033-40. DOI: 10.1017/S0033291797005266
182. Löwe B, Spitzer RL, Zipfel S, Herzog W. Gesundheitsfragebogen für Patienten (PHQ D). Komplettversion und Kurzform. Testmappe mit Manual, Fragebögen, Schablonen. 1. Aufl. Karlsruhe: Pfizer; 2001.
183. Herrmann-Lingen C, Buss U, Snaith RP. HADS-D: Hospital Anxiety and Depression Scale – Deutsche Version. Deutsche Adaptation der Hospital Anxiety and Depression Scale (HADS) von R. P. Snaith und A. S. Zigmond. 3. aktual. u. neu norm. Aufl. Bern: Hans Huber; 2011
184. Bundespsychotherapeutenkammer, ed. BPTK-Studie zu Wartezeiten in der ambulanten psychotherapeutischen Versorgung. Umfrage der Landespsychotherapeutenkammern und der BPTK. Berlin: BPTK; 2011. Available from: <http://www.bptk.de/publikationen/bptk-studie.html>
185. Deutscher Rentenversicherung Bund, ed. Strukturqualität von Reha-Einrichtungen – Anforderungen der Deutschen Rentenversicherung. Stationäre medizinische Reha-Einrichtungen. Berlin: Deutscher Rentenversicherung Bund; 2010. Available from: [http://www.deutsche-rentenversicherung.de/cae/servlet/contentblob/208182/publicationFile/11642/2010\\_Brosch%C3%BCre\\_Strukturanforderungen.pdf](http://www.deutsche-rentenversicherung.de/cae/servlet/contentblob/208182/publicationFile/11642/2010_Brosch%C3%BCre_Strukturanforderungen.pdf)
186. Ladwig KH, Lederbogen F, Albus C, Angermann C, Borggrefe M, Fischer D, Fritzsche K, Haass M, Jordan J, Jünger J, Kindermann I, Köllner V, Kuhn B, Scherer M, Seyfarth M, Völler H, Waller C, Herrmann-Lingen C. Positionspapier zur Bedeutung psychosozialer Faktoren in der Kardiologie. *Der Kardiologe*; 2013;7(1):7-27. DOI: 10.1007/s12181-012-0478-8

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