Review

# Cardiac-disease-induced PTSD (CDI-PTSD): A systematic review

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ABSTRACT

The goal of the current systematic review was to provide an overview of the findings in the field of Cardiac-Disease-Induced Posttraumatic Stress Disorder (CDI-PTSD) in order to establish CDI-PTSD as a valid diagnostic entity for a wide spectrum of cardiac diseases and related medical procedures. In accordance with PRISMA guidelines, we conducted a systematic electronic literature search. Of the 3202 citations identified, 150 studies meeting the selection criteria were reviewed. Our main findings were that the prevalence of CDI-PTSD ranged between 0% and 38% (averaging at 12%) and was highly dependent on the assessment tool used. The most consistent risk factors are of a psychological nature (e.g., pre-morbid distress). The consequences of CDI-PTSD range from psychosocial difficulties to lack of adherence and heightened mortality rates. Much inconsistency in the field was found with regard to patients who present with diagnoses other than acute coronary syndrome (e.g., cardiac arrest) and who undergo potentially traumatic medical procedures (e.g., defibrillator implantation). Yet the current review seems to strengthen the conceptualization of CDI-PTSD as a valid diagnostic entity, at least with regard to acute cardiac events.

## 1. Introduction

Cardiovascular disease (CVD) is a leading cause of death worldwide (Centers for Disease Control and Prevention, 2016). The number of non-institutionalized adults with diagnosed heart disease in the United States alone is 26.6 million (Blackwell, Lucas, & Clarke, 2014), of whom more than 17 million survived an acute coronary event (Edmondson, 2014).

Among cardiac diseases, the most frequent type is ischemic heart disease (IHD), which is precipitated by coronary artery disease (CAD). CAD causes diminished blood flow to the myocardium (Walker & Lorimer, 2004). When the myocardium receives insufficient blood supply it can result in myocardial ischemia, leading to acute coronary syndrome [ACS; i.e., myocardial infarction (MI) and unstable angina (UA)] and even sudden cardiac arrest (SCA). The most common treatments are catheterization (PTA: percutaneous transluminal angioplasty or PCI: percutaneous coronary intervention); heart surgery (CABG: coronary artery bypass graft); and implantation of a pacemaker or ICD (implantable cardioverter defibrillator)[1] (Falvo, 2014).

Among life-threatening illnesses, cardiac events – and especially ACS – seem to consist of unique traumatizing characteristics. Among

these features are the abruptness of the event, the concrete danger of death, and the patients' intense sense of loss of control and helplessness during the event (Kutz, Shabtai, Solomon, Neumann, & David, 1994). Alonzo (2000) adds that the intrusive experience of the treatments – such as coronary surgery, angioplasty, angiography, pacemaker implantation, stress testing and even the side effects of medications – can also be potentially traumatic events leading to the development of posttraumatic stress disorder (PTSD). In addition, although survival rates from cardiac disease are growing, it is still perceived as a significant threat to one's life. Consequently, it gives rise to intense emotional reactions such as fear or anxiety (of dying or recurrence), anger, sadness, and grief (Fisher & Collins, 2012) as well as PTSD.

The Diagnostic and Statistical Manual of Mental Disorders (DSM-5; APA, 2013) defines posttraumatic stress disorder (PTSD) as a trauma- and stressor-related disorder precipitated by a traumatic event and characterized by symptoms of re-experiencing the trauma, avoidance, negative cognitions and mood, and arousal, which cause significant distress and functional impairment. The diagnosis of PTSD requires at least one month of continuous symptoms following exposure to a traumatic event. Posttraumatic stress symptoms experienced within the first month of the traumatic event are classified as acute stress

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[1] Throughout the review, the term CDI-PTSD (cardiac-disease-induced PTSD) will be used as a generic term indicating PTSD resulting from any of the abovementioned cardiac events or treatment procedures.

disorder (ASD), which presents with a similar clinical picture characterized by intrusion, negative mood, dissociation, avoidance, and arousal (APA, 2013). The inclusion in the fourth edition of the DSM (DSM-IV; APA, 1994) of a life-threatening disease as a potentially traumatic event that might trigger the onset of PTSD prompted a new area of research that pointed to the emergence of PTSD in the aftermath of a cardiac event.

The first evidence of CDI-PTSD was reported by Kutz, Garb, and David (1988), who presented four cases of patients who developed posttraumatic symptoms after a myocardial infarction. Since then, the issue of CDI-PTSD has attracted a great deal of attention, and many studies have examined its prevalence, stability and risk factors as well as its psychological and physiological consequences. The results of these studies have been summarized in review papers and one meta-analysis (e.g., Edmondson, 2014; Edmondson, Richardson, et al., 2012; Edmondson, Shaffer, et al., 2012; Gander & von Känel, 2006; Spindler & Pedersen, 2005; Tedstone & Tarrier, 2003).

Yet alongside the growth of research in this area, a contrary trend –which questions the classification of a life-threatening illness as a traumatic event – has begun to emerge. This opposing perspective has been most vigorously manifested in the reservation added to the recent revision of the DSM (DSM-5; APA, 2013) which states that "a life-threatening illness or debilitating medical condition is not necessarily considered a traumatic event" (DSM-5; APA, 2013, p. 274) which qualifies as a Criterion A for PTSD. As some illnesses and medical procedures may be considered *stressors* rather than "traumatizers," the DSM-5 indicates that the distress they evoke should be diagnosed as an adjustment disorder (DSM-5; APA, 2013) rather than as PTSD.

This position calls into question the validity of the CDI-PTSD diagnosis. Therefore, the main goal of the current review is to establish the validity of this diagnostic entity for a wide spectrum of cardiac diseases and related medical procedures, as well as to pinpoint CDI-PTSD's unique characteristics. In order to achieve these goals, the accumulated empirical evidence regarding CDI-PTSD was examined and compared with the features of PTSD resulting from "traditional" Criterion A events. This comparison of observable data enabled the detection of whether the empirical findings accumulated thus far in the field of CDI-PTSD create a convincing enough "nomological net" (Cronbach & Meehl, 1955) for CDI-PTSD to be accurately regarded as a diagnostic entity. Finally, based on the knowledge from other research fields of trauma and illness, an additional aim was to identify topics which are missing from the existing research in the field.

In this review we cover a wide range of cardiac-related-diseases and procedures: myocardial infarction (MI), coronary artery bypass graft (CABG), implantable cardioverter defibrillator (ICD), cardiovascular disease (CVD), coronary artery disease (CAD), acute coronary syndrome (ACS), heart transplantation (HT) cardiac arrest and heart failure. This review includes all of the publications of empirical studies and reviews that we could find on the subject: the first one dating from the year 1988 (Kutz et al., 1988) and the last one from the end of the calendar year 2015. Our review covers relevant issues related to CDI-PTSD, including frequency, stability over time, risk factors, psychological and physiological reactions, psychological interventions, and the experience of patients' family members.

## 2. Method

The current review was executed in accordance with the PRISMA (Preferred Reporting Items for Systematic Reviews and Meta-Analyses) guidelines for systematic reviews and meta-analyses (Moher, Liberati, Tetzlaff, & Altman, 2009a, 2009b). Electronic supplement 1 presents the PRISMA checklist (consisting of the page location of each of the preferred reporting items). Fig. 1 presents the PRISMA flowchart. An electronic literature search was conducted via PsychINFO, PubMed/Medline, and CINAHL databases beginning in the year 1970 and ending on the last day of December 2015. The search combined the terms

"posttraumatic stress" and "PTSD" with the following terms related to cardiac events: myocardial infarction (MI), acute coronary syndrome, cardiovascular disease/disorder, heart failure, bypass/CABG, cardiac surgery, heart transplantation, percutaneous coronary intervention (PCI), percutaneous transluminal coronary angioplasty (PTCA), coronary heart disease (CHD), coronary artery disease (CAD), implantable cardioverter defibrillator (ICD), angina pectoris, and cardiac arrest. We also searched for other, more general terms which intersected with PTSD: physical illnesses/diseases, chronic illnesses/diseases, and medical illnesses/diseases.

Criteria for inclusion in the review were only peer-reviewed, professional scientific publications, i.e., (1) empirical studies; (2) literature reviews (3) meta-analyses (4) and review articles, as well as book chapters. Only studies that were conducted on adult patients were included, and the manuscripts had to have been published in English. Dissertations and conference abstracts were excluded. In order to make the review as broad as possible, no a-priori selection was applied aside from the above mentioned ones. For example, even studies with only a sub-sample of CVD patients were not excluded, nor did we exclude papers based on the diagnostic tool or instrument used to assess PTSD or PTSS. In addition, in order to get as comprehensive an overview of the studies in the field as possible, no a-priori quality ranking was applied. The initial search located 3202 publications from the three databases, yet this number included duplicate papers.

In order to reduce the retrieval data bias based on a single author only, at least two researchers were involved at each stage of the data search, retrieving and coding. Titles and abstracts of all of the 3202 publications were reviewed by the first author (NV) and her PhD student (KF). Duplicate papers were omitted (n = 1660) as were all publications which examined PTSD-induced cardiac events rather than cardiac-disease-induced-PTSD (n = 1383). This phase yielded 159 relevant publications. The first (NV) and second (KG) researchers examined the reference lists of all relevant publications in an effort to detect publications that might not have been included in the database searches, and as a result of this further examination, nine additional papers were located. Following a thorough reading of each of the 168 articles, done by the first (NV) and second (KG) researchers, 18 papers were excluded due to their previously undetected irrelevancy, resulting in a review of a total of 150 relevant publications (120 empirical studies and an additional 30 literature reviews, theoretical papers or book chapters). The total number of non-overlapping participants in all 120 empirical studies was 8973 (not including healthy controls) (see Fig. 1 for the PRISMA flowchart).

The analytical plan was as follows. First, we grouped the studies according to the main medical diagnostic group on which they focused (all medical diagnoses were established by the medical teams). Seventy-two publications focused on ACS/MI patients, and an additional six focused exclusively on cardiac arrest. The remaining publications selected their study samples according to a specific medical procedure related to cardiac disease such as ICD, CABG and HT, rather than diagnosis. All heterogeneous studies were grouped under the category of MI. Only papers which reported on homogeneous samples in which all patients had undergone CA, CABG or HT were grouped separately (Electronic supplement no. 2 specifies the subsamples detected in each study). To be as inclusive as possible, we refrained from setting any a-priori threshold criteria with regard to the number of MI patients needed to be included in studies. Second, we grouped the papers according to the main researcher leading the studies, thus enabling an overview of studies based on the same database. Finally, all papers presenting empirical studies were integrated into a table pre-designed to contain the following information: year of publication, study design, number of participants, diagnosis, response and attrition rate measures of CDI-PTSD, time points when CDI-PTSD was assessed, prevalence of CDI-PTSD and its stability over time, risk factors associated with CDI-PTSD, and consequences (see Electronic supplement no. 2).
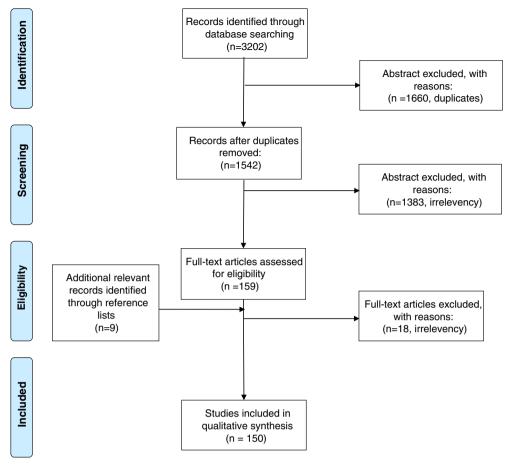
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[Identification]  Records identified through
                  database searching        ──────►  Abstract excluded, with
                  (n=3202)                            reasons:
                                                      (n =1660, duplicates)

[Screening]       Records after duplicates
                  removed:                   ──────►  Abstract excluded, with
                  (n=1542)                            reasons:
                                                      (n=1383, irrelevency)

[Eligibility]  Additional relevant    Full-text articles assessed
               records identified     for eligibility           ──────►  Full-text articles excluded,
               through reference      (n =159)                            with reasons:
               lists                                                      (n=18, irrelevency)
               (n=9)

[Included]                            Studies included in
                                      qualitative synthesis
                                      (n = 150)
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**Fig. 1.** The PRISMA flowchart of the current review.

## 3. Results

Overall, the most common diagnosis was ACS/MI (60% of all papers screened), followed by cardiac arrest (5%). ICD was detected in 12% of the studies, as for CABG (12%) and HT (11%). The minimal time between cardiac event and diagnosis was 48 h, and the maximum was 10 years. (Since many studies applied multiple measurement times, it was impossible to obtain a meaningful calculation of overall mean for the time between cardiac event and diagnosis). The most frequent risk factors[2] found were social support (7%) and age (7%), followed by depression (6%) and negative affectivity (6%).

### 3.1. Former reviews: contributions and shortcomings

During the systematic literature search, we detected the publication of five book chapters and 25 literature reviews including one meta-analysis on this subject, published between the years 2001 (Pedersen, 2001) and 2015 (Moye & Rouse, 2015). Electronic supplement 3 presents a detailed table summarizing the data of all 30 reviews/chapters. While these publications are informative and contributory in their own right, we saw the need for a further review of the accumulated findings in the field. In light of space limitations, an integrative summary of all former reviews and chapters is presented as Electronic supplement 4.

### 3.2. Myocardial infarction (MI)

#### 3.2.1. Prevalence of PTSD

The current section focuses on the studies conducted on ACS patients (mostly MI); therefore, the terms "MI", "CDI (cardiac-disease-induced)," and "ACS" will be used interchangeably. CDI-PTSD in this population was assessed at various time points along the course of the illness, from hospitalization (e.g., Castilla & Vázquez, 2011) to as many as 30 years later (e.g., Chung, Dennis, Berger, Jones, & Rudd, 2011).

Very few studies have focused on the manifestation of CDI-PTSD symptoms during hospitalization or within the first two weeks of the MI (defined as acute stress disorder, or ASD). These studies presented a large variability in the prevalence of MI-induced ASD, ranging from 0% to 26% (e.g., Castilla & Vázquez, 2011; Gao, Zhao, Li, & Cao, 2015; Ginzburg et al., 2003; Meister et al., 2016; Oflaz et al., 2014; Sheldrick, Tarrier, Berry, & Kincey, 2006). An examination of these studies revealed that the prevalence of ASD was a function of the measurement used. When ASD was diagnosed by clinical interview (e.g., Roberge, Dupuis, & Marchande, 2008; Van Driel & Den Velde, 1995), ASD rates were lower than those emerging from studies which used self-report questionnaires (e.g., Gao et al., 2015; Ginzburg et al., 2003; Neumann, 1991). The same pattern was observed in studies that examined CDI-PTSD rates one month after the MI (e.g. Roberge, Dupuis, & Marchande, 2010; Rocha et al., 2008 versus Edmondson et al., 2011; Pedersen, Middle, & Larsen, 2002; Sumner et al., 2015). Finally, studies in which both interviews and self-report questionnaires were applied showed that data collected by self-report yielded higher rates of CDI-PTSD than did data collected via interview (Guler et al., 2009; Rocha et al., 2008; Wiedemar et al., 2008).

Reported rates of PTSD within three to 18 months after the MI ranged from 3% to 21% (e.g., Castilla & Vázquez, 2011). These varia-

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[2] In order to be as faithful as possible to the original data, we used the same theoretical concepts as were used by the original authors with regard to the risk factors studied, and no categorization process was executed by the current review team.

tions in the reported rates may be attributed to a) the difference in sample sizes which in some studies was 50 and lower (e.g., Bennett & Brooke, 1999; Castilla & Vázquez, 2011; Doerfler, 1997; Doerfler, Pbert, & Decosimo, 1994; Girard et al., 2006) and/or b) changes in the defining criteria for PTSD (e.g., a change in cutoff scores: see, for example, Shemesh et al., 2001; Shemesh et al., 2004).

Only a few studies have assessed CDI-PTSD for a period that was longer than 18 months. For example, Van Driel and Den Velde (1995) interviewed 18 MI patients two years post-MI using the structured clinical interview for the diagnosis of PTSD (SCID-R-PTSD; Spitzer, Williams, & Gibbon, 1986), and none of the participants was diagnosed as having PTSD. Wikman, Bhattacharyya, Perkins-Poraz, and Steptoe (2008), who evaluated 179 MI patients three years post-MI using self-report questionnaires (PSS-SR; Foa, Riggs, Dancu, & Rothbaum, 1993), classified 13% as having PTSD. The same rate (13.3%), on the basis of a self-report questionnaire (PTSD Inventory; Solomon et al., 1993), was reported by Ginzburg and Ein-Dor (2011) in a sample of MI patients eight years post-MI.

In order to pinpoint the differences in CDI-PTSD prevalence according to the instrument used, Einsle, Kraft, and Kollner (2012) focused specifically on comparing different diagnostic tools for PTSD among coronary artery disease (CAD), heart transplantation and cardiac arrhythmia patients, but again found limited agreement between self-report measures (e.g., Impact of Events Scale-IES-R; Horowitz, Wilner, & Alvarez, 1979; The Post-Traumatic Stress Syndrome 10-Questions Inventory-PTSS-10; Stoll et al., 2000) and the SCID (Structured Clinical Interview for DSM-IV-TR; APA, 2000). As in the aforementioned studies, the prevalence of CDI-PTSD ranged from 15% to 48% when the PTSS-10 was applied, from 5% to 12% when the IES-R was applied, and only from 1.7% to 9.8% when the SCID was used. The vast variance in interval times – between index event and follow-up – should be taken into consideration, however, as they differed greatly among each of the diagnostic groups (ranging from two months to seven years).

In a recent review paper, Edmondson, Richardson, et al. (2012) and Edmondson, Shaffer, et al. (2012) suggested an aggregated prevalence estimate of 12% (95% confidence interval [CI], 9%–16%) for clinically significant symptoms of ACS-induced PTSD. Overall, the prevalence estimate was 16% when self-report instruments were used, while the clinical diagnostic interview yielded a prevalence estimate of 4%.

### 3.2.2. CDI-PTSD stability over time

Several longitudinal studies have followed MI patients over time and assessed the pattern of change in CDI-PTSD symptoms. Symptoms during the first two weeks post-MI predicted PTSD diagnosis and severity one month (Doerfler, 1997; Roberge et al., 2010), three months (Bennett, Owen, Koutsakis, & Bisson, 2002; Doerfler, 1997; Whitehead, Perkins-Poraz, Strike, & Steptoe, 2006), seven months and eight years later (Ginzburg & Ein-Dor, 2011; Ginzburg et al., 2003). Similar findings were reported by Wikman, Molloy, Randall, and Steptoe (2011), who revealed that a patient's CDI-PTSD severity six months post-MI could have been predicted by his/her PTSD severity at two to three weeks after hospitalization. In fact, PTSD symptoms assessed anytime post-MI predicted subsequent PTSD (e.g., Hari et al., 2010; Marke & Bennett, 2013).

Few studies have documented a general pattern of decrease in PTSD rates over the first year post-MI (e.g., Castilla & Vázquez, 2011; Hari et al., 2010; Sheldrick et al., 2006. For example, Hari et al. (2010) followed 274 MI patients for approximately two and a half years after their MI. The researchers documented a significant decrease in post-traumatic stress symptoms from the time of study entry to the time of follow-up. Yet most of the MI patients with CDI-PTSD at follow-up (71.4%) had been identified with CDI-PTSD at baseline.

Other studies have documented a pattern of stability over time during the first three years post-MI (e.g., Shemesh et al., 2006; von Känel, Kraemer, et al., 2010; von Känel, Schmid, et al., 2010; Wikman

et al., 2008). Ginzburg (Ginzburg & Ein-Dor, 2011; Ginzburg et al., 2003) showed that despite a general trend of recovery among those with ASD, a small group consisting of 6% of the MI patients was characterized as a "chronic PTSD group." This chronic group showed a stability of PTSD diagnosis eight years post-MI.

### 3.2.3. Risk factors for CDI-PTSD

3.2.3.1. Demographics. In some studies, a younger age (i.e., early illness onset) was shown to be a risk factor for PTSD (e.g., Bennett & Brooke, 1999; Dinenberg, McCaslin, Bates, & Cohen, 2014; Rocha et al., 2008). Other studies, however, did not find any age effect (e.g., Roberge et al., 2008), or showed an opposite effect (Gao et al., 2015).

This inconsistency in findings was also detected with regard to gender. Some studies found that female MI patients were more prone to develop PTSD than male MI patients (Hari et al., 2010; Roberge et al., 2010). Other studies did not find any gender differences (e.g., Marke & Bennett, 2013; Wikman et al., 2012), or found that male patients were at higher risk for PTSD than female patients (Gao et al., 2015). Ethnicity was also shown to be a risk factor in some studies (e.g., Kutz et al., 1994; Wikman et al., 2008), but not in others (e.g., Ayers, Copland, & Dunnmore, 2009; Wikman et al., 2011). Finally, all but one study (Wikman et al., 2008) found no effect for socio-economic status (SES) (e.g., Ginzburg et al., 2003; Kutz et al., 1994; Whitehead et al., 2006; Wikman et al., 2011; Wikman et al., 2012).

Other demographic variables including marital status, cohabitation, number of children, income, education, employment, and religion were found to be unrelated to CDI-PTSD (e.g., Ayers et al., 2009; Dinenberg et al., 2014; Edmondson et al., 2011; Ginzburg et al., 2003; Kutz et al., 1994; Roberge et al., 2008). Thus, no demographic variable was consistently found to be a reliable risk factor for CDI-PTSD.

3.2.3.2. Objective severity of MI. Studies that examined objective measures of MI severity in terms of the development of ASD and PTSD yielded inconclusive findings. Some findings showed that more intrusive medical procedures were themselves risk factors for CDI-PTSD. Patients who underwent medical procedures or interventions such as bypass surgery and angioplasty tended to report more CDI-PTSD symptoms than patients who had experienced an MI but had not undergone these procedures (Chung, Berger, & Rudd, 2008). Other indicators of MI severity such as number of days of hospitalization following the MI (Ginzburg & Ein-Dor, 2011) and previous MI (e.g., Wikman et al., 2008) were also associated with CDI-PTSD.

Other studies did not find that clinical indicators of ACS severity (including left ventricular ejection fraction, GRACE (Global Registry of Acute Cardiac Events) risk score for major cardiac events, C-reactive protein (CRP), level of creatinine kinase enzymes, number of coronary occlusions, type and location of MI, duration of delirium, recurrent cardiac symptoms, and recurrent MI) predicted PTSD (e.g., Guler et al., 2009; von Känel, Baumert, Kolb, Cho, & Ladwig, 2011; Whitehead et al., 2006; Wikman et al., 2011).

In most cases, therefore, the concrete indications of the illness severity from the patient's perspective – such as number of days in the hospital or medical procedures – seemed to be relevant to the development of CDI-PTSD. Signs of MI severity which were *not* part of the patient's immediate experience – such as enzyme levels – seemed to be less associated with PTSD.

3.2.3.3. Perceived severity of MI and illness representations. Bennett and Brooke (1999), in a retrospective study done six to 12 months post-MI, found a positive correlation between awareness that the incident had been an MI and the occurrence of intrusive thoughts. Other studies showed that pain intensity and pain duration – as well as perceived severity of the MI and the perception of a threat to one's life during the MI – were risks for PTSD (e.g., Ayers et al., 2009; Oflaz et al., 2014; Wikman et al., 2012).

Several longitudinal studies detected the implications of a range of

negative *illness representations* for PTSD (e.g., Sheldrick et al., 2006). These representations included patients' negative emotional representation, concern regarding and comprehension of their diagnosis, negative perception of consequences, and anticipation of permanent disability (e.g., Marke & Bennett, 2013; Wikman et al., 2011). Collectively, these findings show that perceived severity of illness and negative illness representations are associated with elevated levels of CDI-PTSD among MI patients.

### 3.2.3.4. Distress during MI or during hospitalization.
The level of distress during the MI and the presence of depressive symptoms were shown to be associated with ASD symptoms (Roberge et al., 2008). Depression, anxiety and negative affect during the cardiac event were shown to predict subsequent CDI-PTSD (e.g., Bennett, Conway, Clatworthy, Brooke, & Owen, 2001; Wikman et al., 2008). Peri-traumatic distress (i.e., intensity of fear, threat, helplessness, loss of control, or feelings of horror during MI), as well as being surprised by the event, were also shown to be CDI-PTSD risk factors (Bennett et al., 2002; Marke & Bennett, 2013).

Finally, Edmondson and his colleagues showed that emergency department (ED) crowding – measured as the number of patients admitted to the ED during the ACS patient's stay in the ED – and its interaction with in-hospital depression predicted CDI-PTSD one-month post-hospitalization (Edmondson, 2014; Edmondson, Shimbo, Ye, Wyer, & Davidson, 2013). Another recent study showed that the perception of a hectic hospital environment was associated with the development of ASD symptoms during hospitalization (Meister et al., 2016).

### 3.2.3.5. Previous psychological vulnerability.
Exposure to negative life events prior to an MI was found to be associated with posttraumatic symptoms following an MI. More specifically, stressful life events experienced before an MI were associated with ASD during hospitalization, with CDI-PTSD three months following the MI, and with a slower recovery from CDI-PTSD from the time of hospitalization up to eight years post-MI (Ayers et al., 2009; Ginzburg & Ein-Dor, 2011). Similarly, CDI-PTSD was shown to be associated with prior non-cardiac PTSD, depression and reports of psychological problems (Ayers et al., 2009; Kutz et al., 1994; Whitehead et al., 2006). Thus, pre-MI psychological vulnerability contributes to the development of PTSD symptoms following an MI.

### 3.2.3.6. Social support, ways of coping and personality traits.
The availability of social support was shown to be associated with low risk for CDI-PTSD (e.g., Marke & Bennett, 2013; Pedersen, Middle, & Larsen, 2003; Pedersen et al., 2002). In a recent longitudinal study, Dinenberg et al. (2014) found that the absence of social support, as perceived among a large sample of patients who did not have PTSD, predicted the development of CDI-PTSD five years later. Yet, a few studies did not find an effect of perceived social support or size of social network on CDI-PTSD (Roberge et al., 2008; Whitehead et al., 2006).

Dysfunctional coping strategies (emotional, cognitive and behavioral avoidance) were shown to be associated with CDI-PTSD in one study (Ayers et al., 2009). Other studies, showed that problem- and emotion-focused ways of coping were associated with lower odds of developing CDI-PTSD (Marke & Bennett, 2013).

Neuroticism (Chung, Berger, Jones, & Rudd, 2006; Chung et al., 2011) and anxious attachment (Gao et al., 2015) as well as Type-D personality (the consistent inclination to experience both negative affectivity and social inhibition - Denollet, 2005) were associated with CDI-PTSD (Pedersen & Denollet, 2004; Whitehead et al., 2006; Wikman et al., 2008). Yet inconsistencies were revealed with regard to emotional inhibition and emotional disclosure (Ginzburg, Solomon, & Bleich, 2002; So & La Guardia, 2011). Similarly, in-consistent findings were detected with regard to the association between alexithymia (an individual's difficulty in identifying feelings) and CDI-

PTSD (Bennett & Brooke, 1999; Gao et al., 2015; Marke & Bennett, 2013; Whitehead et al., 2006).

In sum, when either ASD or PTSD were assessed (from 48 h to six months post-MI), most risk factors were viewed as falling under the category of psychological distress – either premorbid distress or distress during the event – or personality difficulties.

### 3.2.4. Consequences of CDI-PTSD
Several studies have examined the implications of CDI-PTSD on subsequent major adverse cardiac events (MACE) or all-cause mortality (ACM), yielding mixed results. Shemesh et al. (2001) found that patients with above-threshold PTSD symptoms at six months post-event had a 42–50% prevalence of experiencing other serious cardiac events during the next six months compared with a 26–32% prevalence of the same among patients without PTSD. More recently, Edmondson et al. (2011) found that severity of intrusions measured one-month post-first MI predicted recurrent MACE/ACM 42 months later. Patients scoring higher than 11 on the intrusions dimension of the Impact of Event Scale -IES (Horowitz et al., 1979) ran more than three times the risk of developing MACE/ACM than did those classified as low on this symptom cluster. Similarly, von Känel et al. (2011) found PTSD to predict non-fatal CVD-related hospital readmissions one to four years later. Yet Pedersen, Van Domburg, and Larsen (2004) found that PTSD measured four to six weeks post-MI was not predictive of MACE nine months later. Finally, Ginzburg et al. (2015) demonstrated that dissociative symptoms during hospitalization predict 15-year all-cause mortality among MI patients.

As for re-hospitalizations, increasing levels of CDI-PTSD, measured by the PTSD Symptom Scale - PSS (Foa et al., 1993) and the IES were associated with readmissions due to cardiovascular causes (Doerfler, Paraskos, & Piniarski, 2005). PTSD patients (also classified using the IES) were more than twice as likely as those without PTSD to be readmitted during the year following their MI (Shemesh et al., 2004). However, findings of Hari et al. (2010) showed that PTSD measured by the PDS may not be associated with frequency of CVD-related hospital readmissions.

Using the same database, von Känel et al. (von Känel, Abbas, Begre, Gander, et al., 2010; von Känel, Abbas, Begre, Saner, et al., 2010; von Känel, Abbas, Schmid, et al., 2010; von Känel, Kraemer, et al., 2010; von Känel, Schmid, et al., 2010; von Känel et al., 2009) – focusing on biomarkers known to be associated with cardiac illness – showed that PTSD was associated with elevated levels of inflammatory markers (IL-6, sCD40L, and CRP), cellular adhesion molecules (CAM), High Density Lipoprotein Cholesterol (HDL C), and liver enzymes, but not with coagulation and stress hormones. Though innovative in the sense of assessing the putative associations among CDI-PTSD and biomarkers, these findings can only be considered correlational, since the relevant studies lacked baseline measures of the biomarkers in question, and a causal relationship cannot be ascertained.

Most studies did not find CDI-PTSD to associate with risk factors for a future MI such as BMI, high blood pressure, diabetes, non-participation in physical activity, alcohol consumption, smoking and lack of medication adherence (e.g., Chung et al., 2008; Edmondson et al., 2011; Roberge et al., 2008). In a series of longitudinal studies, however, Shemesh et al. did find a positive association between CDI-PTSD and non-adherence to medication (Shemesh et al., 2001, 2004, 2006), as did Sumner et al. (2015) more recently.

Both retrospective and prospective studies detected the detrimental effects of CDI-PTSD on psycho-social functioning including, among others: poorer quality of life and health-related quality of life, depression, anxiety, anger, overall psychopathology, lower levels of resiliency, failure to return to work, less social activity, and incapacitation (e.g., Chung et al., 2008; Doerfler et al., 1994; Doerfler et al., 2005; Ginzburg & Ein-Dor, 2011; Meister et al., 2015; Shaffer, Kronish, Burg, Clemow, & Edmondson, 2013; Wikman et al., 2008).

### 3.2.5. Treatment

Despite the many studies that have focused on MI-induced PTSD, and despite its proven negative consequences, only a few interventions have been reported, mainly as case reports (e.g., Kutz et al., 1988; Shalev, Schriber, Galai, & Melamed, 1993). Due to the small number of patients involved, the effectiveness of these techniques cannot be determined. Only two studies, both conducted by Shemesh and colleagues (Shemesh et al., 2006; Shemesh et al., 2011), empirically examined the effectiveness of trauma-focused Cognitive Behavioral Therapy (CBT) (based on prolonged exposure principles, Foa, 2000) treatment on MI-induced PTSD. The findings were modest most probably due to lack of sufficient statistical power.

In sum, findings show that rates of MI-induced PTSD are substantive (4–16% depending on the measurement tool used) and relatively stable over time, though a trend toward recovery exists. It was also found that PTSD symptoms assessed any time after an MI predicted subsequent PTSD. A small number of patients can therefore be considered chronic, and they sustain symptoms for a long period of time after their initial cardiac episode. Among the many variables suspected to be CDI-PTSD risk factors, only two emerged most consistently: distress during the acute event or after, and perceived severity of the event. Finally, CDI-PTSD post-MI was found to be associated with a wide range of negative physical and emotional consequences, from overall psychopathology to mortality. Evidence shows that the implications of MI-induced PTSD are not only wide but also long-lasting.

### 3.3. Cardiac arrest

Cardiac arrest (CA) is the cessation of normal circulation of blood due to failure of the heart to contract effectively, and it can lead within minutes to sudden cardiac death. Immediate treatment via cardiopulmonary resuscitation (CPR) and advanced cardiac life support (ACLS) is therefore essential. Patients who go through such a near-death experience – or even those who have simply been told, post-event, how critical their situation was – will likely be at risk for the development of PTSD. Only three studies to date have assessed CDI-PTSD among this population (Gamper et al., 2004; Ladwig et al., 1999; O'Reilly, Grubb, & O'Carroll, 2004).

#### 3.3.1. Prevalence, risk factors, and consequences

The prevalence of CDI-PTSD in these studies ranged from 15% to 38% depending on the measure used. A high PTSD prevalence was also detected when a psychiatric interview was applied, for example, SCID (First, Spitzer, Gibbon, & Williams, 1997): 19% in O'Reilly et al. (2004).

Putative risk factors which were evaluated within this population were not found to be associated with the onset of CDI-PTSD. These factors included demographics, characteristics of the location and time of onset of the cardiac event, number of electric shocks delivered, return of spontaneous circulation before arrival of the emergency physician, in- or out-of-hospital CPR, causes of cardiac arrest, length of time of chest compressions, amount of administered epinephrine, endotracheal intubation/ventilation, length of ICU stay, amount and timing of sedative or analgesic drugs used. Only two factors – sedation at the moment of cardiac arrest, and older age – were found to be protective against CDI-PTSD. Finally, CDI-PTSD among cardiac arrest patients was related to various measures of emotional distress, impaired quality of life and preoccupation with somatic symptoms.

The aforementioned three studies contain several substantial shortcomings including small samples, lack of clarity and inconsistency with regard to PTSD criteria, and a too-wide range of follow-up timing (between 9.6 and 45 months after the index event). Despite these drawbacks, however, these studies provide a preliminary indication of the high incidence of PTSD among this population and the substantial adverse consequences that result from it.

### 3.4. Implantable cardioverter defibrillator (ICD)

Over the last 25 years, the implantable cardioverter defibrillator (ICD) has become the therapy of choice for the prevention of sudden cardiac death in patients with organic heart disease. When the ICD detects a malignant arrhythmia, it first monitors and then restores normal heart rhythm. In the event of ventricular tachycardia or fibrillation, the ICD delivers an electric shock to the heart (usually a very painful experience for the patient). While this device saves lives, it is also a constant reminder of the patient's mortality and the fact that he/she is living with a chronic and perhaps fatal disease (Hamner, Hunt, Gee, Garrell, & Monroe, 1999; Matchett et al., 2009).

ICD patients may also undergo episodes of intensive device-firing ("electric storms," defined as at least three shocks in a 24-hour period, Kapa et al., 2009) – frightening experiences which leave the patient feeling helpless and at the mercy of forces outside of his/her control (Hamner et al., 1999). The prevalence of electric storms is high, ranging from 4% to 28% (Jordan, Titscher, Peregrinova, & Kirsch, 2013). It is therefore likely that ICD implantation and especially actual episodes of device-firing will ultimately be found to be yet another risk factor for CDI-PTSD.

#### 3.4.1. Prevalence and stability over time

Following the initial case studies in the field (Fricchione & Stefano, 1994; Hamner et al., 1999; Maryniak, Szumowski, Orczykowski, Przybylski, & Walczak, 2009), more systematic studies using larger samples found a PTSD prevalence of between 7.6% and 30% among ICD implantation patients (e.g., Habibovic, van den Broek, Alings, Van der Voort, & Denollet, 2012; Lang et al., 2014; Morken et al., 2013; von Känel et al., 2011).

Following individuals' courses of CDI-PTSD, Versteeg, Theuns, Erdman, Jordaens, and Pedersen (2010) found that 60% of ICD patients with CDI-PTSD, at three months post-implantation, also retained their diagnosis at six months post-event. Less stability was reported by Lang et al. (2014), who followed a group of ICD patients (43 months after implantation, on average) for five months. In this study, 29% of those identified with CDI-PTSD at time 1 were also diagnosed with CDI-PTSD at time 2, and 7.7% developed new-onset PTSD at time 2. Assessing patients over a period of five years, however, revealed that only 19% of patients had CDI-PTSD both at baseline as well as at follow-up, and an additional 18% of patients without CDI-PTSD at baseline developed CDI-PTSD later on (von Känel et al., 2011).

Only one study detected a higher CDI-PTSD prevalence among ICD patients who experienced electric storms compared to ICD patients who did not (Kapa et al., 2009). This difference however was significant only at baseline – i.e., two months post-ICD implantation - but not at the 6-month and 12-month follow-ups.

#### 3.4.2. Risk factors and consequences

Younger age, emotional distress before device-implantation, Type-D personality, non-constructive support and greater frequency of chest pain were found to be associated with higher levels of PTSD (Ladwig et al., 2008; Morken et al., 2013; Versteeg et al., 2010; von Känel et al., 2011). Other demographic variables (gender, marital status, education level, and employment status), LVEF status or any classic CHD risk factors, and the prescription of standard cardiac disease medication, were not (e.g. Ladwig et al., 2008). Actual firing of the device was found to be predictive of CDI-PTSD in some studies (e.g. Versteeg et al., 2010; von Känel et al., 2011), but not in others (e.g. Ladwig et al., 2008; Prudente, Reigle, Bourguignon, Haines, & DiMarco, 2006).

Ladwig et al. (2008) showed that the hazard ratio of death among post-traumatic ICD patients was almost four times higher than among non-CDI-PTSD patients. In this study, however, PTSD was measured comparatively, not absolutely; it is therefore difficult to determine the exact prevalence of PTSD cases and the authors' findings should be interpreted with caution.

Finally, Prudente et al. (2006) called attention to the unique phenomenon termed *phantom shocks*. Phantom shocks are the shocking sensations that individuals feel in the absence of a true firing of the device, i.e., shocks that are not confirmed to have taken place during a subsequent device interrogation. Although neither these researchers nor others (Bilanovic et al., 2013) have documented an association between the phantom shock experience and PTSD symptoms, this phenomenon may be interpreted as a PTSD symptom in and of itself, as representing a flashback or intrusion.

### 3.4.3. Treatment

Ansari and Arbabi (2014) and Bhuvaneswar, Ruskin, Katzman, Wood, and Pitman (2014) both reported on small studies in which pharmacotherapy resulted in a reduction of CDI-PTSD symptoms in an ICD patient with PTSD. In line with their hypothesis, they showed a nonsignificant trend according to which patients taking lipophilic beta-blockers, which are capable of crossing the blood-brain barrier and thus opposing the potentiation of memory consolidation by stress, had lower levels of PTSD than those taking hydrophilic beta-blockers, which do not cross this barrier.

In sum, high levels of CDI-PTSD were detected among patients who had an ICD implantation; however, as was found among MI patients, a trend toward recovery over time was also identified among this population. As with MI patients, emotional factors were also the most prominent risk factors for CDI-PTSD in ICD implantation patients. Almost no studies have focused on CDI-PTSD consequences among this population; however, findings regarding the possibility of much higher death rates among posttraumatic ICD patients do exist.

### 3.5. Coronary artery bypass surgery (CABG)

MI can sometimes necessitate emergency surgery. Yet most studies on PTSD among patients undergoing surgery focused on *elective* surgery. Most studies in this category provided data on both coronary artery bypass graft surgery (CABG – interchangeably referred to as bypass surgery, coronary artery bypass surgery, or "open-heart surgery") and valve replacement surgery (VR), which also requires an open heart surgical procedure in order to replace the diseased valve.

What was unique among studies focusing on this population of patients was the assessment of PTSD *pre-surgery*, i.e. as a consequence of the diagnosis and anticipation of surgery. Bayer-Topilsky et al. (2013) referred to the diagnosis itself as a potentially traumatic event and indeed found higher levels of PTSD among patients who received a diagnosis of mitral regurgitation – a mitral illness which often requires surgery to repair or replace the valve - as compared to control groups.

### 3.5.1. Prevalence and stability over time

Oxlad et al., in a series of studies based on the same database of patients undergoing elective CABG (Oxlad, Stubberfield, Stuklis, Edwards, & Wade, 2006a; Oxlad, Stubberfield, Stuklis, Edwards, & Wade, 2006b; Oxlad & Wade, 2006; Oxlad & Wade, 2008), reported a 7% prevalence of clinically significant CDI-PTSD *prior to* surgery. A higher prevalence rate of pre-operational CDI-PTSD (12.7%) was detected when measured *retrospectively* a few days after an emergency operation (Boyer et al., 2013). Aside from the difference in study designs (prospective versus retrospective), this difference in CDI-PTSD prevalence is likely attributable to the different context of the operation (elective versus emergency) and might influence the development of CDI-PTSD.

In the few studies which assessed prevalence of CDI-PTSD over time, results were equivocal. Whereas a few studies detected a reduction of prevalence both at a six-month follow-up (Oxlad & Wade, 2008—5.8%) and at a one-year follow-up (Rothenhousler et al., 2005—6.6%), others (e.g. Tarsitani et al., 2012) detected a much higher prevalence (19.7%) a year after surgery.

### 3.5.2. Risk factors

Medical factors such as disease severity, a longer operation, post-operative delirium, the duration of postoperative intensive care unit (ICU) treatment and the use of invasive versus less invasive medical procedures were all significantly associated with higher PTSD scores measured after surgery (Gulielmos et al., 1999; Rothenhousler et al., 2005; Schelling et al., 2003; Stoll et al., 2000). As for psychological risk factors, a premorbid mental health disorder, low preoperative health-related quality of life (HRQL), poorer self-rated health, negative illness representations, increased use of avoidant coping and less optimism pre-operatively (Oxlad & Wade, 2006, 2008; Rothenhousler et al., 2005; Schelling et al., 2003) were all significantly associated with increased CDI-PTSD symptomatology.

### 3.5.3. Consequences

CDI-PTSD did not predict cardiac-disease-related readmissions (Oxlad et al., 2006b); it was, however, found that lower levels of CDI-PTSD (in relation to diagnosis) predicted a longer hospital stay after CABG (Oxlad et al., 2006a). The authors explained this finding by suggesting that a shorter hospital stay might have served as a way of coping via avoidance or as showing a tendency for lower adherence to the required medical regimen. However, in the absence of information regarding the exact medical recommendation for a longer hospital stay, this explanation can only be regarded as speculative.

CDI-PTSD cases detected six months post-surgery showed no improvement in HRQL (Health Related Quality of Life) and no gain in physical functioning; they also showed a deterioration in social functioning and mental health, as measured pre-operatively, compared to patients without CDI-PTSD (Schelling et al., 2003).

In sum, the levels of CDI-PTSD as a consequence of CABG were somewhat lower than those found among MI patients, and an inconsistency exists regarding the stability of symptoms over time. The risk factors for CABG-induced PTSD are mostly psychological; however, a few illness-related factors were also detected. As has been seen in all other cardiac-related diagnoses and procedures discussed so far, the consequences of CDI-PTSD were negative here as well. The variance among studies focusing on this specific population makes it difficult to establish concrete conclusions.

### 3.6. Heart transplantation

Only those patients whose heart failure is so severe that it cannot be treated with medication or other forms of treatments are the designated recipients of heart transplantation (HT). In HT, the highly damaged heart is removed and replaced with a healthy heart from a donor. Currently, the one year survival rate following HT is almost 81% (Lund et al., 2013). Despite the fact that the overall mortality rates of patients waiting for heart transplantation has decreased, the mortality on the waiting list is still substantial (Johnson et al., 2010). Ventricular assist devices (VADs) are artificial hearts used increasingly as "bridges" to heart transplantation among patients who are in danger of dying before the HT can take place (Dew, Kormos, Winowich, et al., 2001, p. 1200).

### 3.6.1. Prevalence and stability over time

Prevalence of PTSD as detected via clinical interview ranged from 10.8% to 19.3% 12 months after transplantation (e.g., Dew et al., 1996; Dew et al., 1999). Thirty-six months after transplantation, reported rates were 22% (e.g., Dew et al., 1996; Dew et al., 1999; Dew, Kormos, DiMartini, et al., 2001; Stukas et al., 1999). Lower rates (9.8%–13%) were reported in samples that were more heterogeneous in terms of time post-transplantation (one to five years: Favaro et al., 2011; four months to seven years: Kollner et al., 2002). However, neither Inspector, Kutz, and David (2004) nor Bunzel et al. found any acute PTSD among their samples of HT patients (e.g., Bunzel, Roethy, Znoj, & Laederach-Hofmann, 2008).

In a study that compared patients who underwent heart transplan-

tation after having been first supported by VAD with patients who underwent heart transplantation without having had the VAD – 12 months post heart transplantation surgery – no differences were found between the two groups' CDI-PTSD rates (16.7% and 19.7%, respectively) (Dew, Kormos, Winowich, et al., 2001).

### 3.6.2. Risk factors

Dew, Kormos, DiMartini, et al. (2001) identified the following risk factors for a cumulative spectrum of anxiety disorders [i.e., general anxiety disorder (GAD), PTSD and adjustment disorder with anxiety symptoms]: psychiatric history, gender (female), longer hospitalization post-transplantation, and low support from family members. Unfortunately, the authors did not assess the specific risk factors for CDI-PTSD exclusively.

### 3.6.3. Consequences

Recipients who met the criteria for CDI-PTSD during the year after heart transplantation surgery were over 15 times more likely to die by the three-year post-transplantation mark (Dew et al., 1999) than were recipients who did not meet the CDI-PTSD criteria. The effect of CDI-PTSD on mortality, however, was not replicated by Favaro et al. (2011) in a sample of patients one to five years post-transplantation.

Patients with CDI-PTSD had a significantly poorer quality of life when compared to patients without CDI-PTSD (Kollner et al., 2002). However, as this was a retrospectively designed study, no unequivocal differentiation between consequences and associated factors can be made. Favaro et al. (2011) noted a trend toward an association between the presence of transplantation-related PTSD intrusive symptoms and poor medical adherence. No associations were found among CDI-PTSD and rejection, malignancies or mortality.

## 4. Discussion

In the following pages, we will summarize the main findings of the current literature review. Primarily, we wish to highlight the most salient questions emerging from the integration of findings. As such, we will focus on the unique nature of cardiac disease as a potentially traumatic event as well as the unique features of CDI-PTSD as a valid diagnostic entity. We will also bring attention to the problem of establishing the actual prevalence of CDI-PTSD, and the issue of partial CDI-PTSD. Finally, we will conclude by identifying those areas in the field that call for more scientific and clinical attention, such as the lack of research on appropriate treatments for this population, and the existence of CDI-PTSD among patients' caregivers.

### 4.1. Main findings

About half of all of the studies reviewed were conducted on patients following an MI, whereas the minority of studies focused on patients who had experienced cardiac arrest or had undergone specific medical procedures (e.g., cardiac surgery, heart transplantation). Comparing the rates of CDI-PTSD across illnesses, diagnoses and procedures revealed that cardiac arrest yielded the highest CDI-PTSD prevalence, followed by the other cardiac-disease-related diagnoses and invasive procedures, among which the prevalence of CDI-PTSD was similar. Given the fact that individuals who have undergone a cardiac arrest have, in a way, experienced death, this finding is not surprising. Yet we should view this prevalence hierarchy with caution since very few studies have focused specifically on cardiac arrest patients.

Overall, findings show that rates of CDI-PTSD are relatively stable over time, though a trend toward recovery exists. A small group of cardiac patients exhibited chronic CDI-PTSD; and not enough data has been accumulated regarding delayed PTSD – the development of CDI-PTSD symptoms after an initial period in which patients were asymptomatic, or at least without a clinical-level CDI-PTSD (Hari et al., 2010). Similar trends of fluctuations in PTSD over time have been observed

in survivors of other types of trauma such as accidents (Carty, O'donnell, & Creamer, 2006) and war (Andersen, Karstoft, Bertelsen, & Madsen, 2014).

No demographic variable was consistently found to be a reliable risk factor for CDI-PTSD. In other areas of post-trauma, however, the following variables were found to be reliable risk factors for PTSD: female gender, younger age, racial/ethnic minority status, and lower intelligence (e.g., Baily & Elmore, 1999; Brewin, Andrews, & Valentine, 2000). This discrepancy may be attributed to some of the specific characteristics of cardiac disease (i.e., a higher prevalence of male patients than female patients, who have their first cardiac episode at a younger age than women do; Bjarnason-Wehrens, Grande, Loewel, Völler, & Mittag, 2007).

The most consistent risk factor found for CDI-PTSD – as has also been found for PTSD resulting from other ("traditional") events (King, Pless, Schuster, & Potter, 2012) – was psychological functioning, whether it was conceptualized as premorbid distress, distress during the event, or premorbid personality difficulties.

Focusing on medical- and illness-related putative risk factors, it was the *perceived* severity rather than the *objective* severity which was associated with the development of CDI-PTSD. This finding corresponds with numerous findings in the health psychology field attesting to the more important role played by perceived illness severity than by objective illness severity in terms of determining both patient distress and adherence (DiMatteo, Haskard, & Williams, 2007; Grigioni et al., 2003; Naidoo & Wills, 2000).

CDI-PTSD was found to be associated with a wide range of negative physical and emotional consequences, from overall psychopathology to mortality. Since most studies reviewed were either cross-sectional or retrospective, however, it was often difficult to differentiate the consequences of CDI-PTSD from its risk factors. Yet the findings regarding elevated levels of mortality and cardiac events associated with CDI-PTSD are worthy of continued study, considering the worldwide prevalence of cardiac disease.

### 4.2. Emerging questions

#### 4.2.1. "If it looks, walks, and swims like PTSD, IS it PTSD?"

The cardiac patient who experienced a heart attack in his bed while the air-conditioner was running and now can neither sleep in his bed nor operate the air-conditioner; the female nurse who experienced her MI on a cruise where the medical aid was absent and is now unable to resume her life; the retired high-ranking officer who was able to bear witnessing the deaths of his soldiers on the battlefield but has been unable to sleep since going through an electric storm caused by his implantable cardiac defibrillator: Can these episodes be regarded as what Naomi Breslau (2012, p. 84) refers to as "different from other 'ordinary' stressful experiences?"

This question mirrors the emerging discussion in the field of trauma regarding whether the concept of trauma has become too broad, including as it does low magnitude stressors and seeing them as equivalent to severe traumatic events [i.e., fulfilling Criterion A (McNally, 2003). For the debate on this issue see, for example Kilpatrick, Resnick, & Acierno, 2009; Brewin, Lanius, Novac, Schnyder, & Galea, 2009; Spitzer, First, & Wakefield, 2007; Roberts et al., 2012; Kilpatrick et al., 2013; Friedman, Resick, Bryant, & Brewin, 2011; Weathers & Keane, 2007]. This trend, toward broadening the definition, has been termed the "conceptual bracket creep" by McNally (2003, 2009), and the resultant wariness has contributed to the new rigorousness of the Criterion A definition with regard to illnesses in the latest edition of the DSM (DSM-5, APA, 2013).

As suggested by Kilpatrick et al. (2009), the question of whether a certain event qualifies as a Criterion A event, potentially evoking PTSD, should be answered by empirically-informed studies. Such studies would examine whether events, and their specific characteristics, increase the risk of PTSD. Reviewing the data accumulated so far seems

to validate the assumption that *acute* cardiac events qualify as meeting even the strict DSM-5 definition of Criterion A, and can thus be seen as potential causes of PTSD.

According to the current review, a substantial prevalence of CDI-PTSD was detected among cardiac patients. But could this prevalence be the consequence of applying PTSD questionnaires to these individuals? Do we compel an experience to be named PTSD simply because we know how to measure it? Should the psychological manifestations of these patients indeed be classified as PTSD?

We propose a two-pronged answer to these questions. First, most of the empirical research shows that overall we are witnessing a phenomenon which very much resembles PTSD resulting from other origins. Risk factors for its occurrence are mainly psychological, as in PTSD resulting from other causes. Also, both the physical and emotional consequences are consistently negative, as they are with PTSD stemming from other causes. Cardiac patients who have experienced a cardiac event do not find subsequent intrusions, or avoidant behaviors, to be in any way off the spectrum of their post-event-experience, and they easily respond to these items on the PTSD questionnaires. Also, it is the emotion of fear of death which dominates the experience of patients with CDI-PTSD, just as it is with PTSD deriving from other sources (Ball & Stein, 2012). Applying Cronbach and Meehl's (1955) terminology, we detected a substantive nomological net for CDI-PTSD in terms of prevalence, risk factors, consequences and course of the disorder. Therefore, our answer, based on empirical findings, is affirmative: the accumulated data strongly attest to the existence of PTSD as emerging from a cardiac event, at least in the case of an acute coronary event, the subject of most of the conducted research.

The unique characteristics of cardiac disease – namely being internal, ongoing, and future-oriented – must also be kept in mind. In a life-threatening illness, such as a cardiac illness, the source of the threat is internal; the threat and the individual, therefore, cannot be separated (Buckley, Green, & Schnurr, 2004; Edmondson, 2014; Green, Epstein, Krupnick, & Rowland, 1997). Since the event (the illness) is often the acute manifestation of a permanently disrupted physiological system, the patient is often exposed to an ongoing internal somatic threat (Edmondson, 2014). This threat is not exclusively related to past experience; rather, it is chronic, and in many ways it is anchored in fears and worries about the future, vis-a-vis treatment, illness progression, potential recurrence and even death (Green et al., 1997; Mundy & Baum, 2004). These unique illness characteristics shape the *nature* of CDI-PTSD symptoms. While the classic PTSD intrusive symptoms focus on the past event, in cardiac disease most of the intrusive symptoms relate to the future (Edmondson, 2014; Mundy & Baum, 2004). Thoughts and images related to the threat of recurrent heart attack, fear of future death, worries about forthcoming doctor appointments, and other future-oriented concerns may constitute what Holmes and colleagues (2007) termed "flash-forward" intrusions.

In addition, avoidance – a strategy used to cope with intrusive memories and elevated arousal – is almost impossible for the MI patient (Mundy & Baum, 2004), who because of his/her condition must continue to see physicians, enter hospitals, and undergo medical procedures. Moreover, there are somatic signals –the sense of quickened heartbeat, shortness of breath, dizziness – which serve to continuously remind the patient of the traumatic heart event he/she experienced. Finally, for the cardiac patient coping with CDI-PTSD, the hyper-arousal symptoms are identical to the symptoms indicative of life-threatening cardiovascular reactivity. These symptoms are somatic cues which condemn the CDI-PTSD patient to a continuous cycle of anxiety and sympathetic/cardiovascular reactivity (Edmondson, 2014).

In sum, adopting a generic approach that views CDI-PTSD as PTSD suggests that the process of adjustment to the traumatic (acute cardiac) event is similar in nature to that of PTSD resulting from other origins. Nevertheless, consideration must be given to the unique expressions of CDI-PTSD, and how CDI-PTSD differs from PTSD resulting from other sources.

The question thus emerges as to whether the standard instruments used to assess PTSD are applicable to the cardiac patient population. It may well be that new and more accurate instruments should be developed and applied, instruments which would consist of items tapping the unique manifestations of CDI-PTSD; for example, future-oriented intrusions. More suitable PTSD measures may be better able to pinpoint those individuals whose unique CDI-PTSD manifestations are not picked up by the standard PTSD instruments.

Finally, the DSM-5 clearly states that: "A life-threatening illness or debilitating medical condition is not necessarily considered a traumatic event. Medical incidents that qualify as traumatic events involve sudden, catastrophic events…" (p. 274). Nevertheless, several studies detected the existence of PTSD symptoms emerging as a consequence of other, much less abrupt events, such as receiving a diagnosis of a cardiac disease (Bayer-Topilsky et al., 2013), or knowing about the specific kind of cardiac operation one is about to undergo (Gulielmos et al., 1999). Whether or not the diagnosis of a severe physical illness, per-se, can be conceived as Criterion A for PTSD may bear important consequences for intervention (Kangas, Henry, & Bryant, 2002).

### 4.2.2. The prevalence enigma

The studies presented a large variance in the prevalence of CDI-PTSD, ranging from 0% to 38%, but averaging at 4%–16% depending on the diagnostic tool used (Edmondson, Richardson, et al., 2012; Edmondson, Shaffer, et al., 2012). These rates are compatible with reports of prevalence rates of lifetime PTSD in the general population (5–10%, Ozer, Best, Lipsey, & Weiss, 2008; 8–12%, Norris & Slone, 2007) as well as with rates of PTSD among those exposed to a traumatic event (10%–20%, Norris & Slone, 2007). Thus, just as with individuals who have been exposed to other kinds of traumatic events (King et al., 2012), most cardiac patients do not develop post-traumatic symptoms; indeed many experience distress that diminishes over time, but only a minority of patients have enduring negative reactions considered to be above the clinical threshold.

Many of the studies conducted on coronary event patients detected high odds of *partial* CDI-PTSD (e.g., Castilla & Vázquez, 2011; Chung, Berger, Jones and Rudd, 2008; Chung, Berger and Rudd, 2008). Since people with partial PTSD often present considerable distress and impairment in functioning compared to people without PTSD, it is also a phenomenon worthy of attention. Moreover, these high rates of partial PTSD should be reconsidered in light of the recent revisions in the DSM-5, stating that a trauma survivor in whom "the response to a stressor that meets PTSD Criterion A does not meet all other PTSD criteria (or criteria for another mental disorder) (APA, 2013, p. 279) will be diagnosed as having an adjustment disorder" (Carta, Balestrieri, Murru, & Hardoy, 2009). Whereas patients with a clear PTSD diagnosis are often referred to evidence-based treatments such as cognitive processing or prolonged exposure (Seal et al., 2010), patients with partial PTSD who are diagnosed with an adjustment disorder are less likely to be referred for such treatments.

Finally, the question as to why clinical interviews yielded lower rates of CDI-PTSD than did self-report questionnaires is still to be answered. First, this gap is not exclusive to cardiac patients or to PTSD (e.g., Adams, Soumerai, Lomas, & Ross-Degnan, 1999; Fairburn & Beglin, 1994; Garber, Nau, Erickson, Aikens, & Lawrence, 2004). Self-report measures tend to lend themselves more easily to response bias than do interviews (Bovin & Weathers, 2012), and this bias may cause the inflated PTSD rates when self-reports are administered. It may also be that questionnaires are better at detecting general distress than they are at detecting specific facets of it (Shalev, Peri, Orr, Bonne, & Pitman, 1997); they may therefore be less sensitive than clinical diagnostic interviews in differentiating between PTSD and other manifestations of distress, such as depression: a differentiation which is the essence of a clinical diagnostic interview.

That said, in view of the magnitude of negative consequences for CDI-PTSD (diagnosed either by interview or by self-report questionnaire), it would seem irresponsible to focus only on those 4% who were diagnosed via clinical interview while overlooking the additional mass of patients identified by self-report; doing so might also result in the oversight of a large number of posttraumatic patients.

### 4.2.3. Therapy: the lack of evidence-based practice

Despite the established negative consequences of CDI-PTSD and the vast literature on treating PTSD of other origins (e.g., Back et al., 2014; Blount, Cigrang, Foa, Ford, & Peterson, 2014; Foa & Rothbaum, 2001) only two studies, both conducted by Shemesh and colleagues, empirically examined the effectiveness of treatment for MI-induced PTSD (Shemesh et al., 2006; Shemesh et al., 2011). These findings, though preliminary, provide initial evidence for the effectiveness and safety of exposure therapy for this population.

A few researchers have suggested guidelines for intervention among specific groups of cardiac patients. For example, Neel (2000) focused on ICD patients who coped with CDI-PTSD and mentioned the beneficial effect of group therapy, relaxation therapy, and acute crisis intervention soon after the critical event occurred. Smith, Fogel, and Friedman (1998) advocated for the provision of proactive psycho-education for all patients receiving ICD implants, in order to enhance individual adjustment to the procedure. These recommendations, however, have not been subjected to any kind of systematic empirical scrutiny. Jordan et al. (2013) reported on an ongoing study trial of a psychotherapeutic intervention for ICD patients who experienced electric-storm-induced-PTSD (i.e., they had received multiple electric shocks), which according to preliminary findings seems to be efficient.

Overall, given the unique manifestations of CDI-PTSD, it would seem that the optimal treatment for this disorder should be more *pro*spective than *retro*spective in nature, focusing more on future illness management than on a reconstruction of past systematics (Moye & Rouse, 2015). Much research is still required in order to provide safe and effective psychological interventions to patients who struggle not only with cardiac diseases, but also with secondary PTSD, and psychologists with expertise in diagnosis and treatment should be an integral part of all cardiac units.

### 4.2.4. Family members and CDI-PTSD

Another issue which has been almost completely neglected in the CDI-PTSD literature is the experience of family members, who are themselves potential trauma victims. Partners are the family members most vulnerable in the context of cardiac disease, as the average age of disease onset is late adulthood, when the primary caregiver is usually the spouse. Cardiac partners are usually the ones most proximate to the patients, both concretely when accompanying them during their stay in the Cardiac Care Unit, as well as emotionally when coping with the patients' emotional and behavioral reactions to the disease (e.g., Delon, 1996; Randall, Molloy, & Steptoe, 2009). Therefore, partners of cardiac patients might be highly susceptible to developing CDI-PTSD themselves.

From a theoretical perspective, intimate proximity and caregiving efforts throughout the entire recovery process might put the partners of CDI-PTSD patients at high risk for acquiring what Figley (1995) termed secondary traumatic stress or secondary traumatization. However, studies have shown that partners frequently develop CDI-PTSD *regardless* of the identified patients' own post-traumatic symptoms. These studies found high levels of PTSD among partners (e.g., 23%, 26%) even when the patients themselves presented less or even no symptoms of PTSD (e.g., Bunzel et al., 2008; Fait et al., 2016). These findings corroborate the recent change in the DSM-5 Criterion A which suggests that PTSD can emerge not only when experiencing but also when "*witnessing,* in person, the event(s) as it occurred to others' or "learning that the traumatic event occurred to a close family member or close friend…" (American Psychiatric Association, 2013, pp. 271). Due to the

very limited number of studies focusing on the cardiac partners' experience, however, it is unclear whether the actual witnessing of the event increases the odds of developing PTSD due to the direct, unmediated exposure, or whether secondhand knowledge alone encompasses enough potential for the development of CDI-PTSD, due to heightened feelings of helplessness and loss of control.

### 4.3. Main shortcomings of the studies in the field

As presented in Electronic supplement 2, in this review we analyzed the methodological qualities of the studies in the field such as study design, sample size, response rate, attrition rate and modes of PTSD assessment (i.e., identifying which studies applied clinical impressions only in comparison to clinical interviews, structured clinical interviews, or self-reports). Mapping the field according to these dimensions enabled us to evaluate each study's contribution and trace the most prevalent methodological challenges that still need to be overcome.

Among the most ubiquitous problems detected in the studies reviewed have been the use of extremely heterogeneous samples: combining first-episode patients with those who have suffered recurrent events; combining patients who have different diagnoses; combining patients with vastly different time lags since experiencing their first episode; and even combining patients and their caregivers.

Often an individual's cardiac history has not been measured or controlled for. In addition, many studies lack measurement of pre-cardiac event psychiatric history such as pre-morbid depression or PTSD symptoms; it is therefore impossible to disentangle the genuine consequences of CDI-PTSD from those of pre-morbid psychiatric history in these studies.

Reports of effect sizes are missing in the great majority of studies. This absence makes it difficult to determine the strength of the associations tested.

In addition, many studies lack explicit reports on response and attrition rates (which therefore had to be calculated by the authors of this literature review). Overall, response rates were high and attrition rates were low in cases of procedures such as ICD implantation and cardiac surgeries (including transplantation) in comparison to rates when patients with acute coronary events were recruited.

Another problem in the CDI-PTSD field is the use of diverse diagnostic and screening questionnaires, with different cut-off points and different items being used to measure and identify CDI-PTSD. This lack of uniformity makes it difficult to compare the results that have emerged from different studies. Also, the lack of baseline measurements of many tested variables creates a great challenge in differentiating CDI-PTSD risk factors from CDI-PTSD consequences, even in the longitudinally-designed studies. Finally, due to the fact that this review was conducted before the release of the recent DSM-5 (APA, 2013), the reviewed studies were in accordance with the former DSM-IV (APA, 1994) criteria for PTSD. Therefore, the new cluster of negative alterations in mood and cognitions, which seems highly relevant in the case of CDI-PTSD, was not taken into account.

### 4.4. Limitations, final conclusions and future directions

Before concluding, a few of the current review's limitations should be noted. First, we refrained from executing a meta-analysis due to the enormous variability in the field in terms of different diagnoses, different measurement tools, different time lags since the index event, etc. The review is also limited to peer-reviewed papers which were published in English. We also deliberately refrained from reviewing "grey literature" (e.g., conference abstracts) due to the less rigorous scrutiny it generally receives. Also, we did not include studies conducted on CDI-PTSD among children and their family members, as we thought it important to refrain from trying to cover too much territory in this one review. Nevertheless, very few studies have been conducted on CDI-PTSD among children, and it is a population that

warrants an increased amount of scientific attention. The current review does not tap PTSD as a consequence of strokes (cerebral vascular accidents). While a stroke is also a type of vascular event, its unique manifestations (in particular cognitive deficits) may lead to a different process with regard to PTSD emergence and coping than do cardiac events. Finally, as comprehensively as we tried to cover the scope of the issue – and for this reason searched three major databases – we acknowledge that the current review is of course somewhat limited and that additional databases such as Embase may have yielded more papers.

Overall, the current review sheds light on the phenomenon of CDI-PTSD, emerging among a small segment of the cardiac-disease patient population. Given the absolute number of cardiac-disease patients around the globe, this "small segment" effectively adds up to a large number of individuals (both patients and family members) who may be underdiagnosed and undertreated. More studies are needed in order to 1) develop the optimal diagnostic tool for CDI-PTSD, in accordance with the new DSM-5 diagnostic criteria of PTSD; 2) explore putative buffering variables, such as individual or relational variables for the development of CDI-PTSD; 3) assess the magnitude of CDI-PTSD among family members of cardiac patients; and 4) design specific empirically-supported interventions for those who cope with CDI-PTSD. We would also suggest channeling more effort into establishing whether or not the DSM-5-Criterion A should be reconceptualized to consist of not only experiencing an acute abrupt physical event, but also receiving a cardiac-disease diagnosis or going through an invasive cardiac procedure.

Perhaps the field's most urgent need is to generate more randomized controlled trials for the purpose of developing an evidence-based practice for both cardiac patients and their family members. The results of these efforts would be patients' better ability to cope with the disease, patients' heightened adherence to their medical regimen and, in effect, patients' longer and healthier lives.

Supplementary data to this article can be found online at http://dx.doi.org/10.1016/j.cpr.2017.04.009.

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## Appendix A. Medical abbreviations

| | |
|---|---|
| ACLS | advanced cardiac life support |
| ACM | all-cause mortality |
| ACS | acute coronary syndrome |
| AICDs | automatic implantable cardioverter defibrillators |
| AMI | acute myocardial infarction |
| ASD | acute stress disorder |
| AVR | aortic valve replacement |
| CA | cardiac arrest |
| CABG | coronary artery bypass graft surgery |
| CAD | coronary artery disease |
| CAM | cellular adhesion molecules |
| CDI-PTSD | cardiac-disease-induced PTSD |
| CHD | chronic heart disease |
| CPR | cardiopulmonary resuscitation |
| CVD | cardiovascular disease |
| GAD | generalized anxiety disorder |
| HRQL | health related quality of life |
| HT | heart transplantation |
| ICD | implantable cardioverter defibrillators/device |
| ICU | intensive care unit |
| IHD | ischemic heart disease |

| | |
|---|---|
| LVEF | left ventricular ejection fraction |
| MACE | major adverse cardiac events |
| MI | myocardial infarction |
| PCI | percutaneous coronary intervention |
| PTCA | percutaneous transluminal coronary angioplasty |
| PTSD | posttraumatic stress disorder |
| UA | unstable angina |
| VAD | ventricular assist devices |
| VR | valve replacement |

## Appendix B. Instruments names

| | |
|---|---|
| ASDI | Acute Stress Disorder Interview |
| ASDS | Acute Stress Disorder Scale |
| CAPS–IV | Clinician Administered PTSD Scale, Fourth Edition |
| CCS | Charlson Comorbidity Scores |
| CDIS | Computerized Diagnostic Interview Schedule for DSM-IV |
| CIDI | Clinical Interview using the Michigan version of the WHO/ADAMHA Composite International Diagnostic Instrument |
| DTS | Davidson Trauma Scale |
| GRACE | Global Registry of Acute Cardiac Events |
| HTQ | Harvard Trauma Questionnaire - Part IV |
| IES | Impact of Events Scale |
| PCL-C | PTSD Checklist - Civilian Version |
| PDEQ | Peritraumatic Dissociative Experiences Questionnaire |
| PDS | Posttraumatic Stress Diagnostic Scale |
| PSS | PTSD Symptom Scale |
| PTSDSS | Posttraumatic Stress Disorder Symptom Scale |
| PTSS-10 | Post-Traumatic Stress Syndrome 10-Questions Inventory |
| MPSS-SR | Modified PTSD Symptom Scale—Self-Report |
| RI | Reaction Index |
| SASRQ | Stanford Acute Stress Reaction Questionnaire |
| SCID–IV–ASD | Structured Clinical Interview for DSM-IV, ASD module |
| SCID-DSM-IV-TR | Structured Clinical Interview for DSM-IV |
| SCID-R-PTSD | Structured Clinical Interview for the Diagnosis of PTSD |

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